NATIONAL CANCER INSTITUTE

Tobacco and Alcohol: Understanding Co-Use to Reduce Cancer Incidence and Mortality

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Tobacco and Alcohol: Understanding Co-Use to Reduce Cancer Incidence and Mortality

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About the National Cancer Institute Tobacco Control Monograph Series

The National Cancer Institute established the Tobacco Control Monograph series (formerly the Smoking and Tobacco Control Monograph series) in 1991. The series provides comprehensive scientific reviews of tobacco use, treatment, and prevention topics to inform the work of researchers, clinicians, and public health practitioners working to reduce cancer morbidity and mortality. All 24 Tobacco Control Monographs and their supplemental materials can be downloaded from cancercontrol.cancer.gov/monographs.

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Foreword

Tobacco and alcohol are two of the most commonly used addictive substances in the United States. Whether consumed individually or together, their negative toll on health is substantial. Tobacco use is the leading risk factor for cancer, accounting for nearly 20% of cancer incidence, while alcohol is the third leading cause. In addition to cancer, tobacco and alcohol cause many other chronic diseases; adverse reproductive outcomes; and, especially for alcohol, increased risk of accidents and injury. As this monograph describes, use of both tobacco and alcohol products—known as "co-use"—is common, and there is strong correlation between dependence on tobacco and alcohol. In the United States, an estimated 11.8% of adults (approximately 30 million people) report both smoking cigarettes and drinking alcohol in the past month. Initiation of tobacco and alcohol use often begins in adolescence, and early onset of use is a strong predictor of dependence, cancers caused by tobacco, and many other chronic and acute health conditions.

There are strong biological relationships between tobacco and alcohol. Nicotine (the primary addictive constituent of tobacco) and ethanol (the primary addictive constituent of alcohol-containing beverages) each affect the three major neurobiological circuits implicated in dependence and addiction (i.e., the basal ganglia, the extended amygdala, and the prefrontal cortex). Tobacco use increases the risk of cancer through numerous carcinogens, including those formed from combustion. Alcohol use causes cancer via the effects of ethanol and its metabolic product, acetaldehyde (which is also found in tobacco smoke) and via effects on metabolism, oxidative stress, inflammation, and hormonal pathways. Alcohol may also influence tobacco-related carcinogenesis through its effects on membrane permeability, epigenetic changes, and promotion of tumor growth. In addition to each causing cancer individually, co-use of tobacco and alcohol is associated with synergistically higher risk of head and neck and esophageal cancers, and potentially with cancer at other sites.

With this background in mind, the publication of a National Cancer Institute (NCI) Tobacco Control Monograph is a critical step to increase attention on the importance of addressing co-use of tobacco and alcohol for cancer prevention and health promotion. A key goal of the monograph is to examine how tobacco control, including prevention and cessation of tobacco use, can be improved by addressing alcohol use. As the monograph explains, the co-use of tobacco and alcohol is driven by complex biological and genetic factors, including the neurobiology of addiction, the physiological effects of tobacco and alcohol exposures, behavioral genetics, carcinogenesis, and psychological processes. Tobacco and alcohol co-use is also influenced by an intricate and interconnected landscape of social, economic, and policy factors. The monograph emphasizes each of these important factors by summarizing the literature, highlighting research needs, and drawing conclusions relevant to diverse audiences.

Research focused on tobacco and alcohol has long been funded and conducted by the National Institute on Drug Abuse (NIDA), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and NCI. The work of these three institutes spans neuroscience and behavior, epidemiology, public health interventions, clinical treatment, and medication development, to name just a few scientific domains. In 2013, the National Institutes of Health (NIH) formed the Collaborative Research on Addiction at NIH (CRAN) initiative, an NIH-wide initiative to enhance integration of the work of these three NIH Institutes in better understanding and

addressing morbidity and mortality from alcohol, tobacco, and other addictive substances. The signature project of CRAN is the Adolescent Brain Cognitive Development (ABCD) Study, launched in 2015. This long-term study of brain development and child health enrolled nearly 12,000 children ages 9–10 who are being followed for at least a decade using structural and functional brain imaging together with genetic, neuropsychological, behavioral, and other health assessments to understand brain development in adolescents. Among the questions to be addressed by ABCD is how the use of tobacco and alcohol affects the structure and function of the adolescent brain. This volume clearly highlights the importance of this question because both tobacco and alcohol use typically begins during adolescence, and early onset of substance use is linked to greater dependence and higher risk of disease. By disseminating the latest evidence and effective practices related to addressing tobacco and alcohol co-use, this monograph makes an important contribution to the enhanced integration that was envisioned when CRAN was formed.

This monograph describes numerous research needs that may inform scientists in these fields. A few are highlighted here:

1. Improved measurement and epidemiological surveillance of tobacco and alcohol use is frequently identified as an important area for improvement. Many studies would benefit from assessing both tobacco and alcohol use and co-use in greater depth, including considering the evolving marketplace of both tobacco and alcohol products.
2. A better understanding of the cancer risks of tobacco and alcohol co-use, especially for cancers in addition to those of the head and neck and esophagus (for which synergistic risk is already known). Attention is needed to the potential for synergistic risks, along with a better understanding of the biological mechanisms underlying these risks.
3. Although there are effective treatments for tobacco use and alcohol use and/or use disorder individually, few interventions have been developed to treat co-use. Given the large number of people who use both substances, this is an important area of continued study.
4. Better evidence concerning integrated interventions (e.g., program, policy, public education, clinical) to prevent tobacco and alcohol use and co-use among adolescents and young adults is urgently needed to advance cancer prevention and public health.

The monograph's key findings and conclusions will also inform public health practitioners, clinicians, and the public. For example, because alcohol use, especially heavy alcohol use, is a barrier to successful smoking cessation, people trying to quit smoking should be advised and assisted to reduce or stop their alcohol use. Additionally, providing smoking cessation treatment during treatment for alcohol use disorder does not threaten sobriety outcomes; this is important because a high percentage of people entering treatment for alcohol use disorder or substance abuse disorder also smoke cigarettes. However, many substance use treatment facilities have yet to incorporate tobacco cessation services into their program offerings, and there are many opportunities, including smokefree policies and integrated treatment systems, to implement these systems of care.

NIDA, NIAAA, and NCI have supported research to reduce the morbidity and mortality caused by tobacco and alcohol products for many decades, and success in tobacco control has dramatically reduced the burden of cancer caused by tobacco. However, more work is needed to achieve comprehensive tobacco control and relatively little progress has been made in addressing

tobacco and alcohol co-use or addressing alcohol alone as a target for cancer prevention and control. Notably, more effort is needed to address barriers between the fields—both with regards to individual substances and to research domains—to better understand the complex patterns and determinants of substance use among both youth and adults. This monograph is an important step to better integrate work to address tobacco and alcohol use, which are among the nation's most commonly used hazardous and addictive substances. Advancing research on the co-use of tobacco and alcohol will help shape effective interventions to mitigate the combined impact of these addictive products on cancer incidence and mortality.

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Table of Abbreviations and Acronyms

Abbreviation/Acronym	Definition
ABV	Alcohol by volume
ADH	Alcohol dehydrogenase
aHR	Adjusted hazard ratio
AI/AN	American Indian and Alaska Native
AICR	American Institute for Cancer Research
ALDH	Aldehyde dehydrogenase
aOR	Adjusted odds ratio
AUD	Alcohol use disorder
BAC	Blood alcohol content
BRFSS	Behavioral Risk Factor Surveillance System
CBT	Cognitive behavioral therapy
CDC	U.S. Centers for Disease Control and Prevention
CI	Confidence interval
COVID-19	Coronavirus disease 2019
CYP2A6	Cytochrome P450 2A6
CYP2E1	Cytochrome P450 2E1
DNA	Deoxyribonucleic acid
DSM	Diagnostic and Statistical Manual of Mental Disorders
EMA	Ecological momentary assessment
FDA	U.S. Food and Drug Administration
FSPTCA	Family Smoking Prevention and Tobacco Control Act
FTC	U.S. Federal Trade Commission
ENDS	Electronic nicotine delivery system
GWAS	Genome-wide association study
HED	Heavy episodic drinking
HID	High-intensity drinking
HINTS	Health Information National Trends Survey
HNSCC	Head and neck squamous cell carcinoma
HIV	Human immunodeficiency virus
HR	Hazard ratio
HTP	Heated tobacco product
HPV	Human papillomavirus
IARC	International Agency for Research on Cancer
MLDA	Minimum legal drinking age

Abbreviation/Acronym	Definition
MSA	Master Settlement Agreement
MTF Study	Monitoring the Future Study
nAChR	Nicotinic acetylcholine receptors
NAS	National Alcohol Survey(s)
NCI	National Cancer Institute
NESARC	National Epidemiologic Survey on Alcohol and Related Conditions
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NIDA	National Institute on Drug Abuse
NIH	National Institutes of Health
NNK	Nicotine-derived nitrosamine ketone
NNN	N'-nitrosonornicotine
NMR	Nicotine metabolite ratio
NSDUH	National Survey on Drug Use and Health
NRT	Nicotine replacement therapy
NYTS	National Youth Tobacco Survey
OR	Odds ratio
PATH	Population Assessment of Tobacco and Health
PHS	Public Health Service
RCT	Randomized controlled trial
RR	Relative risk or risk ratio
RTD	Ready-to-drink
SAMHSA	Substance Abuse and Mental Health Services Administration
SEER	Surveillance, Epidemiology, and End Results (program)
SES	Socioeconomic status
SNP	Single nucleotide polymorphism
SUD	Substance use disorder
T21	Tobacco 21 (law)
TUD	Tobacco use disorder
TUS-CPS	Tobacco Use Supplement to the Current Population Survey
U.S.	United States of America
USDA	U.S. Department of Agriculture
USDHHS	U.S. Department of Health and Human Services (HHS)
USPSTF	United States Preventive Services Task Force
WCRF	World Cancer Research Fund

Abbreviation/Acronym	Definition
WHO	World Health Organization
YRBS	Youth Risk Behavior Survey

Glossary

Term	Definition
Alcohol	A chemical substance found in drinks such as beer, wine, and liquor. It is also found in some medicines, mouthwashes, household products, and essential oils (scented liquid taken from certain plants). It is made by a chemical process called fermentation that uses sugars and yeast. There are different types of alcohol. The type used to make alcoholic drinks is called ethyl alcohol (ethanol).
Alcohol by volume (ABV)	A standard measure of how much alcohol (ethanol) is contained in a given volume of a drink. The amount of alcohol in a container is shown as a percentage of the overall volume of the drink.
Alcohol use disorder	A medical condition characterized by an impaired ability to stop or control alcohol use despite adverse social, occupational, or health consequences. It encompasses the conditions that some people refer to as alcohol abuse, alcohol dependence, alcohol addiction, and the colloquial term alcoholism. Considered a brain disorder, alcohol use disorder can be mild, moderate, or severe.
Beer	Fermented beverage using cereal grains, commonly malted barley. The alcohol by volume for beers typically ranges between 3% to 6%. Some beers have 15% or more alcohol by volume.
Cider	Fermented beverage using fruits. The alcohol by volume for ciders ranges from 4% to 8%.
Electronic nicotine delivery systems (ENDS)	A rapidly changing class of tobacco products known by many different names, including e-cigarettes, e-cigs, vapes, mods, and tank systems. ENDS deliver an aerosol to the user that typically contains nicotine, propylene glycol, vegetable glycerin, and flavoring chemicals.
Liquor	Fermented beverages that include two broad categories—liqueurs and distilled spirits. Liqueurs are flavored and sweetened beverages with a typical alcohol by volume range of 15% to 30%. Distilled spirits have a typical alcohol by volume range of 35% to 55%.
Nicotine	An addictive, poisonous chemical found in tobacco. It can also be made in the laboratory. When it enters the body, nicotine causes an increased heart rate, an increased use of oxygen by the heart, and a sense of well-being and relaxation.
Nicotine replacement therapy	A type of treatment that uses special products to give small, steady doses of nicotine to help stop cravings and relieve symptoms that occur when a person is trying to quit smoking. These products include nicotine gums, nicotine inhalers, nicotine nasal sprays, nicotine lozenges, and nicotine patches, and some are available without a prescription. They do not contain any of the other chemicals found in tobacco products.
Pack year	A standard measure of the amount a person has smoked over a period. It is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.
Ready-to-drink beverages (RTDs)	Alcoholic beverages often mixed with soft drinks. RTDs are sometimes referred to as “alcopops” or “flavored alcoholic beverages,” with their alcohol by volume ranging from 3% to 7%.
Smoking	Cigarette use.
Smoking cessation treatment	Treatment aimed at smoking reduction, smoking cessation, and relapse prevention after treatment.
Tobacco use	The use of tobacco products including cigarettes, cigars, hookah, ENDS, and smokeless tobacco. In this monograph, tobacco use refers to the use of commercial tobacco and not the sacred and traditional use of tobacco by some American Indian communities.
Wine	Fermented beverage using grapes. Includes red wines, white wines, rose wines, sparkling wines, and fortified wines. The ABV for wines varies widely, with most ranging from 8% to 14%.

NCI Monograph 24
Tobacco and Alcohol: Understanding Co-Use to Reduce Cancer Incidence and Mortality

Chapter 1
Introduction and Overview

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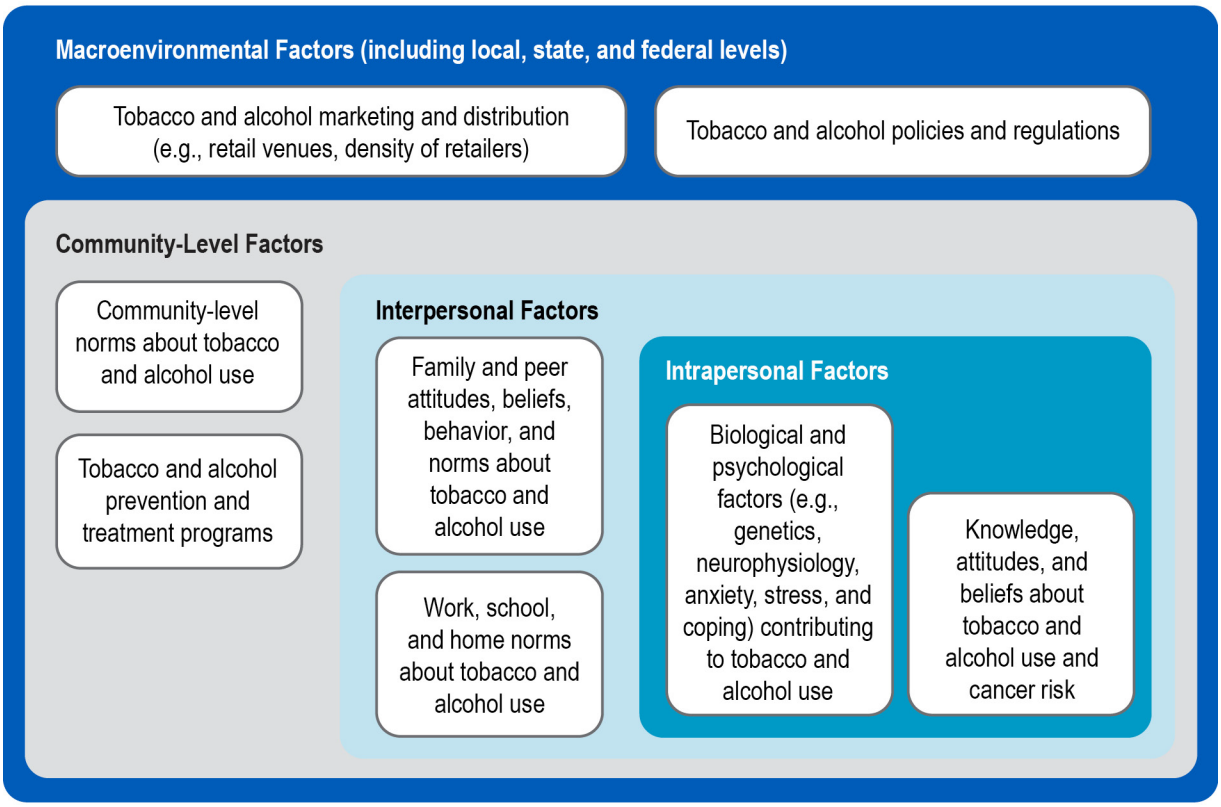
Chapter 1
Introduction and Overview

Introduction

Tobacco and alcohol are two of the most widely used addictive and carcinogenic substances in the United States.[1,2] Nicotine, found in tobacco, and ethanol (also known as ethyl alcohol or grain alcohol) have psychoactive effects and can create strong dependence, especially when used in high doses.[3-5] Importantly, all tobacco products contain, or generate, many carcinogenic compounds when used as intended. Combusted tobacco products—such as cigarettes, cigars and pipes—pose the greatest cancer risks due to exposure to carcinogens in the product and additional exposure to the diverse toxicants produced by combustion.[6,7] For alcohol products, exposure to the main psychoactive and addictive ingredient, ethanol, and its metabolite acetaldehyde, are the major cause of alcohol-induced carcinogenesis.[8] Separately and together, tobacco and alcohol have enormous effects on the U.S. burden of disease.[1,9] Tobacco use has long been the primary modifiable risk factor for cancer incidence and mortality in the United States.[7] In 2019, tobacco use was responsible for an estimated 344,000 cancer cases and almost 170,000 cancer deaths.[1] In 2019, alcohol use was the third leading cause of cancer, responsible for almost 97,000 cancer cases and more than 24,000 cancer deaths.[1] Tobacco and alcohol co-use, defined as the use of both substances within a given time frame (e.g., same hour, same day, past month), is of particular concern for tobacco control, reducing the harms of tobacco and alcohol, and improving cancer control.

Together, tobacco and alcohol account for almost 25% of cancer cases in the United States, more than one-half of all cancer cases attributed to modifiable risk factors,[1] making their use—and the factors influencing their use—primary targets for cancer prevention and control. When considering all causes of death (i.e., not just cancer) worldwide, tobacco and alcohol are leading preventable risk factors. Globally, more than 7 million people die from tobacco use every year and 2.6 million die from alcohol use.[10,11] Tobacco and alcohol are often used separately, as well as in complex patterns of co-use. Use patterns are influenced by numerous intersecting factors, including intrapersonal (biological and psychological), interpersonal (family, peer), and social, (community) as well as macro-environmental factors such as the policies, systems, and environments that shape accessibility, affordability, and marketing of these substances (represented in Figure 1.1). Understanding how tobacco and alcohol use intersect across these factors is essential for enhancing cancer prevention, preventing tobacco use and alcohol use, and supporting tobacco cessation and the reduction or cessation of alcohol use.

Figure 1.1 Multilevel Factors Affecting Tobacco and Alcohol Co-Use



Individuals often use both tobacco and alcohol during the same time period, and co-use often extends across the lifespan.[12,13] Co-use of these substances has been shown to contribute to cancer risk, including synergistic risk for head and neck cancers.[14-16] Furthermore, co-use of tobacco and alcohol may influence the probability of continued use and present a barrier to successful tobacco cessation and reduction in harmful drinking.[17,18] As discussed in chapters 2 and 3 of this volume, the overall population prevalence of tobacco and alcohol co-use has declined as overall tobacco use has declined; however, associations between tobacco and alcohol use have persisted, particularly among people with heavier use of both substances and those with an alcohol use disorder.[12,19-25] These associations are observed across demographic groups that differ in the overall prevalence of tobacco and/or alcohol use. Despite dramatic reductions in overall tobacco use[13] and declines in drinking among adolescents and adults,[2,26] the burden of cancer and other chronic diseases caused by tobacco and alcohol use and co-use remains extremely high.[1,27] Continued investment in research and public health measures addressing tobacco and alcohol use is needed to reduce this burden.

The Need for a Monograph on Tobacco and Alcohol Co-Use

Both tobacco and alcohol are known causes of cancer,[28,29] and reducing their use and co-use is vital for cancer prevention and control. The primary goal of this monograph is to synthesize research to provide insight into the state of the science concerning tobacco and alcohol co-use. Specifically, the monograph is intended to advance cancer prevention efforts by summarizing the evidence on tobacco and alcohol co-use and identifying research needs. Multiple levels are

considered, including individual use and co-use, cancer etiology, biological mechanisms underlying tobacco and alcohol use, interventions for prevention and cessation, and the social, policy and economic environment in which these factors occur. Figure 1.1 portrays these multilevel biological, psychological, and social processes that influence tobacco and alcohol use and co-use, their health consequences, and interventions addressing different aspects of these processes. The focus of this volume is to better understand the relationships and synergies between tobacco and alcohol co-use from a health, treatment, and prevention perspective. Achieving this goal will contribute to the overall health and well-being of the nation.

The monograph is primarily aimed at informing researchers. However, others seeking to understand and reduce the harms of tobacco and alcohol use and improve cancer control will find this volume useful. Diverse entities, including health departments, funding agencies, community-based organizations, those working with adolescents and young adults, substance use treatment specialists, health care providers, and health systems, will also be able to use the volume to inform their work and point them towards promising new directions for tobacco and alcohol co-use interventions.

Effects of Tobacco and Alcohol Co-Use

Based on pooled data from the 2015–2019 National Survey on Drug Use and Health, an estimated 11.8% (95% confidence interval [CI], 11.7%–12.0%) of U.S. adults (approximately 30 million in total) both smoked cigarettes and drank alcohol in the past month, and people who smoked in the past month were more likely to report binge drinking or drinking >7/14 drinks per week for women/men than people who never or formerly smoked (described in more detail in chapter 2). People who smoke and drink alcohol represent a significant subpopulation for whom intervention is an important, but often overlooked, goal. There is a strong correlation between alcohol use and nicotine dependence. Individuals who are alcohol dependent are three times more likely to have nicotine dependence than the general population, and individuals who are dependent on tobacco are nearly four times more likely to be alcohol dependent than people without nicotine dependence.[30] Furthermore, people who smoke and are alcohol dependent or report heavy drinking have more severe nicotine dependence and experience greater difficulty quitting smoking than people who smoke and do not drink heavily,[31,32] and lapses during quit attempts occur more frequently when individuals are drinking alcohol, especially during heavy drinking days.[33] A growing body of literature also highlights the adverse consequences associated with tobacco and alcohol co-use, including less frequent use of both substances. People who smoke tobacco and drink heavily have worse health outcomes for conditions such as cancer and cardiovascular disease than people who use either tobacco or alcohol alone,[15,34,35] and have a faster cognitive decline relative to people who drink moderately and do not smoke.[36] Although both tobacco and alcohol broadly share some common pathways to morbidity and mortality (e.g., accumulated chronic exposure resulting in carcinogenic processes, damage to organ systems),[7,37,38] important differences exist. Deaths caused by tobacco primarily occur in middle- and old-age, and are largely due to cancer, heart disease and stroke, and respiratory disease, among other causes.[7] Deaths caused by alcohol occur at younger ages and in different ways than deaths caused by tobacco.[39] The U.S. Centers for Disease Control and Prevention estimates that about two-thirds of alcohol-related deaths were due to chronic conditions (e.g., cancer, heart disease, liver disease, and alcohol use disorder), while one-third of alcohol-related deaths result from a specific occasion of binge or excessive drinking,

including motor vehicle crashes, alcohol-involved substance overdoses, alcohol poisonings, and deaths by suicide.[40] However, the probability of binge or excessive drinking is also related to alcohol use disorder and the drinking history of individuals.[41,42] substance overdoses, alcohol poisonings, and deaths by suicide.[40-42]

The harms of tobacco and alcohol use are not limited to the individuals who use these products. Exposure to secondhand smoke is well-established to increase risk of cancer and other negative health outcomes related to tobacco.[7] Alcohol use problems can also result in social, economic, and psychological harms to coworkers, families, friends, and social connections. These include job losses or other consequences, motor vehicle crashes, unintentional injuries, sexually transmitted infections, unintended pregnancies, and family breakdowns.[43] These effects provide additional strong motivation to address tobacco and alcohol use and co-use.

Cancer Risk and Mortality

Both tobacco and alcohol increase the risk of cancer at multiple sites and their combined use results in synergistic increases in risk for head and neck cancers.[28,29] In the United States, tobacco use was responsible for an estimated 19.3% of cancer cases and 28.5% of cancer deaths in 2019, while alcohol was responsible for an estimated 5.4% of cancer cases and 4.1% of cancer deaths.[1] This monograph reviews the conclusive evidence that tobacco and alcohol exposures independently and jointly cause multiple types of cancer through a wide variety of distinct and overlapping mechanisms. The carcinogenic mechanisms associated with tobacco use vary by the type of tobacco product used, with combusted tobacco products generally resulting in greater exposure to toxicants than other tobacco products. Notably, it is also well established that smokeless tobacco use causes several cancers, including oral, head, and neck cancers.[6,7] Combustion of tobacco produces thousands of harmful, mutagenic, and carcinogenic products, such as polycyclic aromatic hydrocarbons (PAHs), N-nitrosamines, aromatic amines, 1,3-butadiene, benzene, aldehydes, and ethylene oxide. It is also well-established that cigarette smoke contains at least 80 known carcinogens.[44] The 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*, concluded that cigarette smoking causes various forms of cancer, including (but not limited to) cancers of the oral cavity, pharynx, larynx, esophagus, lung (including trachea and bronchus), stomach, liver, pancreas, kidney and ureter, bladder, cervix, colorectum, and blood (acute myeloid leukemia).[7] In 2020, the report *Smoking Cessation: A Report of the Surgeon General* reaffirmed the cancer risks of smoking and highlighted the benefits of cessation on cancer and other health outcomes.[5]

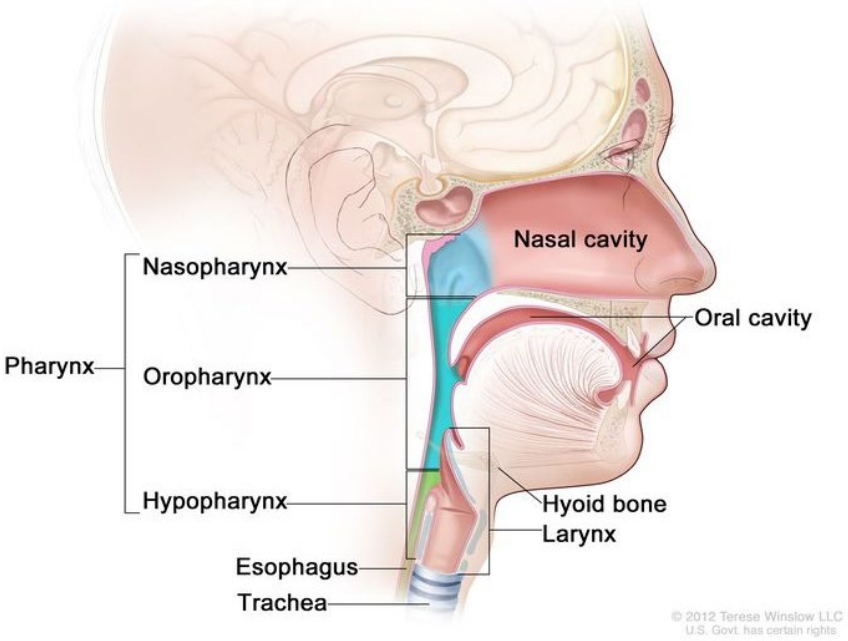
Alcohol acts as a carcinogen through multiple mechanisms. All alcoholic beverages contain ethanol, the major cause of cancer associated with alcohol use through its direct physiological effects and via its primary metabolite, acetaldehyde, a potent carcinogen.[38] The carcinogenicity of alcoholic beverages and of ethyl carbamate (urethane), a frequent contaminant of fermented foods and alcoholic beverages, especially distilled spirits, was the topic of the 1988 and 2010 International Agency for Research on Cancer (IARC) Monographs.[28,45] The 2010 IARC Monograph, *Evaluation of Carcinogenic Risks to Humans: Alcohol Consumption and Ethyl Carbamate (Volume 96),* noted that the consumption of alcohol is one of the top 10 risks for worldwide burden of disease. There is a strong scientific consensus that alcohol use causes oral cavity, pharynx (throat), esophageal, liver, larynx (voice box), female breast, and colorectal cancer.[28] Additionally, inverse associations have been observed for kidney cancer,[28] thyroid

cancer,[46] and non-Hodgkin lymphoma (see chapter 4).[28] Nevertheless, the reduced number of cases of these cancers that might be attributable to alcohol consumption are much lower than the increased number of cancers observed at other sites. For both tobacco and alcohol, the risk of cancer increases as the amount consumed increases. With regard to alcohol, for certain cancers, like female breast, oral cavity, and throat cancers, this risk may increase around one or fewer drinks per day.[38] In 2024, IARC reviewed the evidence that reductions in alcohol use lead to reduced risk of cancer. They concluded that there was sufficient evidence that alcohol reduction or cessation reduces oral and esophageal cancer risk; limited evidence for laryngeal, colorectal, and female breast cancer risk; and inadequate evidence for pharyngeal and liver cancer risk.[47,48] For cancers for which the evidence was limited or inadequate, few high-quality studies have been carried out, indicating the need for further epidemiological research.

Key Findings From Previous Reports Related to Tobacco and Alcohol Co-Use and Health

Research on the use of tobacco and alcohol has identified synergistic effects of the use of both substances on cancer risk. A synergistic relationship between tobacco and alcohol use and cancer risk has been observed for cancers of the head and neck (oral cavity, larynx, pharynx) and esophagus; the anatomy of the oral cavity, pharynx, and larynx is depicted in Figure 1.2. These findings, as well as conclusions for other cancer sites, are summarized in Tables 1.1 and 1.2. Acetaldehyde, the primary metabolite of ethanol, is also one of the main volatile compounds and carcinogens present in tobacco smoke. Acetaldehyde damages DNA in multiple ways, interfering with DNA synthesis and repair, causing point mutations, and damaging chromosomes.[49] Consumption of both tobacco and alcohol products results in increased exposure to acetaldehyde.[6,28,29,45] Importantly, the oral microbiome plays a role in the rapid conversion of ethanol to acetaldehyde, increasing exposure to this compound in the head and neck. Alcohol consumption also may result in increased absorption and increased activation of the carcinogens present in tobacco products, serving as the basis for a synergistic effect on cancer risk.[38,50-52] Chapter 4 of this monograph reviews additional and more recent epidemiological literature to summarize what is known as of 2026 about the synergistic effects of the use of tobacco and alcohol on cancer risk, including among different populations and by tobacco and alcohol product type.

Figure 1.2 Anatomy of the Oral Cavity, Pharynx, and Larynx



Source: National Cancer Institute 2025.[53]

Table 1.1 Findings about the Health Effects of Tobacco and Alcohol Use from Key Reports

Findings from the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*	
The evidence is sufficient to infer a causal relationship between smoking and the following cancers—	• Acute myeloid leukemia • Bladder • Cervix • Colorectal* • Esophageal • Kidney and ureter • Laryngeal • Liver* • Lung (including trachea and bronchus) • Ovary • Oral cavity and pharyngeal • Pancreatic • Stomach
In cancer patients, the evidence is sufficient to infer a causal relationship between cigarette smoking and—	• Adverse health outcomes • Increased all-cause mortality and cancer-specific mortality • Increased risk for second primary cancers known to be caused by cigarette smoking, such as lung cancer
The evidence is suggestive, but not sufficient, to infer a causal relationship between—	• Tobacco smoke and breast cancer* • Active smoking and breast cancer* • Exposure to secondhand tobacco smoke and breast cancer*
In patients with prostate cancer, the evidence is suggestive of—	• Higher risk of death from prostate cancer in smokers than in nonsmokers* • Higher risk of advanced-stage disease and less well-differentiated cancer in smokers than in nonsmokers*

Table 1.1 (continued)

Findings from the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*	
The evidence is inadequate to infer the presence or absence of a causal relationship between smoking cigarettes and—	• Ovarian cancer
The evidence is suggestive of no causal relationship between smoking and—	• Adult brain cancer in men and women • Risk of incident prostate cancer
Findings from the 2010 IARC Monograph, *Evaluation of Carcinogenic Risks to Humans: Alcohol Consumption and Ethyl Carbamate (Volume 96)*	
There is sufficient causal evidence of an association between alcohol consumption and risk of cancer of the—	• Oral cavity and pharynx • Larynx • Esophagus • Liver • Female breast • Colon and rectum

Note. An asterisk (*) indicates a new conclusion from the 2014 report. Statements without an asterisk were made in the 2004 Surgeon General's reports and reaffirmed in the 2014 report.
Source: IARC 2010,[28] USDHHS 2014.[7]

Table 1.2 Summary of Findings on Independent and Synergistic Cancer Risk Related to Tobacco Smoking and Alcohol

Cancer site	Cancer caused by tobacco use	Cancer caused by alcohol use	Evidence of synergy
Esophagus	Yes	Yes	Yes
Larynx	Yes	Yes	Yes
Oral cavity and pharynx	Yes	Yes	Yes
Colon and rectum	Yes	Yes	-
Liver	Yes	Yes	-
Stomach	Yes	Yes	-
Bladder	Yes	-	-
Blood (acute myeloid leukemia)	Yes	-	-
Cervix	Yes	-	-
Kidney and renal pelvis	Yes	*	-
Lungs, bronchus, trachea	Yes	-	-
Pancreas	Yes	-	-
Breast (female)	-	Yes	-

Note. Findings are drawn from IARC monographs, World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) Continuous Update Project, and Surgeon General's reports. Cancers are presented in order of evidence of synergy (caused by tobacco and alcohol), caused by tobacco but not alcohol, caused by alcohol but not tobacco, or caused by neither; they are then listed alphabetically within those clusters. A dash (-) indicates that as of 2026, there is no conclusive evidence regarding a causal relationship with tobacco or alcohol use or synergy related to tobacco and alcohol use. An asterisk (*) indicates inverse association.
Source: IARC 2010,[28] IARC 2012,[29] IARC 2024,[48] USDHHS 2014,[7] WCRF/AIRC 2018.[54]

Awareness of Cancer Risk Related to Tobacco and Alcohol Co-Use

As leading modifiable risk factors for cancer in the United States, it is important for the public to be aware of the risk of tobacco and alcohol use and co-use to prevent and reduce cancer-related morbidity and mortality. Greater awareness of cancer-related risk could influence public attitudes toward alcohol use and help people make more informed decisions about their behavior. Although the vast majority of the public are aware that smoking cigarettes causes cancer,[55] awareness of the link between alcohol use and cancer is much lower, albeit increasing in recent years.[56-58] Data from the 2022 Health Information National Trends Survey (HINTS) found that only 40.6% of U.S. adults recognized that alcohol consumption increases cancer risk.[57] In addition, data from the 2020 HINTS survey showed that perceptions of an alcohol-cancer link were higher for liquor (31.2%) than for beer (24.9%) or wine (20.3%),[59] despite epidemiological evidence suggesting that the total amount of ethanol consumed determines cancer risk rather than differences between beverage types.[38] Other nationally representative surveys, such as the American Institute for Cancer Research's Cancer Risk Awareness Survey, also demonstrate that less than half of the U.S. population is aware that alcohol use is a risk factor for cancer, whereas 89% are aware that tobacco use causes cancer.[60] Further, analysis of 2014 Medical Expenditure Panel Survey data found that only 41.0% of adults ages 35 and older received screening and counseling for alcohol misuse from a healthcare provider.[61] In January 2025, the U.S. Surgeon General released an advisory on alcohol and cancer risk, highlighting the causal link between alcohol use and increased risk for several cancers.[38] Surveys of U.S. adults conducted before and after the advisory was released found that, as of February 2025, 56% of respondents said that regular consumption of alcohol increases your chances of later developing cancer, an increase from 40% in September 2024.[56] Given the large number of people who use both substances, increasing awareness of the link between cancer and tobacco and alcohol co-use is an important aspect of making progress on addressing these modifiable risk factors for cancer. However, although awareness of the cancer related risks of tobacco and alcohol co-use and alcohol consumption is an important part of behavior change, it is vital to invest in prevention and treatment interventions and policy approaches to achieve reductions in prevalence.

About This Monograph

This monograph is the 24th volume in the series of monographs on tobacco control produced by the National Cancer Institute (NCI) of the National Institutes of Health, an agency of the U.S. Department of Health and Human Services. Other recent topics addressed as part of the Tobacco Control Monograph Series include treating people with cancer who smoke (volume 23), health disparities caused by tobacco (volume 22), the economics of tobacco control (volume 21), genetic studies of nicotine use and dependence (volume 20), and the role of the media in promoting and reducing tobacco use (volume 19).

Preparation of the Monograph

This monograph underwent a rigorous development process led by five senior editors forming the Scientific Editorial Committee (SEC). The senior editors were selected based on their expertise in tobacco control; alcohol research; health behavior research; genetics of tobacco and alcohol addiction; clinical treatment of tobacco, alcohol, and other substance use; and tobacco control policy research. The SEC developed a shared vision of the monograph's purpose and focus. Given responsibility for specific topics, SEC members were joined by chapter leads to

develop chapter outlines; identify chapter contributors and reviewers; and contribute to the development, writing, reviewing, and editing of the monograph. Chapter leads and contributors drafted chapters in accordance with the outlines and under the guidance of the SEC. The individuals who contributed to this monograph are listed on pages xxiii–xxix. Across chapters, literature searches and expert knowledge were used to identify important sources, including peer-reviewed articles and existing reviews of the literature, but new systematic reviews were not carried out. Searches were generally restricted to studies conducted in the United States and those in English and specified a relevant age range depending on the topical domain (e.g., adults 18 years and older or adolescents and young adults <25 years). Additionally, across chapters and depending on the topic, international literature was reviewed, particularly where no U.S.-based evidence was available. Each chapter within the volume provides an overview of the literature search strategies that were conducted for this monograph and for the specific chapter or subsection within a chapter. In addition, new analyses of nationally representative survey data (Monitoring the Future, National Alcohol Surveys, and National Survey on Drug Use and Health) were conducted to characterize patterns of tobacco and alcohol co-use among adults and adolescents in the United States. Chapters 2 and 3 provide details regarding the measures and approach used for these analyses.

In addition to internal reviews by the editorial team, each chapter was reviewed by multiple external expert peer reviewers, followed by extensive expert peer review of the full monograph volume. NCI also conducted a final review of the monograph before publication. In all, 51 experts reviewed all or part of the monograph.

Monograph Organization

The monograph is organized into five major topic areas pertaining to the intersection of tobacco and alcohol: the surveillance and behavioral epidemiology of tobacco and alcohol use (chapters 2 and 3); the epidemiology of tobacco and alcohol use as it relates to cancer risk (chapter 4); the physiological, cellular, molecular, and genetic basis of addiction and carcinogenesis as they relate to the co-use of tobacco and alcohol (chapters 5 and 6); tobacco use treatment and prevention in the context of alcohol use (chapters 7 and 8); and the economic, environmental, and policy factors affecting both tobacco and alcohol use and co-use (chapter 9). The final chapter, 10, provides conclusions for the monograph and further highlights important research needed to help reduce the harms of tobacco and alcohol use and co-use.

The monograph comprises the following chapters:

Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Adolescents and Adults in the United States. Chapter 2 describes measures of tobacco and alcohol co-use in the United States and provides details on patterns of co-use from the literature and from new analyses of National Alcohol Surveys (NAS) and National Survey on Drug Use and Health (NSDUH) data. This chapter also considers the most appropriate measures of tobacco and alcohol co-use for public health surveillance.

Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Population Subgroups in the United States. Chapter 3 provides a summary of tobacco and alcohol co-use in specific population subgroups of adults based on a review of the literature and new analyses of NAS and NSDUH data. The chapter reviews tobacco and alcohol co-use among groups by race and/or ethnicity, educational attainment, income, geographic location, sexual minority status, veteran status, and presence of comorbidities including cancer, chronic health conditions, HIV, mental health conditions, and pregnancy.

Cancer Epidemiology of Tobacco and Alcohol Co-Use. Chapter 4 summarizes the epidemiologic research examining associations between cancer risk and use of tobacco and alcohol products independently, as well as the synergistic risk of cancer with use of both types of products resulting in a higher risk of cancer in specific sites. This chapter also evaluates available data on the synergistic effects of tobacco and alcohol use on cancer risk by demographic subgroups and efforts to examine the type of tobacco and alcohol product used to better understand their individual and joint effects on cancer risk.

Physiological, Cellular, Molecular, and Genetic Basis of the Combined Effects of Tobacco and Alcohol Use on Carcinogenesis. Chapter 5 provides an overview of mechanistic interactions between tobacco and alcohol exposures, focusing on how alcohol may influence the main mechanisms of tobacco carcinogenesis. It reviews the evidence for genetic susceptibility to tobacco and alcohol use and co-use and describes how this influences mechanisms for carcinogenesis. Other topics include the limited literature on interaction of nicotine and alcohol as carcinogens and evidence supporting the reversibility of these mechanisms for carcinogenesis and, consequently, the beneficial effects of tobacco use cessation and alcohol use reduction or cessation.

Shared Physiological and Genetic Factors Contributing to Tobacco and Alcohol Co-Use. Chapter 6 describes overlapping genetic factors and neurophysiology contributing to co-use of tobacco and alcohol. The topics reviewed in the chapter highlight the need to understand both environmental and genetic factors that contribute to the development of dependence and disease.

Treatment Approaches for Tobacco and Alcohol Use. Chapter 7 reviews the evidence on approaches to treating tobacco and alcohol use concurrently and the effectiveness of pharmacological and behavioral treatments for concurrent cigarette smoking and alcohol use. Topics reviewed include the neurobiological and behavioral overlap between smoking and alcohol consumption, highlighting key mechanisms that drive co-use and could inform treatment targets, and the use of individual and combined pharmacological and behavioral therapies to treat tobacco and alcohol co-use.

Prevention of Tobacco Use in Relation to Alcohol. Chapter 8 summarizes the evidence regarding the extent to which alcohol-focused and more general substance use prevention programs are effective in preventing tobacco use among adolescents and young adults. It also evaluates the extent to which tobacco-focused and more general substance use preventions are

effective in preventing alcohol use. Topics include psychosocial prevention programs and policies delivered at the individual level (e.g., text-based programs), group level (e.g., school-based and family-based programs), and population level (e.g., minors' access policies, mass media and social media) that focus on tobacco, alcohol, and multisubstance use prevention where tobacco use is a measured outcome.

Economic, Environmental, and Policy Factors Related to Tobacco and Alcohol Use. Chapter 9 reviews the evidence on the influence of economic, environmental, and policy factors for tobacco and alcohol use on tobacco initiation, use, and cessation. The chapter describes how the retail environment for tobacco and alcohol (which includes quantity, type, and location of tobacco and alcohol retailers), and the marketing environment for tobacco and alcohol may interact to influence tobacco use and tobacco and alcohol co-use. It also reviews the evidence related to tobacco and alcohol policy effects on co-use, such as (1) the interaction of tobacco and alcohol taxes on tobacco and alcohol use behavior, (2) the influence of restricting alcohol availability on tobacco use, (3) the effect of smokefree bar policies on alcohol use.

Future Research Directions. Chapter 10 summarizes the key findings from this report and identifies priority areas for future research. The chapter highlights the challenges and opportunities in understanding and addressing how alcohol use influences tobacco use initiation and progression, and the resulting increased cancer risk.

Key Terminology and Concepts

Identifying perfectly aligned and appropriate terminology across two domains of substance use is challenging as tobacco and alcohol have their own research and treatment contexts. The monograph is purposeful in its choices of the terms and concepts used throughout to be consistent with the literature as of 2026 and to avoid stigmatizing language.[62] Key terms used in the monograph and their definitions are highlighted in the "Other Terms" section below.

Tobacco and Alcohol Treatment Terminology

The Diagnostic and Statistical Manual of Mental Disorders (DSM) is the standard classification of psychiatric disorders used for clinical, research, policy, and reimbursement purposes in the United States. This important volume has widespread influence on how such disorders are diagnosed, treated, and investigated. Since the publication of the first DSM in 1952, knowledge about psychiatric disorders, including substance use disorders, has advanced greatly.[63]
Over time, there have been changes in how the DSM—and thus how clinicians, counselors, and researchers—identifies and treats people who use tobacco and alcohol (and people who use both). The most recent edition, DSM-5-TR, was published in 2022.[64] The classification of substance use disorders in the DSM-5-TR combines both the abuse and dependence criteria from the DSM-IV into a single diagnostic tool for substance use disorder and does not use the terms "abuse" and "dependence"; instead, it indicates a level of severity of substance use disorder as mild, moderate, or severe depending on how many criteria are endorsed by the person.[63]
The substance use disorder diagnosis can apply to people who use both tobacco and alcohol as well as use other substances.

Tobacco and alcohol use disorders are each considered a form of substance use disorder, but researchers and clinicians do not always use the terms "alcohol use disorder" or "tobacco use disorder" to describe treatment for people who use tobacco and alcohol. In the tobacco use treatment literature, terms describing both treating use and dependence are varied and often used interchangeably. For example, the treatment may be described as for "nicotine addiction," "nicotine dependence," "tobacco dependence," "tobacco use," "tobacco dependence" or "tobacco use disorder." The DSM-IV used the term "nicotine dependence," whereas the DSM-5-TR replaced "nicotine dependence" with the diagnosis "tobacco use disorder." In this monograph, treatment of tobacco use, dependence and use disorder are described with the terms used in the cited studies. This reflects the terminology used most often in research literature and in the most recent (2008) Public Health Service Clinical Practice Guideline, *Treating Tobacco Use and Dependence*,[65] which is widely used by both clinicians and researchers.

As is seen in the field of tobacco research and treatment, alcohol researchers and clinicians employ a variety of terms to characterize alcohol use problems and their treatment.[62] The terms have changed over time and are not used consistently. In the alcohol use treatment literature, the terms often reflect the criteria and terminology of the DSM, reflecting the more severe continuum of alcohol use and harms. The DSM-5-TR uses the term "alcohol use disorder" (AUD), characterizing the diagnosis by its level of severity (mild, moderate, or severe). Previous versions of the DSM used the terms "alcohol abuse" or "alcohol dependence", and these terms are included in this monograph when the cited studies used older DSM-IV criteria for alcohol abuse or dependence as primary outcomes or eligibility criteria.

Other Terms

Where possible, this monograph uses standardized terms to define tobacco and alcohol use. Some definitions of standardized terms are as follows:

- **Dependence** – A condition marked by physical withdrawal symptoms and continued use despite known adverse consequences to the user (e.g., physical and mental health, social).[43]
- **Co-use** – Use of two substances (e.g., tobacco and alcohol) within the same reference period, such as within the past hour, day, week, month, or year. A subcategory of co-use may be referred to as simultaneous use, which is the coadministration of two substances in the same product, at the same time, or on the same occasion. U.S. national health surveys most commonly assess use of tobacco and alcohol in the past month and/or past year, and co-use is analyzed from these separate variables. Co-use is sometimes called "concurrent" use.
- **Standard drink** – In the United States, a standard drink is defined as any beverage containing 0.6 fluid ounces or 14 grams of ethanol.[66] A standard drink is equivalent to 12 fluid ounces of beer at 5% alcohol by volume, 5 fluid ounces of wine at 12% alcohol by volume, or 1.5 fluid ounces of distilled spirits at 40% alcohol by volume. Throughout this monograph the term "drink" refers to a standard drink as defined here unless otherwise specified.[67]
- **Heavy drinking** – Consumption of 5 or more drinks on any day or 15 or more per week for men and consumption of 4 or more drinks on any day or 8 or more drinks per week for women.[66]

- **Synergy** – When the interaction between two factors (i.e., tobacco and alcohol use) results in a total effect (e.g., cancer risk) that is different from the sum of the individual effects. Synergy may be positive or negative, and additive or multiplicative.[68,69]
- **Tobacco use** – In this monograph, tobacco use or tobacco smoking refers to commercial tobacco products including cigarettes, e-cigarettes, smokeless tobacco, pipe tobacco, cigars, hookah, heated tobacco products, and nicotine pouches. It does not refer to the sacred and traditional use of tobacco by some American Indian and other Indigenous communities.[70-72]
- **Types of tobacco and alcohol use patterns** – In some cases, it is necessary to refer to the terms that describe use patterns provided in the literature to accurately discuss published research findings. For example, the terminology used to describe various patterns of nondaily tobacco use may vary across studies; therefore, this monograph provides the exact terminology used in the referenced article (e.g., by using terms such as nondaily, occasional, light, or social).
- **Race, ethnicity, and ancestry** – This monograph recognizes that the terms race, ethnicity, and ancestry are not uniformly defined and applied throughout the literature, and key differences exist in the use of these terms, especially between the genetics field and other disciplines. Furthermore, the categories used for race and ethnicity in federal surveys have changed over time. Although the applications of these terms continue to evolve, this monograph uses the terminology as defined in the studies or surveys cited and analyzed.
- **Cancer survivor** – A person is considered a cancer survivor from the time of diagnosis through the balance of life. There are many types of survivors, including those living with cancer and those free of cancer.[73]

Tobacco and Alcohol Products

Most of the research in this monograph focuses on the co-use of tobacco in the form of cigarettes and alcohol of all types (i.e., beer, wine, and distilled spirits), where cancer risks are clearly established and where much past research has focused. Over the past 20 years, there have been considerable changes in both the tobacco and alcohol marketplaces, with many new products being added to the market.[74-77] However, there is limited literature available on new products and several other important topics, including the co-use of other tobacco products, such as e-cigarettes, with alcohol and/or other substances and the potential cancer risks of these co-use patterns.

The monograph also recognizes that tobacco and alcohol are increasingly co-used with cannabis. Results from National Survey on Drug Use and Health indicate that cannabis, also referred to as marijuana, has become the most commonly reported substance after tobacco and alcohol.[2] In 2024, 35.0% of young adults ages 18 to 25 (12.2 million people) reported using cannabis in the past year.[2] Use of cannabis is more common among people who smoke cigarettes or drink alcohol than among those who do not.[2,78-81] Although outside the scope of this monograph, the use of cannabis among people who use tobacco and alcohol is an important public health issue and has implications across the tobacco control landscape. Future studies of the use of all three substances (polysubstance use) are needed, such as effects of polysubstance use on tobacco use,

initiation, and cessation; cancer risk; and the policy and commercial landscape of tobacco, alcohol, and cannabis sales and marketing, among other topics.

Monograph Strengths and Limitations

One goal of this monograph is to synthesize and discuss the state of the science of how tobacco and alcohol co-use affects tobacco control. The volume presents what is known about tobacco and alcohol co-use as of 2026 and opportunities for future research efforts. In addition to synthesizing the existing scientific literature, new analyses were conducted of patterns of tobacco and alcohol co-use overall and among population subgroups (highlighted in chapters 2 and 3). This volume concludes that further research to address the co-use of tobacco and alcohol could significantly strengthen cancer prevention and control and that addressing alcohol use in the context of tobacco control is likely to accelerate progress in preventing tobacco use initiation, improving rates of cessation, and avoiding relapse to smoking. Like other monographs in the Tobacco Control Monograph Series, formal systematic reviews were not conducted as part of the monograph development process; rather, the monograph is an expert, narrative review of the existing literature. Specific measurement challenges and limitations of the existing literature are discussed in each chapter, and conclusions made reflect the strength of the existing evidence.

Major Conclusions

The following volume-level conclusions emerged from the monograph chapters.

1. **Tobacco and alcohol are addictive and carcinogenic substances that are commonly used together.** Approximately 12% of adults in the 2015–2019 National Survey on Drug Use and Health reported co-use of cigarettes and alcohol in the past month, representing nearly 30 million people. Adults who use tobacco (e.g., cigarettes, smokeless tobacco, cigars) are more likely to engage in alcohol use, binge drinking, and risky weekly alcohol use than those who do not use tobacco.
2. **Co-use of tobacco and alcohol occurs from the earliest ages of substance use initiation, and its prevalence is substantially higher among certain population subgroups.** Tobacco and alcohol use often begins in adolescence, and long-term use of these two substances makes it more difficult to quit or reduce their use. Adults who report nondaily use of tobacco report the highest prevalence of alcohol use, binge drinking, and risky weekly alcohol use compared with other tobacco use groups. Tobacco use is positively associated with alcohol use across all racial and/or ethnic groups. Compared with the U.S. adult population overall, tobacco and alcohol co-use is elevated in a number of subgroups, including veterans, people living in rural areas, people who are part of a sexual minority group, and people with some types of mental health conditions.
3. **Tobacco use and alcohol consumption individually increase the risk for numerous types of cancer, even at low levels of use, and there is a synergistic increase in risk of squamous cell cancers of the head and neck and esophagus caused by tobacco and alcohol co-use.** In 2019, an estimated 19.3% of incident cancer cases and 28.5% of cancer deaths in the United States were attributable to cigarette smoking. Smoking one or fewer cigarettes per day increases the risk of numerous cancers. Further, an estimated 5.4% of incident cases of cancer and 4.1% of cancer deaths in the United States in 2019 were attributable to alcohol consumption. The risk for head and neck cancers increases

with one drink per day and possibly at lower levels of consumption, and the risk of breast cancer increases at one or fewer drinks per day. Strong evidence from multiple countries demonstrates a synergistic relationship between tobacco and alcohol use and risk for squamous cell cancers of the head and neck and esophagus.

4. **Tobacco and alcohol co-use results in substantially increased acetaldehyde exposure.** Acetaldehyde is a potent carcinogen which damages DNA in multiple ways; it is both a component of tobacco smoke and an important metabolite of ethanol. Acetaldehyde plays a crucial role in the synergistic increases in risk observed for squamous cell head and neck and esophageal cancers in response to tobacco and alcohol co-use.
5. **People with genetic variants that reduce the rate of metabolism of acetaldehyde are at significantly increased risk of cancer if they consume alcohol.** An estimated 540 million people worldwide have genetic variants that reduce the rate of metabolism of acetaldehyde by aldehyde dehydrogenase 2 (ALDH2); these genetic variants are most common in East Asian populations. People with this variant who drink alcohol are at increased risk of squamous cell head and neck and esophageal cancers. Addressing alcohol use and tobacco and alcohol co-use represents important education and intervention opportunities to reduce the burden of cancer in this population.
6. **Tobacco and alcohol use affect multiple, complex neurophysiological processes in the brain.** Networks of brain regions influence how people respond to nicotine and ethanol. Shared biologic pathways in the brain, including those involved in risk-taking and impulsivity, interact and drive use of substances including tobacco and alcohol and substance use disorders.
7. **Studies have identified a substantial number of genetic variants that are associated with both alcohol use and tobacco use across populations, providing evidence for shared underlying mechanisms.** A better understanding of the shared biological mechanisms from additional genetic studies will inform efforts to prevent use disorders and develop pharmacotherapies for tobacco and alcohol co-use and polyuse with other substances.
8. **Smoking cessation interventions are effective for people who smoke and have an alcohol use disorder.** Engaging in smoking cessation treatment during treatment for alcohol use disorder does not jeopardize sobriety outcomes. However, many people in treatment for alcohol use disorder who smoke do not receive smoking cessation treatment. This is a missed opportunity to reduce the burden of cancer in this population.
9. **Addressing alcohol use as a part of smoking cessation treatment is highly likely to advance tobacco cessation goals.** Alcohol use, especially heavy alcohol use, is a barrier to quitting smoking. For this reason, people trying to quit smoking should be advised and assisted to reduce or stop their alcohol use. Integrated tobacco use treatments that address both tobacco and alcohol use may increase reach and effectiveness across multiple settings, including inpatient and outpatient clinical settings and community settings.

10. **Ensuring broad access to, and use of, evidence-based therapies for tobacco and alcohol treatment will help reduce cancer incidence and mortality.** Pharmacological and behavioral therapies for both tobacco and alcohol treatment are underutilized, especially the combined use of both types of therapies, which is the most effective treatment approach for both substances. Moreover, increased understanding of individual genetic variation may inform precision medicine approaches to addressing tobacco and alcohol use.
11. **Prevention programs and interventions that address both tobacco and alcohol may be effective to prevent initiation and ongoing use of both substances.** Effective interventions to prevent tobacco and alcohol use, especially among adolescents and young adults, exist at multiple levels, including the individual, group, community, mass media, and policy levels. Evidence suggests that prevention programs that address both tobacco and alcohol use are feasible. Integrated interventions are needed to prevent both tobacco and alcohol use early in life and across different population subgroups. In addition, intervention studies that focus on either tobacco or alcohol but measure effects on both substances will be highly informative.
12. **Factors pertaining to the marketing, sale, and distribution of tobacco and alcohol products (and the policies to address them) influence patterns and prevalence of tobacco and alcohol use individually and may also influence co-use.** Co-exposure to tobacco and alcohol product marketing and retail availability in the same outlets is common, suggesting the potential for cross-product effects. Greater retail availability of tobacco is associated with greater tobacco use among adults, and some evidence shows that greater availability of alcohol is positively associated with alcohol use among adults. In addition, both tobacco and alcohol retail outlets tend to be concentrated in neighborhoods characterized by greater socioeconomic disadvantage. Assessing how tobacco and alcohol industries' strategies for marketing and distribution of their products influence the marketplace and contribute to tobacco and alcohol co-use is important. Improved understanding of such influences can inform policy approaches to reduce the harms caused by tobacco, alcohol, and their co-use.
13. **Addressing the co-use of tobacco and alcohol has the potential to strengthen tobacco control and reduce cancer incidence and mortality.** Further research on tobacco and alcohol co-use will accelerate progress in preventing tobacco use initiation, improving rates of cessation, and avoiding smoking cessation lapses.
14. **Increased public awareness of both tobacco and alcohol as modifiable risk factors for cancer is an important step towards reducing the cancer burden.** Cancer prevention efforts have primarily focused on smoking and heavy alcohol use. The complex marketplace of available products, the high prevalence of co-use of tobacco and alcohol, and the fact that even moderate alcohol consumption and low levels of tobacco use increase cancer risk, pose challenges to researchers and clinicians. Increased public awareness of the cancer risks associated with tobacco and alcohol co-use will help reduce cancer incidence and mortality.

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Chapter 2
Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Adolescents and Adults in the United States

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Chapter 2
Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Adolescents and Adults in the United States

Introduction

Epidemiologic surveillance of tobacco and alcohol co-use is important to understand population-level and subgroup behavior regarding these substances and to track usage patterns over time.[1-3] Surveillance may also inform clinical and public health efforts to prevent and control use of these hazardous products and to determine where resources and interventions are most needed. Tobacco and alcohol are among the most widely used addictive and carcinogenic substances that, whether used separately or together, have large impacts on the U.S. and global burden of disease.[4,5] These substances have synergistic effects on risk for certain cancers (see chapter 4),[6-8] in addition to the risk for other cancers and serious chronic diseases associated with their separate use.[9,10]

Tobacco and alcohol use are closely linked; the substances are often used by the same individual, frequently at the same time.[3,11-18] Early onset smoking is a significant predictor of lifetime drinking and the subsequent development of an alcohol use disorder (AUD), and is associated with more severe AUDs compared with late onset smoking and lifetime nonsmoking.[19] People who smoke are at greater risk for AUDs than people who have never smoked yet drink equivalent quantities of alcohol.[20] Among individuals with an AUD, diseases caused by smoking are estimated to cause around one-half of all deaths, a rate greater than that for alcohol-related causes.[21-23] This chapter and chapter 3 present detailed information on use and co-use of tobacco and alcohol across the life course in the United States. Chapters 2 and 3 focus primarily on cigarette smoking, but also present data on use of other tobacco products including cigars, smokeless tobacco, and e-cigarettes.

The chapter first discusses measurement of tobacco and alcohol co-use, defined as the concurrent use of tobacco and alcohol or the use of both substances within a given time frame. It then describes patterns of tobacco and alcohol co-use based on data from published literature and from new analyses of three nationally representative surveys: the Monitoring the Future (MTF) survey, the National Alcohol Surveys (NAS), and the National Survey on Drug Use and Health (NSDUH). These three surveys focus on tobacco, alcohol and substance use, and are the major sources of data on co-use for children and adults in the United States. The chapter draws attention to questions not fully addressed in the literature, including the extent of tobacco and alcohol co-use among individuals in the U.S. population and whether greater intensity of use is associated with co-use. The chapter's findings highlight the strong positive association between tobacco and alcohol use, as well as between cigarette smoking frequency and alcohol quantity and heavy drinking occasions. In chapter 3, data are presented in relation to various demographic subpopulations. Although tobacco and alcohol are often co-used with cannabis, trends in cannabis use and co-use are outside of the scope of this chapter.

Measurement of Tobacco and Alcohol Co-Use

Several major U.S. health surveys measure tobacco and alcohol use among adolescents and/or adults (Box 2.1). A description of the measures of tobacco and alcohol use in these surveys is provided in Appendix 2A.

Box 2.1. Major National Health Surveys With Tobacco and Alcohol Use Measures

- Monitoring the Future (sponsored by the National Institute on Drug Abuse)
- National Epidemiologic Survey on Alcohol and Related Conditions and National Alcohol Surveys (sponsored by the National Institute on Alcohol Abuse and Alcoholism)
- National Health Interview Survey, National Health and Nutrition Examination Survey, and Behavioral Risk Factor Surveillance System surveys (sponsored by the Centers for Disease Control and Prevention)
- National Survey on Drug Use and Health (sponsored by the Substance Abuse and Mental Health Services Administration)
- Population Assessment of Tobacco and Health Study (sponsored by the National Institutes of Health and the U.S. Food and Drug Administration)

Although most U.S. national health surveys include questions about both tobacco and alcohol use, understanding the extent and patterns of co-use of these two substances is often difficult. Few nationally representative studies examine *both* alcohol use and tobacco use in-depth (see Appendix 2A). Rather, surveys often include detailed questions about use of one substance but a far less detailed assessment of use of the other (e.g., with binary [yes/no] response format). In addition, no general population studies measure simultaneous co-use (use of both tobacco and alcohol at the same time and/or during circumstances when one substance is already being consumed), although it is a common behavior with important implications for prevention, cessation, and disease risks. Rather, surveys define co-use of tobacco and alcohol as use of both substances within a particular reference period, which can range from 1 day to 1 week, 2 weeks, 30 days or 1 month, a year, a decade, or a lifetime. The reference period selected for measurement or analysis affects the estimated prevalence of co-use. Current tobacco or cigarette use is often defined as use within the past 30 days. However, this timeframe has limitations for the assessment of current alcohol use because a substantial number of people who use alcohol drink less than monthly.[24,25] Additionally, seasonal variation in alcohol consumption, such as heavier alcohol use in winter months, is not necessarily captured by past-30-day measures.[24,25]

Surveys also vary in their inclusion of detailed measures of frequency and intensity of use (e.g., number of drinks per day or occasion, number of cigarettes per day, or number of days of use within a reference time period), and specific alcohol (e.g., beer, wine, spirits) or tobacco products (e.g., cigarettes, cigars, smokeless tobacco, hookah, e-cigarettes) used (see Appendix 2A). Some surveys assess heavy episodic drinking, also known as binge drinking, defined as the frequency of having five or more drinks on a single occasion or day, or having five or more drinks for men and four or more for women on a single occasion or day. Time

frames for measuring binge drinking may also vary. Some measures of binge drinking refer to drinks consumed within 2 hours so that consumption can be closely correlated to elevated blood alcohol content, while other measures of binge drinking refer to drinking within a day or an undefined "occasion." Less commonly used measures of heavy drinking include high intensity drinking (HID), defined as the frequency of having had a specific, high number of drinks (e.g., more than 8, 10, 12, or 15 drinks) per day or occasion[26-28] and the maximum number of drinks consumed in 1 day during a defined period,[29] such as the past week, month, year, or lifetime.

Almost all national surveillance surveys rely on self-report.[30,31] There are challenges regarding the accuracy of self-reported measures of tobacco and alcohol use and co-use. In particular, individuals may underreport or misreport their tobacco or alcohol use, or the amount, frequency, and patterns of use, due to perceived stigma or social desirability bias.[32-35] This is particularly true for people for whom alcohol and/or tobacco use is highly stigmatized, such as people with cancer or pregnant people.[36-38] Some research indicates that around 1 in 10 patients fail to disclose their actual smoking status to their health care providers.[39-41] Similarly, a review of 54 studies examining the relationship between self-reported smoking status and smoking status verified by cotinine measurement found that self-report underestimates actual smoking by between 4.8% to 9.4%, depending on whether cotinine was measured in saliva, serum, blood, or plasma.[42] Nonetheless, self-reported tobacco product use tends to be valid in population-based studies, and the extent of underreporting has not changed over time.[43-46] Accurate, self-reported quantification of tobacco use intensity can also be challenging, particularly for e-cigarettes and other emerging tobacco products. For example, self-reported number of sessions and amount of e-liquid used may underestimate use of e-cigarettes compared to self-reported number of puffs.[47]

Underreporting is also a challenge for self-reported measures of alcohol use. In particular, stigmatization of people with AUDs can influence self-report of heavy drinking and treatment for AUDs.[48-51] Generally, adult self-report of any current drinking can be expected to be accurate.[52-54] However, self-reported lifetime never drinking has been shown to include adults who drank at some point in the past; typically, past consumption was usually of low frequency and quantity, but about 20% of people reporting lifetime abstention reported having 5 or more drinks at least monthly at another time.[55] In addition, older adults may not accurately recall past alcohol consumption, or may deliberately underreport past alcohol use.[25,56] Analysis of the coverage of sales (i.e., comparison of alcohol sales with reported drinks consumed) in general population surveys shows substantial underreporting of consumption.[57] Explanations for underreporting of actual consumption include social desirability bias, that actual drink sizes are frequently larger than the standard drink size, and the use of survey measures that focus on typical drinking, missing occasions where heavy drinking occurs.[25] In addition, people who drink heavily may be underrepresented in general population surveys.[25]

Overall, the research shows that inaccuracy in self-report is likely to result in underreporting of tobacco and alcohol use behaviors, and therefore self-reported data may underestimate the extent of the problem of tobacco and alcohol use in the United States. Measurement challenges related to tobacco and alcohol use are discussed further in chapter 4.

Differences in the history of minimum legal age sale or purchase of tobacco and alcohol might also influence self-reported use due to social desirability bias. As discussed in chapter 8, the minimum legal age to purchase or possess alcohol in the United States has been 21 since the 1984 National Minimum Drinking Age Act.[58] However, the minimum legal age of sale for tobacco products was only made 21 nationwide in 2019 via Tobacco 21 provisions in the Further Consolidated Appropriations Act.[59] For tobacco, self-reported tobacco use declined after passage of Tobacco 21 legislation, but biomarker results were not as consistent in indicating declines in use.[60] This suggests that self-report is sensitive to changes in minimum legal age, but it is not known how long such an effect will persist and whether social desirability bias differs in relation to tobacco and alcohol.

Methods

To identify studies that estimated the prevalence of tobacco and alcohol co-use in the U.S. population overall, targeted keyword searches using combinations of tobacco and alcohol terms were conducted using Google Scholar, PubMed, Ovid, and Scopus. Literature searches were also conducted for prospective alcohol mortality and morbidity studies, because these studies have also examined tobacco co-use as a confounder. Search terms are listed in Appendix 2B.

The chapter highlights findings from nationally representative studies; in addition, findings from nonnationally representative studies were included to provide information about specific subgroups. New analysis of the NAS and the NSDUH were conducted to better understand tobacco and alcohol co-use patterns among adults, while the MTF was analyzed to better understand co-use patterns among adolescents. These surveys were chosen because they are nationally representative, have large sample sizes, and include detailed tobacco and alcohol measures or subgroup data not provided by other nationally representative surveys.

The Monitoring the Future Study

MTF is a nationally representative survey of U.S. students in 8th, 10th, and 12th grade.[61] The analyses provide annual estimates of past-30-day co-use (use of alcohol with cigarettes, large cigars, smokeless tobacco, and nicotine vaping) prevalence from 2000 to 2023 by grade. Among participants who reported use of cigarettes, large cigars, smokeless tobacco, or nicotine vaping in 2023, prevalence of past-30-day alcohol use and past-2-week binge drinking was also reported. For additional details on the MTF methodology and measures, see Appendix 2C.

The National Alcohol Surveys

The NAS are a population-based survey series representative of noninstitutionalized adults ages 18 or older within all 50 states and the District of Columbia.[62] As of 2026, the NAS is the only U.S. population-based survey that includes measures that allow for the calculation of lifetime-accumulated tobacco and alcohol use. For the current analyses, data from adults surveyed in the 2014–2015 and 2019–2020 NAS (the most recent NAS data available) were pooled, and estimates were weighted to be representative of the U.S. population. Pooled data were needed to analyze tobacco and alcohol co-use by subgroup characteristics; these years of data collection afforded a large enough sample for these comparisons. Because the harms of tobacco and alcohol use are cumulative, estimates of lifetime exposure are important to

understand population risks for cancer and other chronic diseases. The NAS also includes detailed measures, such as the number of beverage-specific drinks consumed, amounts of alcohol consumed in different drinking contexts, and frequency of HID. For additional details on the NAS methodology and measures, see Appendix 2C.

The National Survey on Drug Use and Health

The NSDUH is a nationally representative survey of the noninstitutionalized civilian population ages 12 or older in the United States, excluding people with no permanent residence or living in college dormitories, group homes, or on military bases.[63] For the current analyses, data from participants ages 18 and older in the 2015–2019 NSDUH were pooled, and estimates were weighted to be representative of the U.S. population. The 2015–2019 survey years were the most recent comparable 5-year series available for analysis; methodological changes made in 2020 due to coronavirus disease 2019 (COVID-19) prevent comparisons with earlier years. Pooled data were necessary to produce estimates for less represented subgroups (e.g., people from sexual minority groups, people with health conditions; see chapter 3). The NSDUH allows for examination of differences in tobacco and alcohol use by numerous demographic categories, including sex, age, income, educational attainment, race and/or ethnicity, sexual minority status, and military service. The 2015–2019 NSDUH included information on the frequency and quantity of alcohol consumption and cigarette smoking, cigar smoking, and smokeless tobacco use in the past 30 days, as well as the frequency of binge drinking days in the past 30 days. For additional details on the NSDUH survey methodology and measures, see Appendix 2C.

Measures

The MTF analyses focused on past-30-day cigarette smoking, cigar smoking, smokeless tobacco use, nicotine vaping, and alcohol use, as well as past-2-week binge drinking. The NAS analyses focused on past-year co-use of tobacco and alcohol and lifetime use of tobacco and alcohol. Analyses of past-year and lifetime tobacco use from the pooled 2014–2015 and 2019–2020 NAS included use of cigarettes and tobacco products other than e-cigarettes (based on the question "How often have you smoked tobacco cigarettes or used any other kinds of tobacco in the past 12 months? (Please do NOT include e-cigarettes.)"). The 2019–2020 NAS was the first to measure past-year e-cigarette use; therefore, data on co-use of e-cigarettes and alcohol are presented only for that time period. The NSDUH analyses focused on past-30-day co-use of alcohol and cigarettes, smokeless tobacco, and cigars. Estimates of past-30-day use of tobacco and alcohol and their co-use based on data from the NSDUH will differ somewhat from estimates of past-year use drawn from the NAS. As discussed in the section "Measurement of Tobacco and Alcohol Co-Use" above, the past-30-day measure covers the most recent behavior, which may be recalled more easily by participants, while the past-year measure captures longer-term patterns that are less influenced by seasonality or infrequently occurring events. The past-30-day and past-year time frames used in these surveys are the intervals for assessing "current" use. In addition, estimates of current smoking in NSDUH and current tobacco use in the NAS may differ from estimates in other nationally representative surveys in the way they assess current smoking. Some surveys (e.g., the National Health Interview Survey [NHIS]) predicate current cigarette smoking on having smoked ≥100 cigarettes in one's lifetime while also indicating current smoking at the survey timepoint[64]; however, the NAS and NSDUH do not use a lifetime threshold of cigarette consumption in the definition of current smoking.

The measures used in the MTF, NAS, and NSDUH analyses are described in Table 2.1.

Table 2.1 Tobacco and Alcohol Use Measures in the Monitoring the Future Study, National Alcohol Survey, and the National Survey on Drug Use and Health

Measure	Variable type	Description
2000–2023 Monitoring the Future study		
Tobacco use status in past 30 days		
Past-30-day cigarette smoking	Categorical (2 levels)	• Used in the past 30 days • Did not use in the past 30 days
Past-30-day large cigar smoking (2014–2023 only)	Categorical (2 levels)	• Used in the past 30 days • Did not use in the past 30 days
Past-30-day smokeless tobacco use	Categorical (2 levels)	• Used in the past 30 days • Did not use in the past 30 days
Past-30-day e-cigarette use (nicotine vaping; 2017–2023 only)	Categorical (2 levels)	• Used in the past 30 days • Did not use in the past 30 days
Alcohol use status in past 30 days		
Past-30-day alcohol use	Categorical (2 levels)	• Used in the past 30 days • Did not use in the past 30 days
Binge drinking in past 2 weeks		
Past-2-week binge drinking		• Binge drinking in past 2 weeks • No binge drinking in past 2 weeks
2014–2015 and 2019–2020 National Alcohol Surveys		
Tobacco use status in past year		
Tobacco use status (excluding e-cigarettes)[a]	Categorical (4 levels)	• Never use in lifetime • Former use (use in lifetime but not past year) • Nondaily use in past year • Daily use in past year
E-cigarette use status (2019–2020 only)	Categorical (3 levels)	• No use in past year • Monthly or semimonthly use in the past year (monthly or less, weekly or nearly weekly) • Daily or nearly daily use in past year
Cigarette smoking in lifetime		
Pack years[b] (2014–2015 only)	Categorical (4 levels)	• 0 pack years • 0.1–20 pack years • 20.1–40 pack years • ≥40.1 pack years
Alcohol use in past year		
Drinking within guidelines[c]	Categorical (3 levels)	• No alcohol use in past year • Past-year alcohol use within guidelines (≤7/14 drinks per week or <4/5 drinks per day for women/men) • Past-year alcohol use exceeding guidelines (>7/14 drinks per week or ≥4/5 drinks per week for women/men)
5+ drinking	Categorial (2 levels)	Consuming ≥5 drinks in a single day (yes/no) in the past year

Table 2.1 (continued)

Measure	Variable type	Description
High intensity drinking (HID)	Categorical (2 levels)	Consuming ≥8/12 drinks in a single day for women/men (yes/no) in the past year
Volume consumed	Continuous	Number of standard drinks consumed by drink type (beer, wine, and spirits) in the past year
	Categorial (3 levels)	Frequency of drinking at bars, house parties, or at home in the past year: • Weekly or more • Monthly or less • Never
Alcohol use in lifetime		
Number of 5+ drinking days	Continuous	Number of days over lifetime in which participants reported having 5 or more drinks on one occasion (based on participants' age and questions about drinking in each decade)
2015–2019 National Survey on Drug Use and Health		
Tobacco use status in past 30 days		
Cigarette smoking status	Categorical (5 levels)	• Never use in lifetime • Former use (use in lifetime but not in past 30 days) • Nondaily use in past 30 days, <300 cigarettes per month • Daily use in past 30 days, <300 cigarettes per month • Daily or nondaily use in past 30 days, ≥300 cigarettes per month
Smokeless tobacco use status	Categorical (4 levels)	• Never use in lifetime • Former use (use in lifetime but not in past 30 days) • Nondaily use in past 30 days • Daily use in past 30 days
Cigar smoking status	Categorical (3 levels)	• Never use in lifetime • Former use (use in lifetime but not in past 30 days) • Current use (nondaily or daily use in past 30 days)
Alcohol use status in past 30 days		
At-risk drinking	Categorical (5 levels)	• Never use in lifetime • Former use (use in lifetime but not in past 30 days) • Past-30-day alcohol use within guidelines (≤7/14 drinks per week or <4/5 drinks per day for women/men) • Binge drinking (≥4/5 drinks on an occasion for women/men) without risky weekly use (>7/14 drinks per week for women/men) in past 30 days • Risky weekly use (>7/14 drinks per week for women/men) in past 30 days (with or without binge drinking)

[a]The measure of tobacco use status in the NAS excluded e-cigarettes because survey questions asked about smoking "tobacco cigarettes" or "any other kinds of tobacco," but instructed participants to exclude e-cigarettes. [b]*Pack years* are calculated by multiplying the number of packs smoked per day by the number of years smoked. [c]*Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66]

U.S. Population Patterns of Tobacco and Alcohol Use

This section provides a summary of tobacco and alcohol co-use among adolescents and adults based on a review of the literature and analyses of MTF, NAS, and NSDUH data.

Trends Over Time in Tobacco and Alcohol Consumption

Over the last 50 years, cigarette smoking has declined dramatically among U.S. adults—from more than 40% of adults in 1964 to less than 10% of adults in 2024[67,68]—with concomitant reductions in the rates of cancers and other diseases and premature death caused by tobacco.[2,69,70] Declining smoking prevalence is widely attributed to the implementation of evidence-based tobacco control policies and programs and changing social norms regarding tobacco use.[9,71] Additionally, patterns of smoking have changed; smoking intensity (as measured by cigarettes per day) has declined over time, and the proportion of people who smoke nondaily increasingly make up a greater proportion of people who smoke over time.[72] For example, data from NHIS show that although daily smoking has declined considerably since the 1990s, nondaily smoking has remained relatively stable, with daily smoking prevalence declining from 22.0% in 1991 to 8.0% in 2023; and nondaily smoking prevalence declining from 4.0% to 3.0% in the same time period.[67]

Although the prevalence of both smokeless tobacco use and cigar smoking are substantially lower than that of cigarette smoking, both have remained relatively stable over the last 2 decades; during 2000–2022, around 2% to 3% of adults reported current smokeless tobacco use and around 4% reported current cigar smoking.[67] Prevalence of current tobacco use is higher among men than women and among younger adults (<65 years) than older adults (ages 65 and older).[2,73] Further details concerning various demographic groups are presented in chapter 3 and in the 2024 Surgeon General's report, *Eliminating Tobacco-Related Disease and Death: Addressing Disparities*.[3]

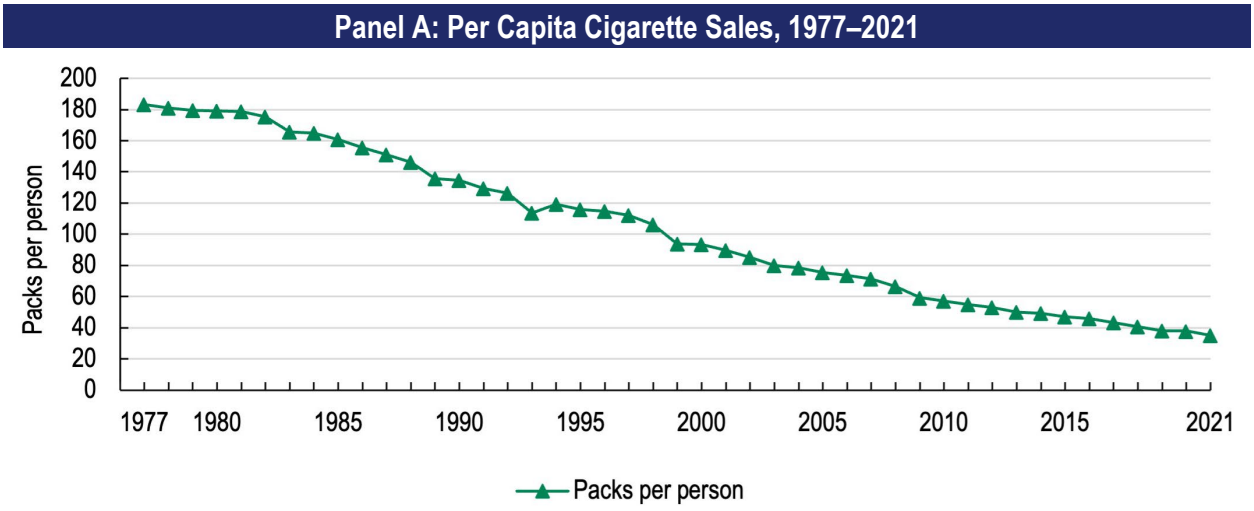
In recent years, the U.S. tobacco product marketplace has become more diverse.[74] As of 2026, the market includes combusted forms of tobacco (cigarettes, cigars, waterpipes, and others), smokeless tobacco, e-cigarettes (also known as vapes or electronic nicotine delivery systems), heated tobacco products, and nicotine pouches.

The U.S. alcoholic beverage market consists of beer, wine, and distilled spirits, products which differ widely in alcohol content.[75] Per capita alcohol consumption and alcohol-related mortality increased in the first decades of the 21st century.[76,77] Age-period-cohort analyses of NAS data have documented declines in volume of alcohol consumed and number of heavy drinking occasions from 1979 to 1995 followed by increasing use levels to 2010.[78-80] Deaths from excessive alcohol use (i.e., ≥4/5 drinks on an occasion for women/men or >7/14 drinks per week for women/men) have also increased between 2016 and 2021, with a notable increase from 2019 to 2020 after the onset of the COVID-19 pandemic.[81,82] NSDUH data show that past-month binge drinking (≥4/5 drinks on an occasion for women/men) decreased from 2021 to 2024; among adults ages 18–25, binge drinking decreased from 30.0% to 26.7%, and among adults ages 26 or older, binge drinking decreased from 22.5% to 21.0%.[83]

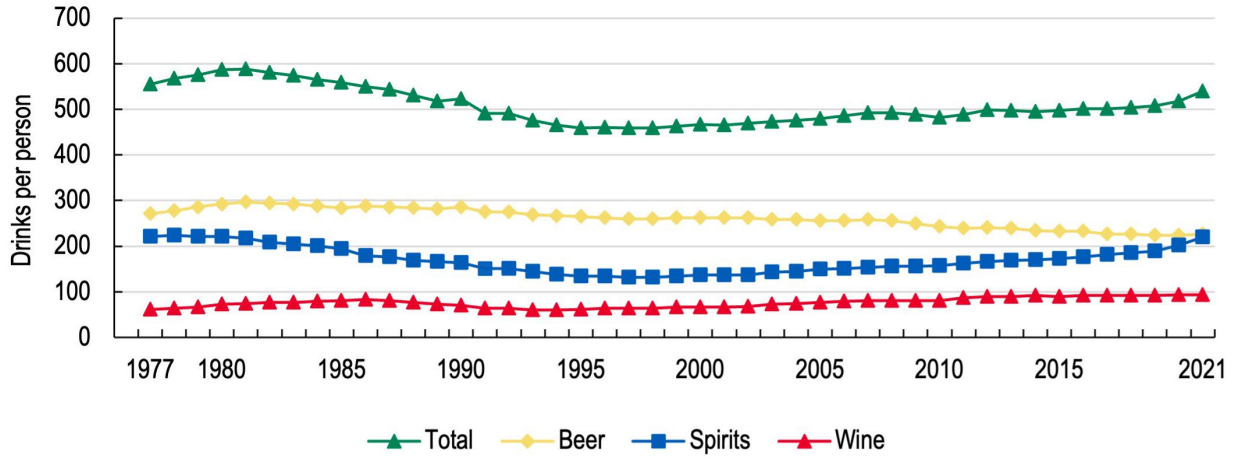
Figure 2.1 shows U.S. per capita cigarette and alcohol sales from 1977 to 2021. Per capita sales of cigarettes declined from 1977 until 2021, and per capita sales of alcohol declined from 1981 to 1995. After 1995, alcohol sales began to steadily increase. Per capita sales of cigarettes were highest in 1977 (183.2 packs per person) and declined to 35.1 packs per person in 2021. Per capita sales of alcoholic beverages per individual ages 14 or older peaked in 1981 at 2.76 gallons of ethanol (the type of alcohol used in alcoholic beverages) per year, and then declined through 1995 to 2.15 gallons before rising again, reaching 2.53 gallons (equivalent to

539 standard drinks) in 2021.[84] Alcoholic beverage preferences have also shifted over time, with beer consumption declining since the 1980s and wine and spirits consumption increasing since the 2000s. The COVID-19 pandemic brought a historically large increase in annual per capita sales of alcohol (2.1% increase) and spirits (6.7% increase) in 2020 and again in 2021 (alcohol: 4.1% increase; spirits: 8.4% increase).[84] In 2021, annual per capita sales were highest for beer (1.06 gallons of ethanol; equivalent to 226 standard drinks), followed by spirits (1.03 gallons of ethanol; 220 standard drinks), and wine (0.44 gallons of ethanol; 94 standard drinks).

Figure 2.1 Trends in U.S. Per Capita Sales of Cigarettes and Alcohol, 1977–2021



Panel B: Per Capita Alcohol Sales, 1977–2021



Note. For cigarette sales, population estimates are for the population ages 15 and older, using population data from the Centers for Disease Control (CDC) Wonder Compressed Mortality File (1977–2004) and the American Community Survey 1-year estimates (2005–2021). Cigarette sales are as reported by cigarette manufacturers. Between 2001 and 2021, values include total number of cigarettes sold and given away as reported by manufacturers. For the years 2011–2015 and 2017–2018, only total sold values are available. Packs per person per year were calculated by dividing total sales data by annual population estimates. For alcohol sales, population estimates are for the population ages 14 and older, using data from the CDC Wonder online query system, as reported by Slater and Alpert.[84] A standard drink contains 0.6 fluid ounces or 14 grams of ethanol. Gallons of ethanol per person reported by Slater and Alpert[84] were converted to standard drinks per person by multiplying the number of gallons by 213.333. Per capita sales were calculated using annual, time-varying, beverage-specific % alcohol by volume (ABV) values to account for variations over time due to changes in the strength of alcoholic beverages and beverage-type consumer preferences. The calculations of time-varying % ABV followed the methodology developed by Kerr and colleagues[85-88] and most recently reported by Martinez and colleagues.[89]
Source: Centers for Disease Control and Prevention,[90] Slater and Alpert 2024,[84] Federal Trade Commission 2021,[91] U.S. Census Bureau.[92]

Co-Use of Tobacco and Alcohol Among Adolescents

Adolescence, defined by the World Health Organization as the period from ages 10 to 19,[93] is a unique developmental period characterized by increased autonomy from parents, a focus on peers, identity formation, experimentation, and risk-taking.[94-97] During adolescence, risk behaviors often occur together, or "cluster," and may have implications for adult health and disease risk.[98-101] The majority of adults who smoke cigarettes began smoking as adolescents.[100] In addition to initiating the processes that may eventually lead to cancer, cardiovascular disease, chronic obstructive pulmonary disease and other adverse health effects, adolescent smoking is a risk factor for later use of alcohol and other substances.[100] Early onset of cigarette smoking is also a risk factor for severe nicotine dependence and development of disease caused by smoking.[100] Similarly, alcohol use often begins during adolescence and young adulthood and early onset of alcohol use is a risk factor for AUD, as well as use of other substances.[102] Adolescent alcohol consumption is associated with a wide array of harmful consequences, including serious injury and death, impaired brain development, and poor decision-making.[102] Additionally, among adolescents who use alcohol, binge drinking is the most common pattern of alcohol use.[102,103] Although experimentation with and initiation of cannabis use also often begins in adolescence,[104] and often co-occurs with tobacco and alcohol use,[105,106] this topic is beyond the scope of this monograph.

As with adults, the prevalence of adolescents' use of tobacco and alcohol products has changed over time. Adolescent cigarette smoking has declined significantly since the mid-1990s.[3,100] For example, among 12th grade students, past-30-day cigarette smoking peaked in 1976 at 39%,[107] declining to 2.9% in 2023 (Appendix Table 2D.3). However, adolescents' use of tobacco products now reflects the diversity of the tobacco product marketplace. Most notably, since 2014, e-cigarettes have been the most commonly used tobacco product among U.S. middle and high school students and use of two or more tobacco products is common.[3,108] Analysis of the National Youth Tobacco Survey found that, in 2023, 10.0% of middle and high school students reported current (past-30-day) use of any tobacco product (12.6% of high school students; 6.6% of middle school students).[109] Additionally, 7.7% of students reported use of e-cigarettes, 1.6% reported use of cigarettes, 1.6% reported use of cigars, 1.2% reported use of smokeless tobacco, and 1.5% reported use of nicotine pouches; roughly one-third of all students who currently used tobacco (3.4% of students overall) reported current use of 2 or more tobacco products.[109] Although alcohol use and binge drinking among adolescents have generally declined since the 1980s, alcohol remains the most commonly used substance among teens and use prevalence increases with age.[107] MTF data indicates that lifetime prevalence of alcohol use among 8th, 10th, and 12th grade students declined from 68.4% in 1993 to 35.7% in 2023,[110] and past-30-day alcohol use declined from 36.3% to 14.3%.[110] In 2023, lifetime alcohol use was 20.1% among students in 8th grade, 35.8% among students in 10th grade, and 52.8% among students in 12th grade,[110] while past-30-day alcohol use by grade level was 5.9% among students in 8th grade, 13.7% among students in 10th grade, and 24.3% among students in 12th grade (see Appendix Tables 2D.1, 2D.2, and 2D.3).

Studies of Co-Use of Tobacco and Alcohol Products Among Adolescents

Selected studies using nationally representative data to understand levels and patterns of co-use of tobacco and alcohol among U.S. adolescents are described in this subsection. These studies draw on self-reported data from the Youth Risk Behavior Survey (YRBS), MTF, NSDUH, or the Population Assessment of Tobacco and Health (PATH) study and may also look at tobacco and alcohol co-use in the context of use of other substances or health risk behaviors. As noted in "Measurement of Tobacco and Alcohol Co-Use" above, self-report may underestimate use; especially in nonconfidential settings, adolescents in particular may be reluctant to disclose their use of alcohol, tobacco, and other substances.[111] In the past decade, more studies have addressed patterns of use and association of alcohol use with cigarette smoking, as well as new and emerging tobacco products.

Several studies used YRBS data to explore the co-use of tobacco and alcohol among adolescents in 9th through 12th grade. A study using 2019 YRBS data found that 7.9% of middle and high school students (ages 14–18) reported co-use of cigarettes and alcohol in the past 30 days, and 13.4% reported co-use of electronic nicotine vapor products and alcohol. Frequent use (use on ≥6 days in the last 30 days) of cigarettes and alcohol or electronic nicotine vapor products and alcohol was less prevalent (1.6% and 6.1% of students, respectively).[112] A study of 2017 YRBS data focused on use of e-cigarettes with other tobacco products, alcohol, and cannabis.[113] Among the 12% of high school students who reported using e-cigarettes, nearly all (93%) also reported use of another substance, and the use of e-cigarettes with alcohol and one or more other substances (tobacco and/or cannabis) was by far the most common product combination. A study of 2013 YRBS data demonstrated that current (past-30-day) use of alcohol varied substantially

by tobacco use status; current (past-30-day) alcohol use was 22.9% among high school students who did not use tobacco, 70.4% among those who used a single tobacco product, and 85.8% among those who used >1 tobacco product.[114] Additionally, students who reported current use of more than one tobacco product were at significantly greater risk of current drinking and binge drinking than students who reported either use of one tobacco product or no use of tobacco.

Daw and colleagues (2013)[115] analyzed MTF data to describe trends in alcohol use, cigarette smoking, and their co-use in the past 30 days among U.S. students in 12th grade (N = 556,731) from 1976 to 2010. Among both male and female students in 12th grade, the past-30-day co-use of cigarettes and alcohol declined fairly consistently over time, as did the co-occurrence of heavy smoking and heavy drinking, defined as use of half-pack per day or more of cigarettes and ≥6 drinks in the past 30 days. Among male students, heavy co-use of cigarettes and alcohol declined from a peak of 4.9% in 1977 to 0.8% in 2010; among female students, heavy co-use of cigarettes and alcohol declined from more than 3.0% in 1977 to 0.2% in 2010. However, the association between cigarette smoking and alcohol use, particularly heavy smoking and drinking, increased over the study period; that is, the prevalence of co-use was higher than expected than if smoking and drinking behaviors were independent of one another. As the authors note:

> These findings suggest that, as tobacco use, alcohol use, and their co-use has declined among 12th graders in the U.S., the segment of the population that engage in smoking and drinking co-use has become increasingly differentiated from the general population of high school seniors. In other words, rather than being more reflective of the population prevalence of smoking and drinking, those who engage in both tobacco use and alcohol use are increasingly driven either by a common disposition for using both or a causal connection between the two behaviors.[115,p.645]

An analysis of 2017–2019 MTF data for students in 8th, 10th, and 12th grade (N = 51,872) assessed the association between cigarette and e-cigarette use and alcohol use.[116] In this sample, past-30-day use of cigarettes, e-cigarettes, or both tobacco products (compared to no tobacco product use) was strongly associated with greater likelihood of past 2-week binge drinking (aOR = 2.80–36.53). As the authors noted, "While all levels of nicotine use are linked to cannabis use and binge drinking outcomes, associations between vaping-only or smoking + vaping and these behaviors are high, especially at the highest levels of binge drinking."[116,p.1077] Finally, a study analyzing MTF data from 2014 found that students in 8th, 10th, and 12th grade who used >1 tobacco product were the most likely to also engage in alcohol use (and other substance use) and to be addicted to these substances.[117] For example, among 12th grade students who used 2 or more nicotine products (e.g., cigarettes, e-cigarettes, and/or other nicotine products), 79.4% reported alcohol use (adjusted odds ratio [aOR] = 11.5, 95% confidence interval [CI] = 8.3–15.9, vs. no current nicotine product use). Among 12th grade students who used only cigarettes, 70.4% (aOR = 4.5, 95% CI = 3.8–12.4, vs. no current nicotine product use) reported alcohol use, and among those who reported only use of e-cigarettes, 60.7% reported alcohol use (aOR = 6.9, 95% CI = 3.2–6.3, vs. no current nicotine product use). Alcohol use was far lower (25.1%) among 12th grade students who reported no use of cigarettes, e-cigarettes, or other nicotine products. In sum, while use of tobacco and use of alcohol by adolescents has declined substantially over time, co-use of tobacco and alcohol is still more prevalent among adolescents than would be expected from the individual declines in use prevalence for each substance.[117]

Conway and colleagues (2018)[118] analyzed data from Wave 1 (2013–2014) of the PATH study, to understand the co-occurrence of tobacco product use, substance use, and mental health conditions among adolescents ages 12–17 (*N* = 13,617). In adjusted analysis, use of any tobacco product or specific tobacco products (cigarettes, e-cigarettes, cigars, pipe, hookah, smokeless tobacco, and snus) was associated with significantly higher odds of use of alcohol relative to non-use. For example, adolescents who reported ever smoking cigarettes were 5 times more likely to report ever using alcohol compared to adolescents who never smoked cigarettes (aOR = 5.6, 95% CI = 4.8–6.5). Longitudinal data from the PATH study were also used to determine initiation and correlates of initiation for cigarettes and other tobacco products among adolescent (ages 12–17) participants in PATH Wave 1 (2013–2014) and PATH Wave 4 (2016–2018).[119] By Wave 4, nearly one-third (32.5%) of adolescents who never used tobacco at Wave 1 had initiated tobacco use; prior use of other tobacco products and ever use of alcohol were associated with initiation of cigarette, e-cigarette, cigar, and hookah at Wave 4. Pérez and colleagues (2021)[120] analyzed data from Waves 1 through 5 of the PATH study (2013–2019) to estimate the age of initiating past-30-day use of multiple tobacco products (cigarettes with either e-cigarettes or cigarillos) and binge drinking. By age 21, 4.4% (95% CI, 3.7%–5.2%) of adolescents initiated use of cigarettes, e-cigarettes, and binge drinking, and 2.0% (95% CI, 1.2%–2.8%) of adolescents initiated use of cigarettes, cigarillos, and binge drinking (participants could fall into both groups). Lastly, Silveira and colleagues (2018),[121] analyzing data from Waves 1 (2013–2014) and Waves 2 (2014–2015) of the PATH study, documented bidirectional associations between tobacco and alcohol use among adolescents ages 12–17, specifically that ever use of e-cigarettes, cigars, or hookah predicted later alcohol use, and ever alcohol use predicted later tobacco use of any tobacco product and of cigarettes, e-cigarettes, any cigars, traditional cigars, cigarillos, filtered cigars, pipes, hookah, any smokeless tobacco, or snus individually.

A study using the 2018 NSDUH adult data (*N* = 43,026) examined the association between adult binge drinking and the age of initiating smokeless tobacco use, defined as early (less than age 18) or late (age 18 or older).[122] In this study, both early initiation of smokeless tobacco use and early initiation of alcohol use were significantly associated with increased likelihood of adult binge drinking.

Monitoring the Future Analysis: Tobacco and Alcohol Co-Use Over Time Among 8th, 10th, and 12th Grade Students

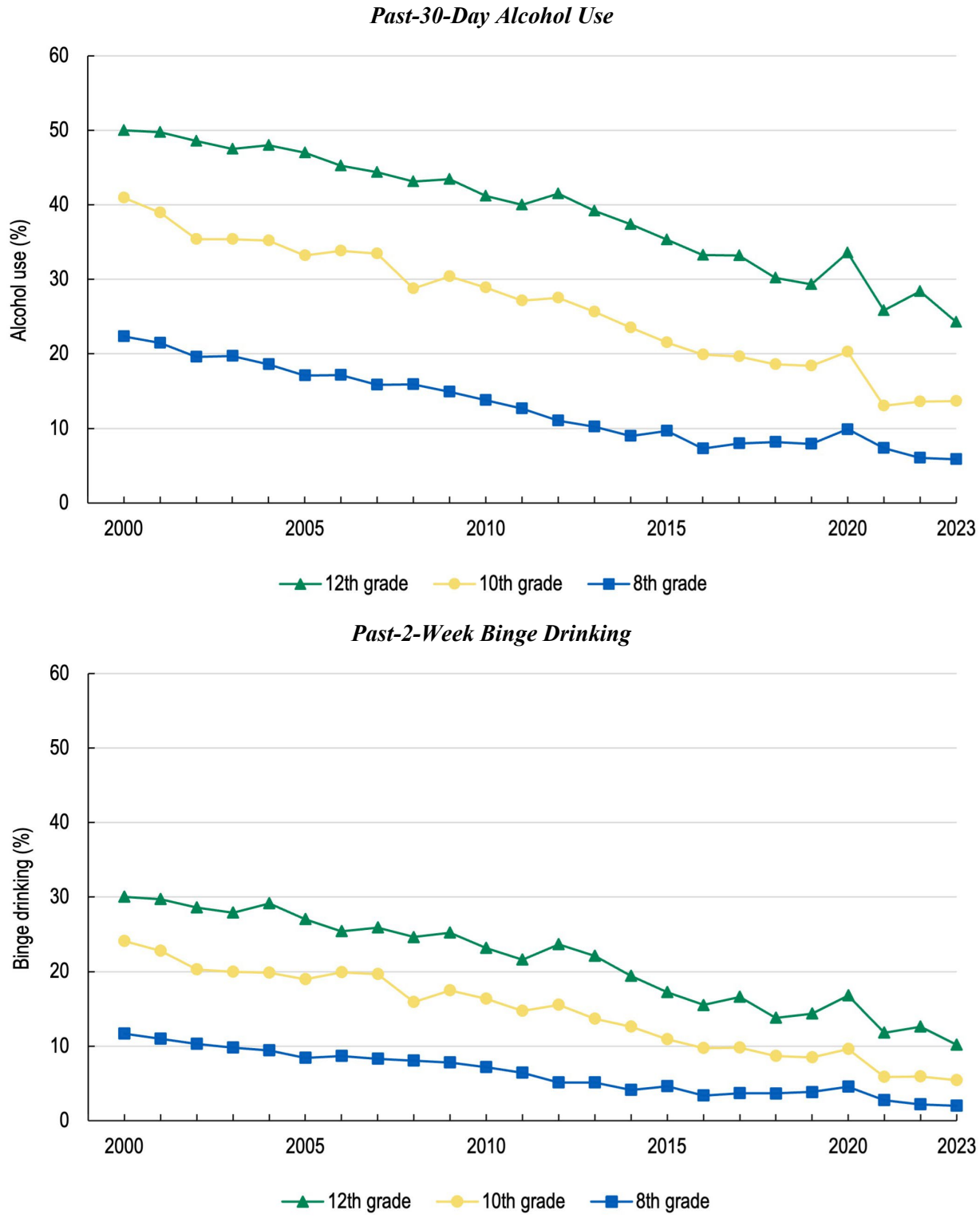
New analyses of 2000–2023 MTF data were conducted to describe trends over time in co-use of tobacco and alcohol among students in 8th, 10th, and 12th grade. Figure 2.2 shows trends in current (past-30-day) use of tobacco (nicotine vaping, cigarettes, large cigars, and smokeless tobacco) and current (past-30-day) alcohol use, as well as trends in co-use of tobacco and alcohol and co-occurrence of tobacco use and past-2-week binge drinking (see Appendix Tables 2D.1, 2D.2, 2D.3). Across all 3 grades, past-30-day alcohol use and past-2-week binge drinking declined fairly steadily over time (Panel A: Alcohol Use by Grade). Overall, co-use of alcohol with nicotine vaping was the most common tobacco-alcohol co-use pattern observed among 8th, 10th, and 12th grade students. In 2017, the MTF study revised its questions on vaping to include separate questions on vaping of nicotine, cannabis, and "just flavoring." Nicotine vaping among students in all 3 grades (8, 10, and 12), rose steadily from 2017 to 2019 and then began to slowly decline (Panel B: Nicotine Vaping and Co-Use with Alcohol by Grade). Trends in co-use of

alcohol and nicotine vaping and binge drinking and nicotine vaping roughly followed trends in students' nicotine vaping. In 2023, a total of 9.9% (95% CI, 7.9%–12.3%) of 12th grade students, 6.2% (95% CI, 5.2%–7.3%) of 10th grade students, and 2.3% (95% CI, 1.7%–3.2%) of 8th grade students reported current (past-30-day) co-use of alcohol with nicotine vaping. About one-half of students who reported past-30-day nicotine vaping and alcohol co-use also reported past-2-week binge drinking, and 5.5% (95% CI = 4.2%–7.0%) of students in 12th grade, 3.2% (95% CI = 2.5%–4.0%) of students in 10th grade, and 1.1% (95% CI = 0.7%–1.7%) of students in 8th grade reported binge drinking and nicotine vaping.

Co-use of cigarettes and cigars with alcohol declined over time, while co-use of smokeless tobacco over time varied. Consistent declines in cigarette smoking and co-use of cigarettes with alcohol, as well as in cigarette smoking combined with binge drinking, were also observed across the 3 grades from 2000 to 2023 (Panel C: Cigarette Smoking and Co-Use with Alcohol by Grade). Indeed, by 2023, less than 3% of students (1.5% [95% CI = 1.0%–2.1%] in 10th grade and 2.1% [95% CI = 1.3–3.2%] in 12th grade) reported co-use of cigarettes with alcohol. Among students in 10th and 12th grade, large cigar smoking declined from 2014 to 2023; co-use of large cigars and alcohol varied over time but also declined since 2014 (Panel D: Large Cigar Smoking and Co-Use with Alcohol by Grade). Among students in 8th grade, large cigar smoking and co-use of large cigars with alcohol was low (<1.0% of students in 2023), but little change in these behaviors was seen since 2014. Variation in the prevalence of smokeless tobacco use, co-use of smokeless tobacco and alcohol, and co-occurrence of smokeless tobacco use with binge drinking was seen from 2000 to 2023 among students in 10th and 12th grade; in contrast, students in 8th grade showed small but steady decreases in all 3 behaviors over time (Panel E: Smokeless Tobacco Use and Co-Use with Alcohol by Grade).

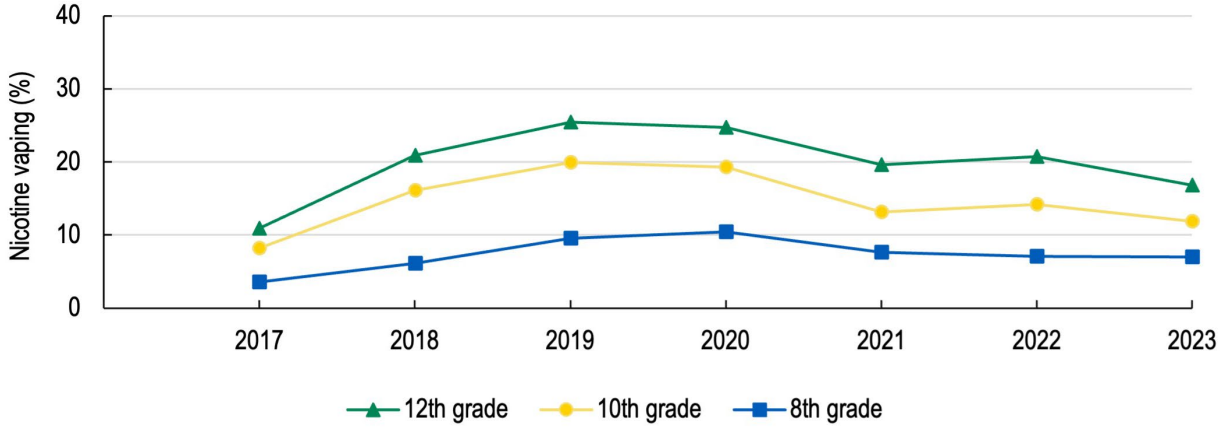
Figure 2.2 Co-Use Prevalence Over Time Among Students in 8th, 10th, and 12th Grade (2000–2023 Monitoring the Future Study)

Panel A: Alcohol Use by Grade

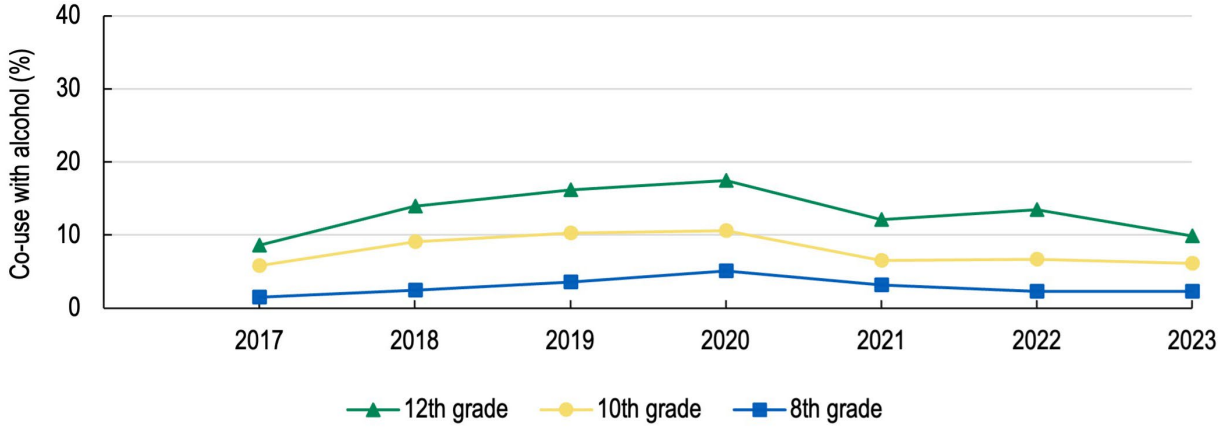


Panel B: Nicotine Vaping and Co-Use with Alcohol by Grade

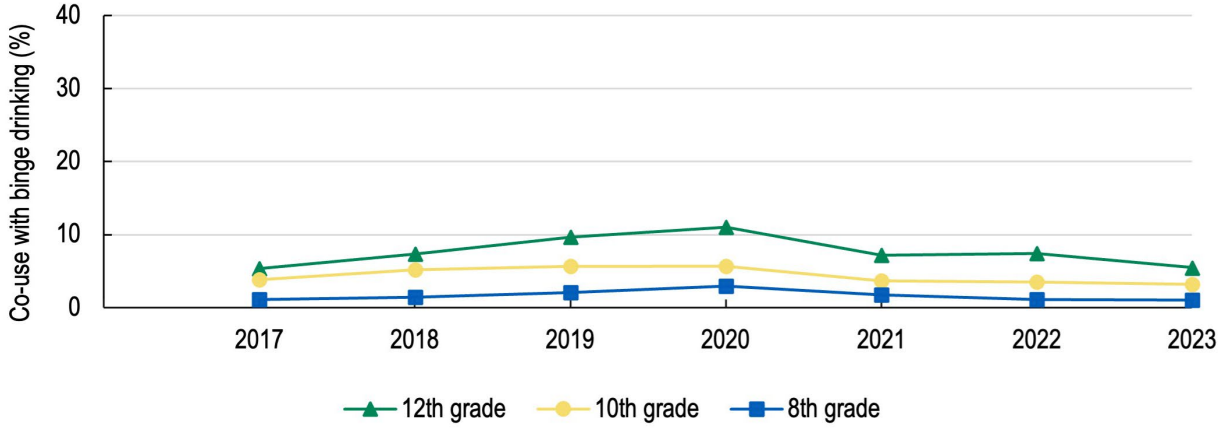
Past-30-Day Nicotine Vaping



Co-Use: Past-30-Day Nicotine Vaping With Past-30-Day Alcohol Use

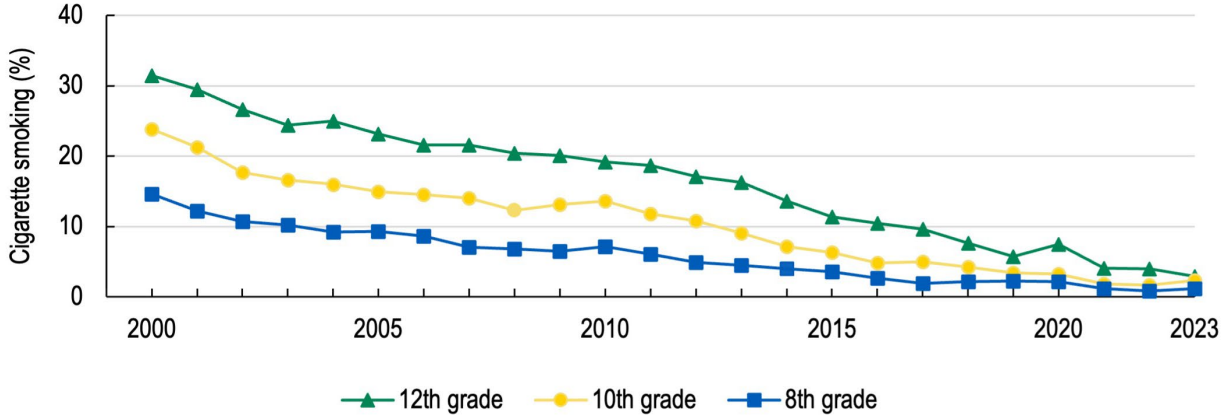


Co-Use: Past-30-Day Nicotine Vaping With Past-2-Week Binge Drinking

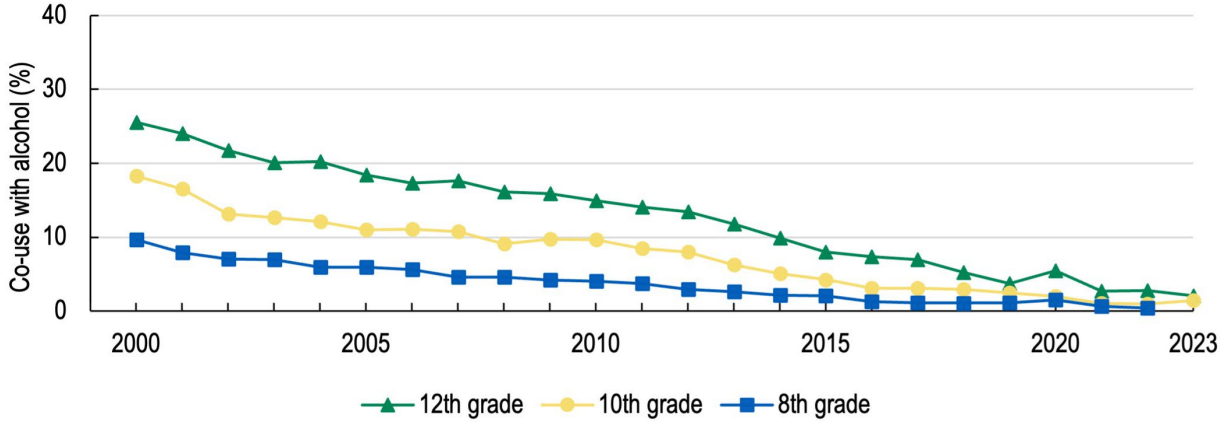


Panel C: Cigarette Smoking and Co-Use with Alcohol by Grade

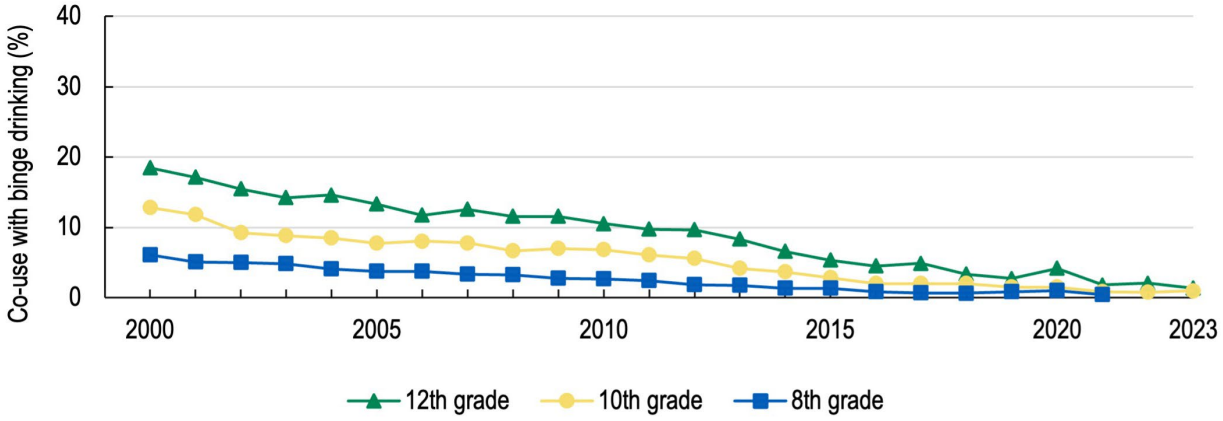
Past-30-Day Cigarette Smoking



Co-Use: Past-30-Day Cigarette Smoking With Past-30-Day Alcohol Use

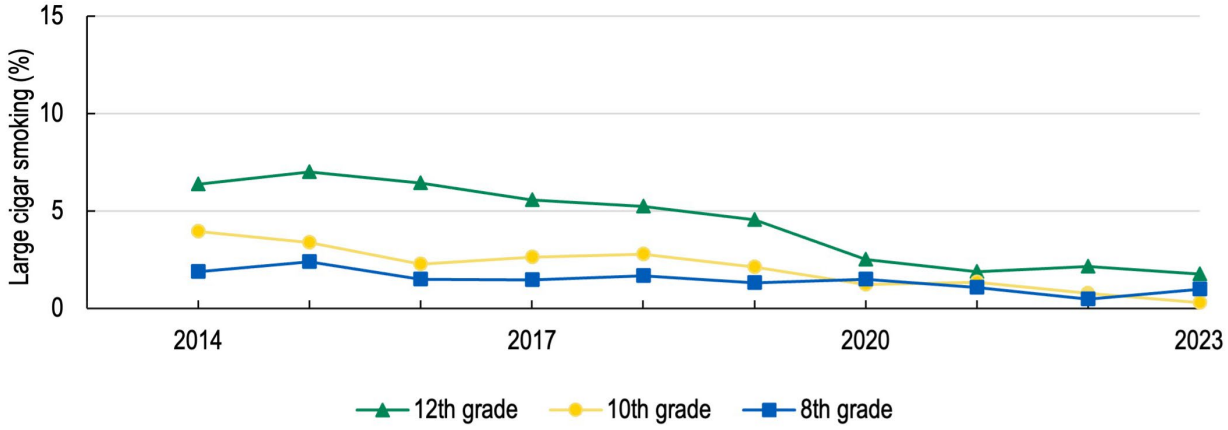


Co-Use: Past-30-Day Cigarette Smoking With Past-2-Week Binge Drinking

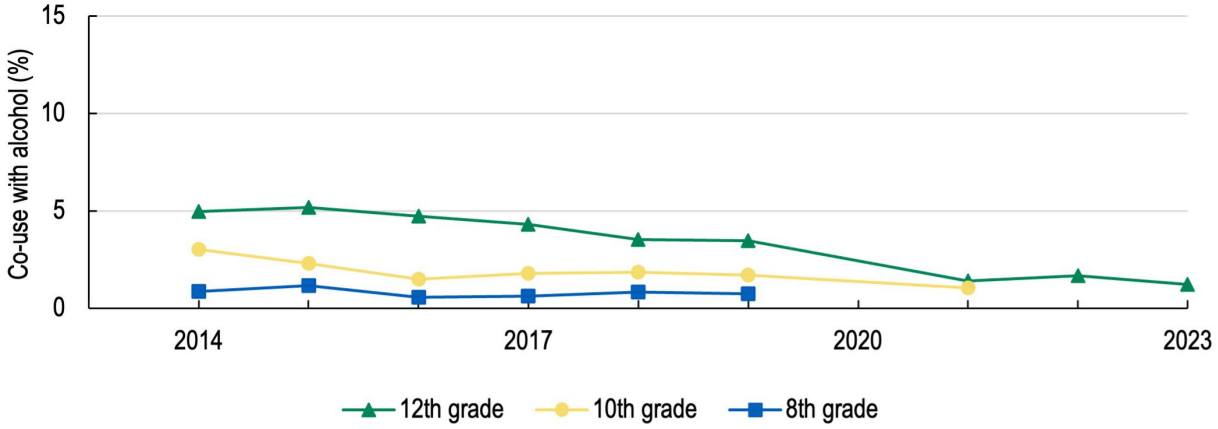


Panel D: Large Cigar Smoking and Co-Use with Alcohol by Grade

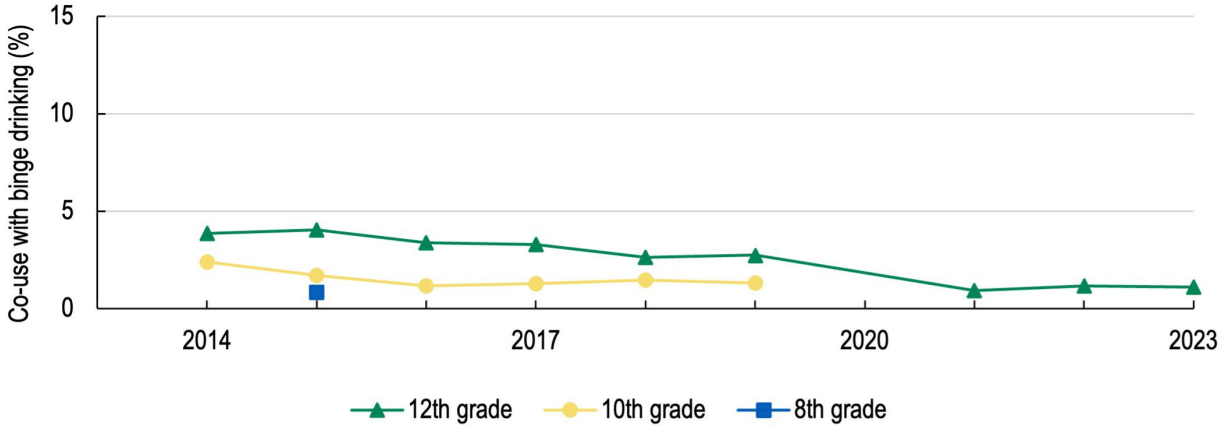
Past-30-Day Large Cigar Smoking



Co-Use: Past-30-Day Large Cigar Smoking With Past-30-Day Alcohol Use

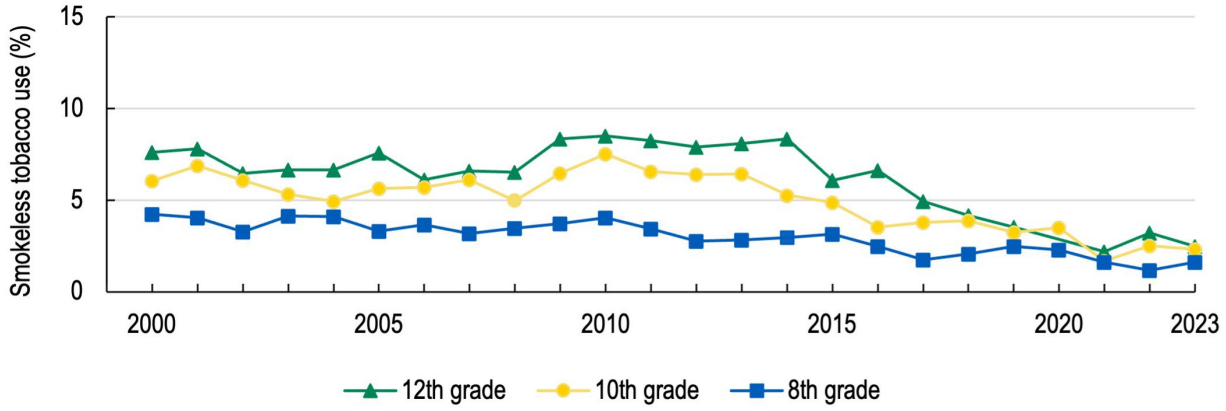


Co-Use: Past-30-Day Large Cigar Smoking With Past-2-Week Binge Drinking

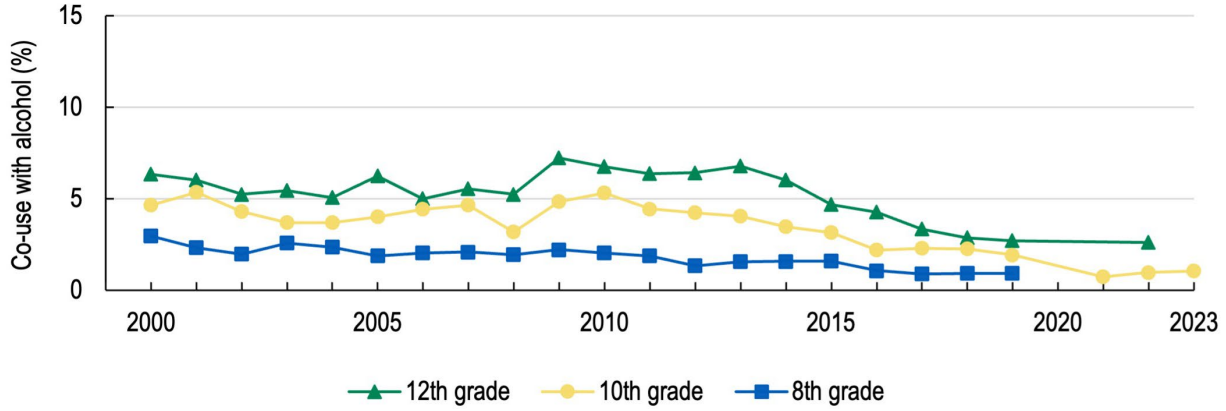


Panel E: Smokeless Tobacco Use and Co-Use with Alcohol by Grade

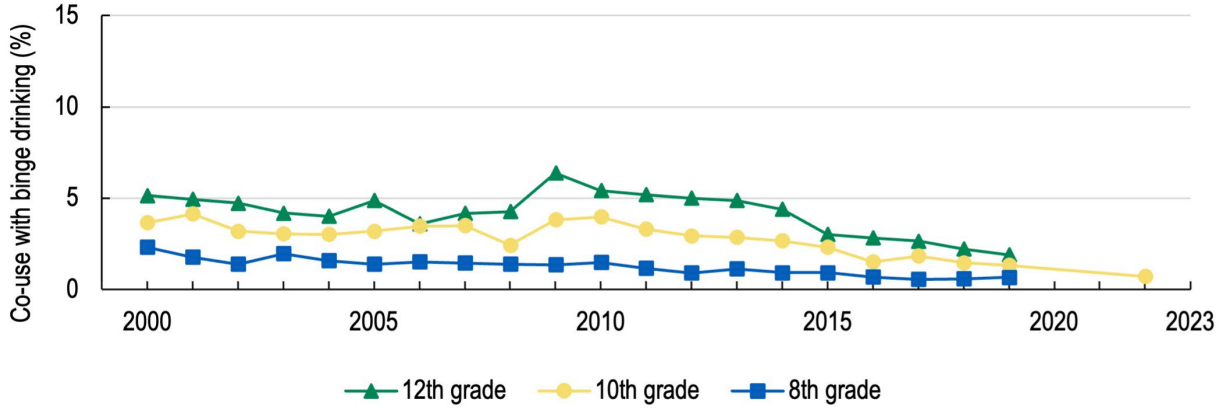
Past-30-Day Smokeless Tobacco Use



Co-Use: Past-30-Day Smokeless Tobacco Use With Past-30-Day Alcohol Use



Co-Use: Past-30-Day Smokeless Tobacco Use With Past-2-Week Binge Drinking

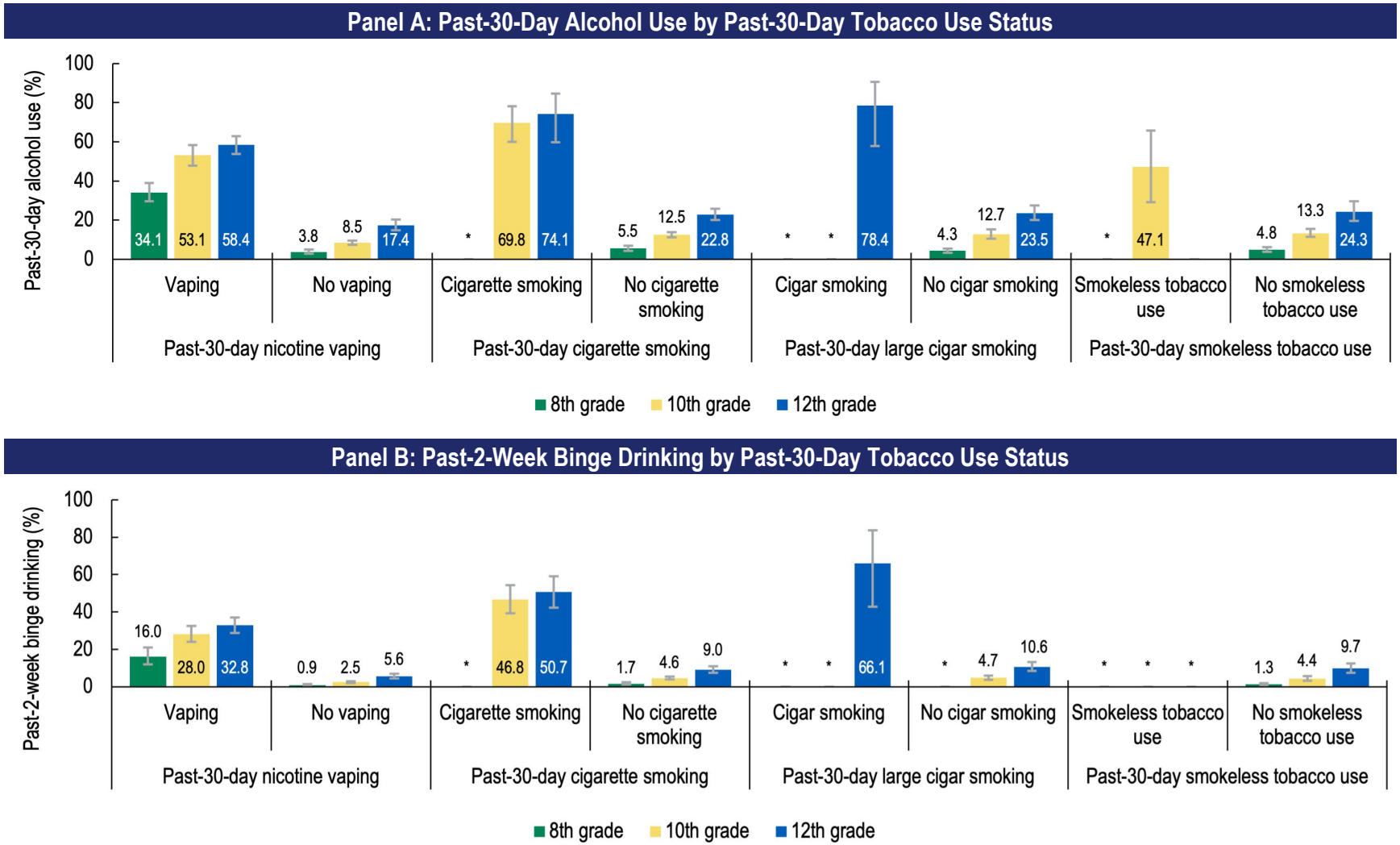


Note. For full data, see Appendix Tables 2D.1, 2D.2, and 2D.3. Some estimates for certain years and grade levels are suppressed due to small sample size. The large cigar question was first asked in 2014. The nicotine vaping question was first asked in 2017. *Binge drinking* is defined as drinking 5 or more drinks in a row over the past 2 weeks. *Tobacco product use* and *alcohol use* are defined as use in the past 30 days. *Co-use* is defined as use of both tobacco and alcohol in the past 30 days, or use of tobacco in the past 30 days and reporting binge drinking in past 2 weeks.
Source: 2000–2023 Monitoring the Future study.

Monitoring the Future Analysis: Alcohol Use Among 8th, 10th, and 12th Grade Students Who Use Tobacco

In 2023, between 47.1% and 78.4% of students in 10th and 12th grade who reported past-30-day use of tobacco products also reported past-30-day alcohol use, as shown in Figure 2.3 (Appendix Table 2D.4). Regardless of grade, past-30-day alcohol use and past-2-week binge drinking levels were far lower among students who reported no past-30-day tobacco use than among their same-age counterparts who reported tobacco use. About one-third (34.1%, 95% CI = 29.5%–39.0%) of students in 8th grade and more than one-half of students in 10th grade (53.1%, 95% CI = 47.8%–58.3%) and 12th grade (58.4%, 95% CI = 53.7%–62.9%) who reported nicotine vaping also reported past-30-day alcohol use. More than two-thirds of students in 10th grade (69.8%, 95% CI = 59.9%–78.1%) and 12th grade (74.1%, 95% CI = 59.8%–84.7%) who reported past-30-day cigarette smoking also reported past-30-day alcohol use. Roughly one-half of the students in 10th grade (46.8%, 95% CI = 39.3%–54.4%) and 12th grade (50.7%, 95% CI = 42.2%–59.1%) who reported cigarette smoking also reported past-2-week binge drinking. Similarly, 16.0% (95% CI, 12.0%–21.1%) of students in 8th grade, 28.0% (95% CI, 24.0%–32.5%) in 10th grade, and 32.8% (95% CI, 28.8%–37.1%) in 12th grade who reported current nicotine vaping also reported binge drinking.

Figure 2.3 Alcohol Use by Tobacco Use Status Among Students in 8th, 10th, and 12th Grade (2023 Monitoring the Future Study)



Note. For full data, see Appendix Table 2D.4. Estimates of alcohol use and binge drinking among students in 8th grade who smoked cigarettes are suppressed due to small sample size. *Binge drinking* is defined as drinking 5 or more drinks in a row over the past 2 weeks. *Alcohol use*, *cigarette smoking*, and *nicotine vaping* are defined as use in the past 30 days.
*Groups with $n < 30$ are suppressed.
Source: 2023 Monitoring the Future study.

Summary: Co-Use of Tobacco and Alcohol Among Adolescents

Tobacco and alcohol use often begin in adolescence. Although the past several decades have seen steep declines in adolescent cigarette smoking, adolescent tobacco use reflects the diversity of the tobacco product marketplace. In particular, e-cigarettes have been the most commonly used tobacco product among adolescents since 2014, and use of multiple tobacco products (polytobacco use) is now common. Although adolescent alcohol use has also declined over the past few decades, alcohol remains the most common addictive substance used by adolescents, and hazardous alcohol use (e.g., binge drinking) among adolescents is not uncommon. Importantly, the proportion of adolescents using both tobacco and alcohol among those using either substance remains high, placing adolescents at risk from the combined effects of tobacco and alcohol.

Analysis of nationally representative studies of youth, including YRBS, MTF, NSDUH, and the PATH study, as well as new analyses of 2000–2023 MTF data conducted for this chapter, provide updated information on the close association between tobacco and alcohol use among adolescents. The literature documents the continued strong association between the use of diverse forms of tobacco products now available in the United States and alcohol use. Among adolescents, tobacco use is a risk factor for alcohol use and alcohol use is a risk factor for tobacco use. The risk for alcohol use and binge drinking is far greater among adolescents who use cigarettes, e-cigarettes, smokeless tobacco, or other tobacco products than among those who do not use a tobacco product. Most adolescents who use a tobacco product also drink alcohol; conversely, most adolescents who drink alcohol also use a tobacco product. Analysis of MTF data shows that, in 2023, co-use of alcohol with nicotine vaping was the most common tobacco-alcohol co-use pattern observed among 8th, 10th, and 12th grade students. Regardless of grade, past-30-day alcohol use and past-2-week binge drinking levels were far lower among students who reported no past-30-day tobacco use than among their same-age counterparts who reported tobacco use. Additionally, the literature strongly suggests that polytobacco use is an even stronger risk factor for alcohol use than use of one tobacco product or no use of tobacco products. This finding has important implications for prevention, given the increased frequency of polytobacco use among adolescents.

Co-Use of Tobacco and Alcohol Among Adults

Selected studies that use nationally representative data to estimate levels of tobacco and alcohol co-use among U.S. adults, or to examine trends in co-use over time, are described in this section.

Studies analyzing nationally representative data from the early to mid-2000s indicate that around one in five adults reported co-use of any tobacco product and alcohol in the past year.[123,124] In the first wave of the nationally representative, National Institute on Alcohol Abuse and Alcoholism (NIAAA)-sponsored National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) conducted in 2001–2002, past-year co-use of alcohol and a tobacco product (cigarettes, cigars, pipes, and/or smokeless tobacco) was reported by 21.7% of adults ages 18 and older (27.5% of men; 16.4% of women).[123] A study using data from the 2012–2013 NESARC-III, which assessed co-use of alcohol and nicotine, found a similar prevalence of past-year co-use: 21.6% of adults reported use of alcohol and nicotine (25.0% of men; 18.2% of women), while 4.6% of adults reported use of alcohol, nicotine, and cannabis (6.0% of men; 3.2% of women).[124]

NESARC data also indicate that cigarette smoking prevalence, nicotine dependence, and nicotine use disorders are higher among people with AUDs, and, conversely, diagnoses of AUDs are more prevalent among people who smoke.[123-133] In the 2012–2013 NESARC, nearly one-half of adults with a past-year AUD reported cigarette smoking in the past year.[131,133] Around one-third of adults with an AUD also reported nicotine dependence (2001–2002 NESARC),[123,127] a nicotine use disorder (DSM-5 criteria; 2012–2013 NESARC),[124] or past-year daily smoking (2012–2013 NESARC).[133] A study using 2012–2013 NESARC data found that the odds of having any nicotine use disorder were higher (aOR = 2.47, 95% CI = 2.25–2.72) among those with an AUD than among those without an AUD.[125] Among young adult (ages 18–25) participants in the 2001–2002 NESARC, more than 30% of those who smoked in the past year reported a past-year AUD diagnosis, compared with 10.8% who did not smoke.[128] Finally, a study examining e-cigarette use among 2012–2013 NESARC participants found that those who used e-cigarettes in the past year had higher odds of having a past-year AUD (nondaily e-cigarette use aOR = 2.44, 95% CI = 2.09–2.84; daily e-cigarette use aOR = 1.89, 95% CI = 1.42–2.52) than those who did not use e-cigarettes, and those who used e-cigarettes and cigarettes nondaily had the highest odds of having a past-year AUD (aOR = 5.20, 95% CI = 3.77–7.17).[134]

Using more recent data from Wave 1 of the PATH study (2013–2014), Cohn and colleagues (2018)[135] found that the prevalence of past-30-day co-use of tobacco and alcohol ranged from around 1% to 5% depending on the respondent's age and type of tobacco product(s) used. Not using tobacco, alcohol, or cannabis was the most common use pattern overall (33.5% among young adults ages 18–24; 36.3% among adults ages 25 and older), followed by alcohol-only use (20.7% among young adults ages 18–24; 32.2% among adults ages 25 and older). Co-use of alcohol and cigarettes was the most common tobacco-alcohol co-use pattern with a prevalence of 3.7% among young adults ages 18–24 and 5.4% among adults ages 25 and older. Other tobacco-alcohol co-use patterns included use of alcohol with hookah (1.9% among young adults; 0.2% among adults ages 25 and older), alcohol with both cigarettes and e-cigarettes (1.3% among young adults; 1.4% among adults ages 25 and older), alcohol with both cigars and cigarettes (1.2% among young adults; 0.8% among adults ages 25 and older), alcohol with cigars (0.9% of young adults and 1.1% of adults ages 25 and older), and alcohol with other tobacco products (0.7% of young adults and 1.0% of adults ages 25 and older). However, estimates of tobacco and alcohol co-use prevalence in this study refer to exclusive use of particular products. For example, the estimated co-use prevalence of alcohol and cigarettes was defined as use of only alcohol and cigarettes, without use of any other tobacco or alcohol products (or cannabis).

Studies using NSDUH data have also examined co-use patterns among adults. Weinberger and colleagues (2019)[136] analyzed data from the 2002–2016 NSDUH to examine trends in cigarette smoking prevalence among people ages 12 and older with and without a past-year AUD. The prevalence of smoking decreased over time among both those with and without an AUD; however, in each year of the study, smoking was more than twice as common among people with an AUD than among those without an AUD. For example, in 2016, the prevalence of cigarette smoking was 37.8% among people with an AUD and 16.3% among those without an AUD. Additionally, the prevalence of smoking was higher among people with severe AUD (52.2%) than among those with mild AUD (34.6%) or moderate AUD (35.4%). Pham and colleagues (2024)[137] analyzed data from the 2010–2019 NSDUH to examine tobacco use and nicotine

dependence among people with no alcohol use, "low-risk" alcohol use (≤7/14 drinks per week for women/men), and "high-risk" alcohol use (>7/14 drinks per week for women/men). In all study years, past-month tobacco use prevalence was consistently higher among people who used alcohol, compared to those who did not use alcohol. Prevalence of nicotine dependence was similar between the no alcohol use and low-risk alcohol use groups but higher in the high-risk alcohol use group. Among people who reported high-risk alcohol use, younger age (versus ages 65 or older) and being male (versus female) were associated with both increased odds of tobacco use and increased odds of nicotine dependence. Another study used NSDUH data (2002–2019) to examine co-use patterns of cigar smoking among adults.[138] In this study, one of the few to examine co-use of cigars with alcohol, past-year alcohol or substance use disorders were associated with an increased likelihood of past-30-day cigar smoking, compared to adults without such disorders (aORs from 1.70 to 3.26).

Osibogun and colleagues (2022)[139] used 2020 Behavioral Risk Factor Surveillance System (BRFSS) data to examine the relationship between heavy alcohol consumption (>14 drinks/week for men; >7 drinks/week for women) and current every or some day use of cigarettes, e-cigarettes, and dual use of cigarettes and e-cigarettes among adults ages 18 and older. Heavy alcohol consumption was higher among those who reported cigarette use (12.3%), e-cigarette use (13.3%), or dual use of cigarettes and e-cigarettes (14.7%) than among those who reported not using a tobacco product (3.2%). Heavy alcohol consumption was associated with higher odds of cigarette use, e-cigarette use, and dual use of cigarettes and e-cigarettes than no heavy alcohol consumption (aORs ranging from 3.02 to 4.78). Finally, a study using 2016–2017 BRFSS data found that adults who used smokeless tobacco were more likely to engage in heavy alcohol use (>14 drinks/week for men; >7 drinks/week for women) than adults who did not use smokeless tobacco (15% vs. 6%, respectively). Use of smokeless tobacco was associated with increased odds of heavy alcohol consumption (aOR 2.36, 95% CI = 2.17–2.56).[140]

Several studies also looked specifically at co-use among young adults, typically defined as adults ages 18–26.[141] This developmental period, which bridges adolescence and independent adulthood, brings numerous transitions, challenges, and obligations as young people complete their schooling, enter the workforce, and become more independent. As during adolescence, young adults may engage in health risk behaviors, with significant consequences for later life. Studies of young adults indicate that co-use of tobacco and alcohol is common, and that the number of cigarettes smoked is positively associated with number of drinks consumed.[17,135,142,143] A 21-day diary study of college students (N = 86) found that co-use was predicted by number of drinks (aOR = 1.18; Beta = 0.16, standard error [SE] = 0.03) and being at a party (aOR = 3.57, Beta = 1.28, SE = 0.30) or at a bar (aOR = 2.17; Beta = 0.78, SE = 0.36) compared to other locations.[17] Three times as many cigarettes were reported on drinking occasions, compared with not drinking (3.35 vs. 0.85 cigarettes), and an average of 5.16 drinks were consumed while smoking versus 4.05 consumed while not smoking.[17] In a sample of 18- to 25-year-olds (N = 1,405) who reported cigarette and alcohol use in the past 30 days, cigarettes were smoked on 85.7% of the days on which binge drinking was reported.[142] Those who reported binge drinking four or more days in the last month also reported smoking more cigarettes per day than those who reported zero binge drinking days. Further, a study of college students (N = 341) found that any cigarette smoking and e-cigarette use (versus no cigarette smoking or e-cigarette use) were associated with increased odds of same-day alcohol co-use (cigarette smoking:

aOR = 3.18, 95% CI = 2.45–4.12; e-cigarette use: aOR = 2.51, 95% CI = 2.05–3.06), and drinking alcohol (versus not drinking alcohol) was associated with increased odds of same-day cigarette smoking (aOR = 3.29, 95% CI = 2.53–4.29) and same-day e-cigarette use (aOR = 2.54, 95% CI = 2.08–3.10).[143]

Box 2.2. Trends in Use and Co-Use of Tobacco and Alcohol During the COVID-19 Pandemic

A global outbreak of a novel human coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), began in December 2019. The outbreak was declared a pandemic by the World Health Organization in March 2020, and the United States also declared a national emergency in March 2020, which was lifted in May 2023.[144,145] The pandemic resulted in lockdowns, closures, stay-at-home orders, social distancing recommendations, and restrictions on numerous activities to limit the spread of the virus, although the implementation of these policies varied considerably by jurisdiction.[146-149] In the United States, the pandemic is estimated to have caused more than 1.2 million deaths between January 1, 2020, and May 31, 2025,[146] and caused significant economic and social disruption.[150,151] The impact of the pandemic on excess mortality varied substantially within the population.[152] The pandemic also increased social isolation, worsened physical and mental health, and disrupted health services, among many other consequences, with some subpopulations groups experiencing worse outcomes than others.[153-155] Additionally, the pandemic affected surveillance, as surveys had to interrupt their procedures midstream, affecting analyses of trends over time.[156] There were also substantial and lingering effects on study participation and response rates.

A limited number of nationally representative studies have assessed the COVID-19 pandemic's impact on tobacco and alcohol co-use. A study using nationally representative PATH data examined changes in past-30-day tobacco and alcohol use prevalence (separately) between 2018–2019 and 2020 and found that prevalence of both tobacco use and alcohol use declined among adolescents ages 13–17.[157] Among adult PATH participants, tobacco use prevalence declined for all age groups between the two time periods. Alcohol use prevalence did not change except among those ages 21–24; in this age group, use increased from 60.2% to 65.2% between 2018–2019 and 2020. A study using data from the Adolescent Brain Cognitive Development (ABCD) Study, a prospective, longitudinal, nationwide cohort of children ages 9–10 years at enrollment, examined substance use among 7,842 adolescents who completed 3 assessments between May and August 2020.[158] Prevalence of past-30-day individual substance use remained fairly stable across the three assessments. Collapsing data across all timepoints, the study observed that 20% of adolescents reporting alcohol use also used tobacco products (cigarettes, e-cigarettes, cigar, hookah, pipe, or smokeless tobacco), and that 21% of adolescents reporting tobacco use also used alcohol. In another study, Bandi and colleagues (2022)[159] used BRFSS data to examine smoking cessation attempts by adults (ages 18 and older) during the COVID-19 pandemic and correlates of any changes in smoking cessation behavior from 2011 to 2020. Although the study did not investigate tobacco and alcohol co-use per se, the researchers reported that, for the first time since 2011, past-year smoking cessation attempts decreased (from 65.2% in 2019 to 63.2% in 2020), and that the decline in smoking cessation attempts did not vary by past-30-day heavy drinking status (self-report of >14 drinks/week among men and >7 drinks/week among women).

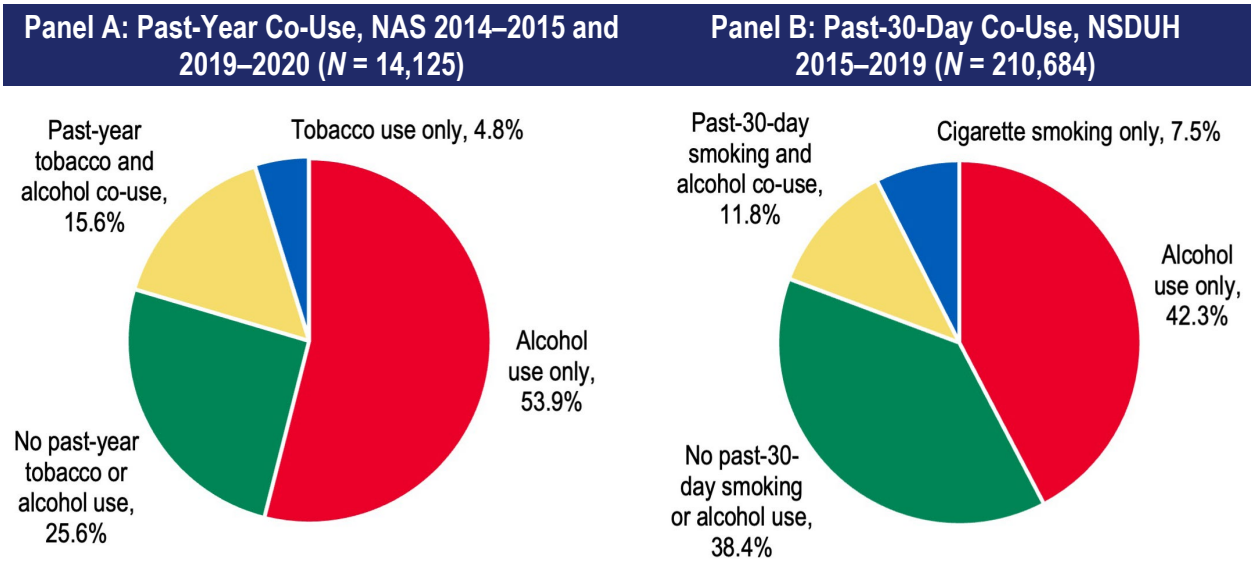
Continued efforts to improve surveillance of co-use of tobacco and alcohol in multiple demographic groups can inform public health efforts and determine whether pandemic-related changes in substance use patterns persist.

National Alcohol Surveys and National Survey on Drug Use and Health Analyses of Tobacco and Alcohol Co-Use

This section presents overall population tobacco and alcohol co-use prevalence and past-year and past-30-day alcohol use by tobacco/smoking status from new analyses of both the pooled 2014–2015 and 2019–2020 NAS and the pooled 2015–2019 NSDUH. Past-year average number of drinks consumed and alcohol use by setting, stratified by tobacco use status, are also presented based on data from NAS. Finally, this section describes NAS data on lifetime heavy drinking, defined as the number of days on which five or more drinks were consumed, by tobacco use status and cigarette pack years (the number of packs smoked per day multiplied by the number of years smoked). An overview of the demographics of the pooled 2014–2015 and 2019–2020 NAS is provided in Appendix Table 2D.5, and an overview of the participant demographics of the pooled 2015–2019 NSDUH is provided in Appendix Table 2D.6.

Overall, 15.6% (95% CI, 14.7%–16.6%) of NAS participants reported past-year co-use of tobacco and alcohol; 11.8% (95% CI, 11.7%–12.0%) of NSDUH participants reported past-30-day co-use of cigarettes and alcohol (Figure 2.4; Appendix Table 2D.7). More than one-half (53.9%, 95% CI = 52.7%–55.1%) of NAS participants and 42.3% (95% CI, 42.0%–42.6%) of NSDUH participants reported only alcohol use in the past year and past 30 days, respectively (Appendix Table 2D.7). Regarding only tobacco use, 4.8% (95% CI, 4.3%–5.4%) of NAS participants reported only past-year tobacco use (defined as use of tobacco products other than e-cigarettes), while 7.5% (95% CI, 7.3%–7.6%) of NSDUH participants reported only past-30-day cigarette smoking (Appendix Table 2D.7). Differences in levels of co-use between the NAS and NSDUH may be attributed to survey year, different measurement timeframes (past year in NAS; past month in NSDUH), tobacco product measured (cigarette smoking in NSDUH; tobacco products other than e-cigarettes in NAS), and other factors. Using the estimated U.S. adult population for 2019 (251 million)[160] these percentages indicate that approximately 39.2 million U.S. adults reported co-use of tobacco and alcohol in the past year and about 29.6 million in the past month.

Figure 2.4 Co-Use Prevalence in 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125) and 2015–2019 National Survey on Drug Use and Health (*N* = 210,684) Among Adults Ages 18 and Older



Note. NAS = National Alcohol Surveys; NSDUH = National Survey on Drug Use and Health. For full data, see Appendix Table 2D.7. *Tobacco use* in the NAS is defined as use of tobacco products (other than e-cigarettes) in the past year.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys, 2015–2019 National Survey on Drug Use and Health.

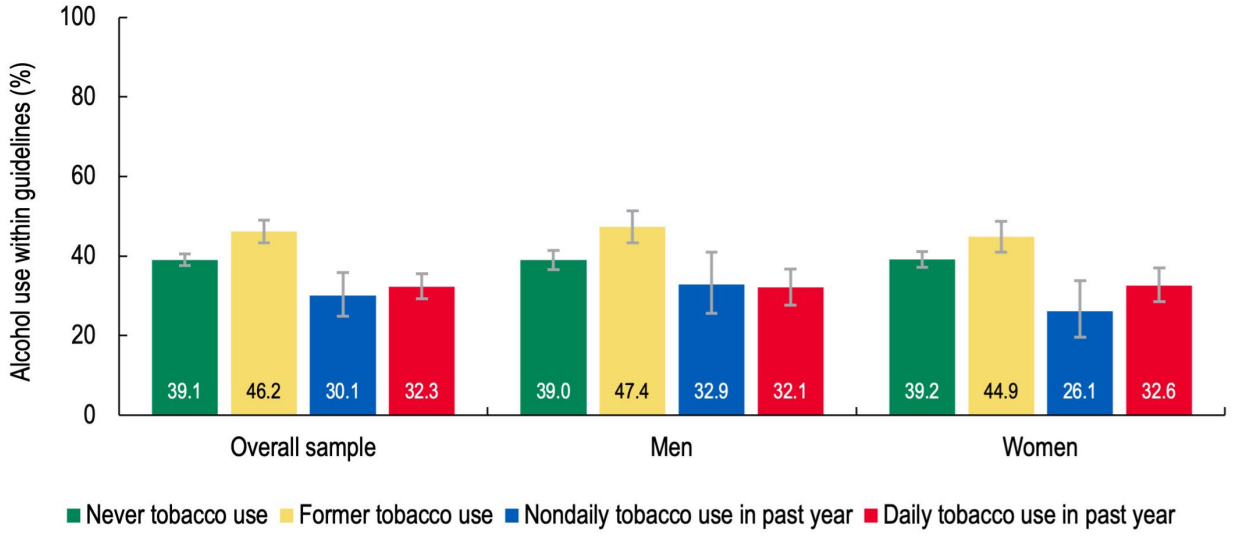
National Alcohol Surveys Analysis: Past-Year Co-Use of Tobacco and Alcohol: Overall and by Age and Sex. Figure 2.5 (Appendix Tables 2D.8 and 2D.9) shows past-year alcohol use prevalence by past-year tobacco use status (never, former, nondaily, or daily) for the pooled NAS sample, for men and women, and by age group. There was a statistically significant bivariate association between tobacco use and alcohol use ($p < .001$). Overall, people who never used tobacco were the most likely to report no alcohol use (35.1%, 95% CI = 33.7%–36.6%). In contrast, people who used tobacco nondaily were the least likely to report no alcohol use (13.8%, 95% CI = 10.5%–18.0%), and the most likely to report alcohol use exceeding guidelines: more than one-half (56.1%, 95% CI = 50.0%–61.9%) of those who used tobacco nondaily reported alcohol use exceeding guidelines (55.0% of men [95% CI, 46.7%–63.0%]; 57.6% of women [95% CI, 48.9%–65.8%]). Among people who used tobacco daily, 41.1% (95% CI, 37.7%–44.4%) reported alcohol use exceeding guidelines (45.2% of men [95% CI, 40.4%–50.1%]; 36.4% of women [95% CI, 32.0%–40.9%]).

Across age groups, those who never used tobacco had the highest prevalence of reporting no alcohol use and the lowest prevalence of reporting alcohol use exceeding guidelines. Alcohol use exceeding guidelines declined with age and was lowest among those in the oldest age group who never used tobacco (9.6%, 95% CI = 8.1%–11.4%). Conversely, across age categories, people using tobacco had the highest prevalence of alcohol use exceeding guidelines. Among participants who reported any past-year tobacco use, prevalence of alcohol use exceeding guidelines was highest among those ages 18–29 (61.9%, 95% CI = 54.9%–68.4%), followed by those ages 30–59 (45.4%, 95% CI = 41.6%–49.3%), and lowest among those ages 60 and older (22.2%, 95% CI = 17.8%–27.4%). The prevalence of alcohol use exceeding guidelines was

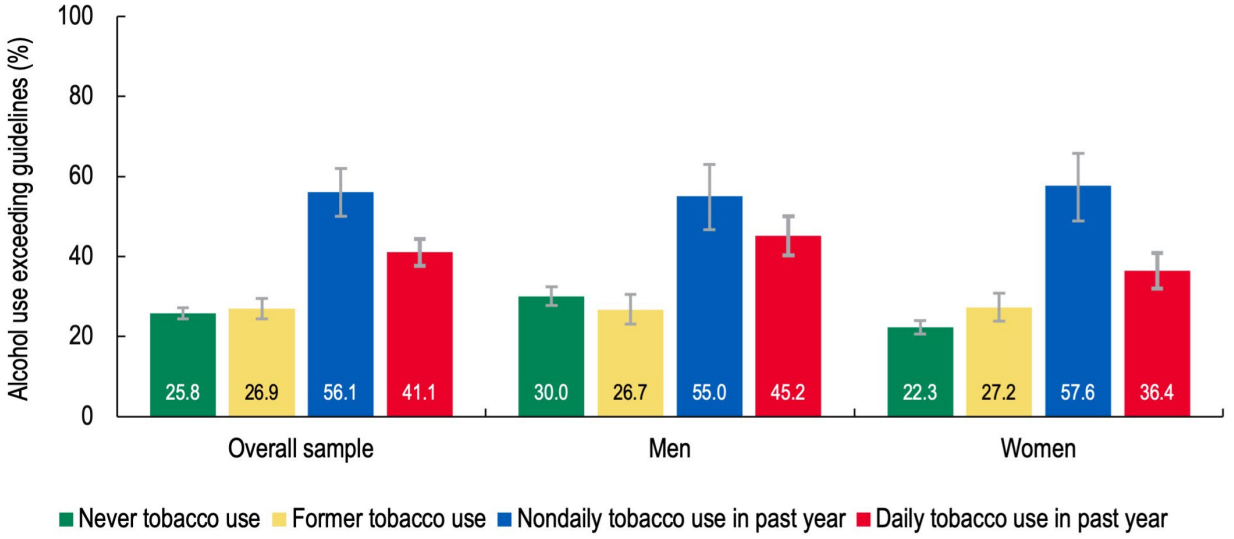
higher than for individuals reporting never using tobacco products in each respective age category (ages 18–29: 40.4% [95% CI, 37.1%–43.8%]; ages 30–59: 26.5% [95% CI, 24.5%–28.5%]; ages 60 and older: 9.6% [95% CI, 8.1%–11.4%]). Furthermore, people ages 30–59 and ages 60 and older who formerly used tobacco had a higher prevalence of exceeding drinking guidelines (ages 30–59: 35.1%, 95% CI = 31.0%–39.5%; ages 60 and older: 17.5%, 95% CI = 14.7%–20.6%) than people who never used tobacco, albeit to a lesser extent than those who used tobacco in the past year. Similar patterns were evident for both men and women; however, alcohol use among women was generally lower than among men across age groups and tobacco use groups (Appendix Table 2D.9).

Figure 2.5 Past-Year Alcohol Use by Past-Year Tobacco Use Status (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

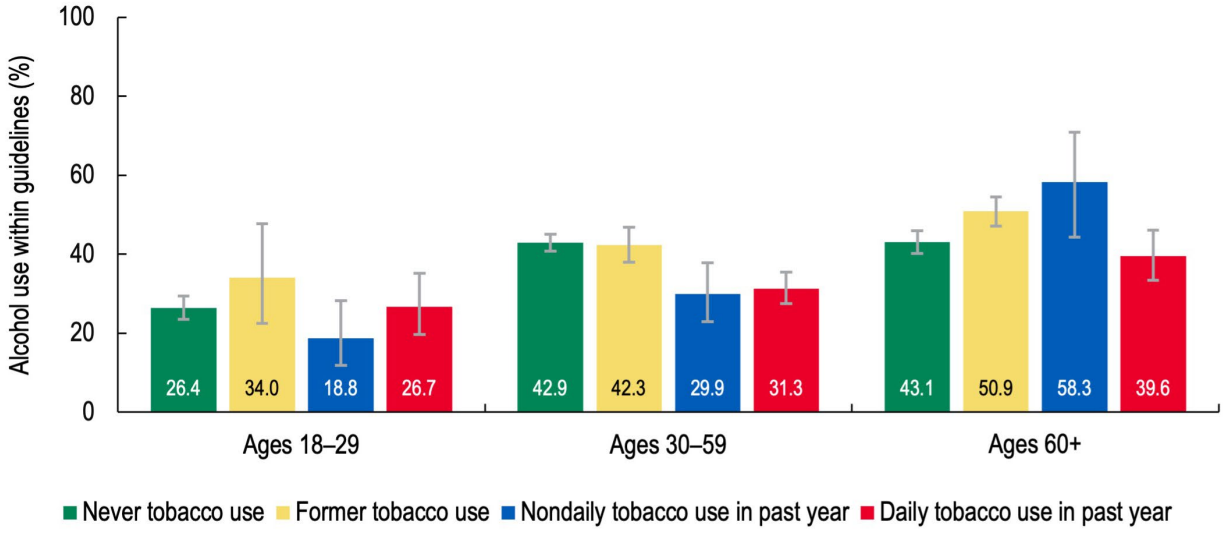
Panel A: Alcohol Use Within Guidelines, by Tobacco Use Status and Sex



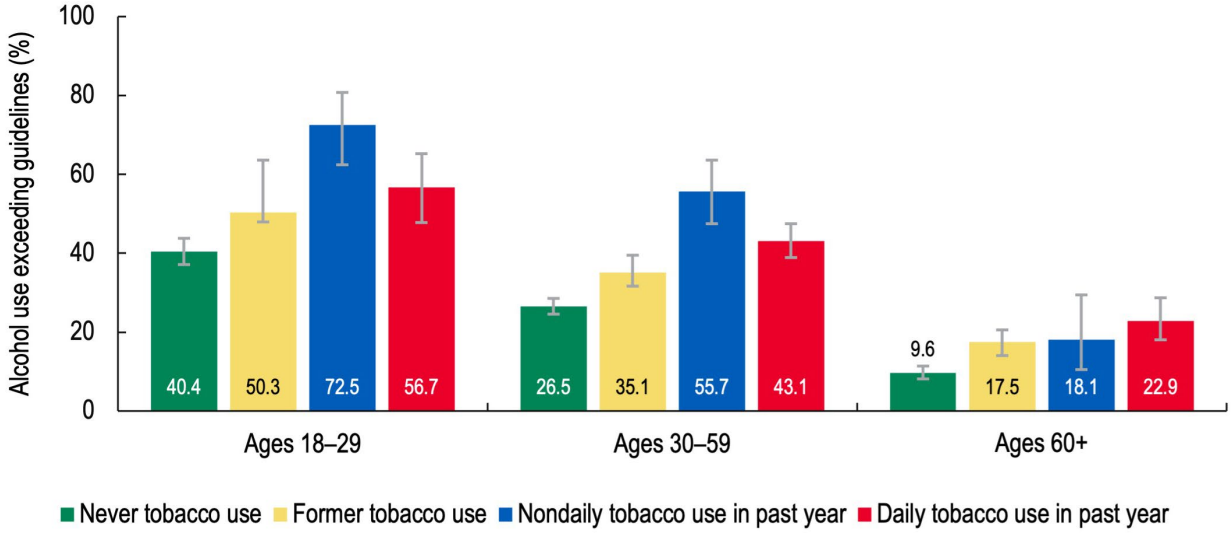
Panel B: Alcohol Use Exceeding Guidelines, by Tobacco Use Status and Sex



Panel C: Alcohol Use Within Guidelines, by Tobacco Use Status and Age



Panel D: Alcohol Use Exceeding Guidelines, by Tobacco Use Status and Age



Note. For full data, see Appendix Tables 2D.8 and 2D.9. *Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Nondaily* and *daily current tobacco use* are defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured in the past year. No past-year alcohol use is not shown in the charts. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66]
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

The association between tobacco use and hazardous alcohol use was also statistically significant at the $p < .05$ level when looking at more specific definitions of hazardous alcohol use: 5+ drinking (defined as having had 5 or more drinks in a day) and HID (defined as having had at least 8 drinks in a day for women and 12 for men). Overall levels of past-year 5+ drinking and HID declined with age (Appendix Table 2D.10). Among individuals ages 60 and older, 7.3% (95% CI, 6.3%–8.5%) reported past-year 5+ drinking and 1.2% (95% CI, 0.8%–1.7%) reported past-year HID. Among women in the oldest age group, 2.8% (95% CI, 2.1%–3.8%) reported

past-year 5+ drinking and 0.7% (95% CI, 0.4%–1.1%) reported past-year HID. Despite these overall declines with age, across all age groups, people who used tobacco either daily or nondaily had the highest levels of 5+ drinking and HID, while people who never or formerly used tobacco had the lowest levels of these hazardous alcohol use behaviors (Appendix Table 2D.10). For example, people younger than 60 who used tobacco nondaily had more than twice the prevalence of consuming ≥5 drinks on any day in the past year compared to those who never used tobacco (64.8% [95% CI, 53.7%–74.5%] vs. 30.6% [95% CI, 27.5%–33.8%] among those ages 18–29; 43.0% [95% CI, 35.2%–51.2%] vs. 19.2% [95% CI, 17.4%–21.1%] among those ages 30–59). Although hazardous alcohol use did decline with age, it remained more prevalent among individuals reporting past-year tobacco use than individuals who reported never using tobacco products across all age groups.

National Alcohol Surveys Analysis: Past-Year Co-Use of Alcohol and E-Cigarettes. Table 2.2 provides information about alcohol use prevalence by e-cigarette use status in the 2019–2020 NAS, which included questions on any past-year e-cigarette use; participants who reported e-cigarette use could also report never, former, or past-year use of cigarettes or other tobacco products. There was a statistically significant bivariate association between e-cigarette and alcohol use categories ($p < .001$). Overall, few participants reported use of e-cigarettes; 94.7% (95% CI, 93.9%–95.3%) reported no past-year e-cigarette use, 2.8% (95% CI, 2.4%–3.4%) reported monthly or semi-monthly e-cigarette use, and 2.5% (95% CI, 2.1%–3.0%) reported daily or nearly daily e-cigarette use (data not shown). Prevalence of e-cigarette use was similar among men and women, with 2.9% (95% CI, 2.2%–3.7%) of men and 2.2% (95% CI, 1.7%–2.9%) of women reporting daily or near daily e-cigarette use in the past year.

Among participants who used e-cigarettes, more than one-half reported alcohol use exceeding guidelines (54.1% [95% CI, 45.1%–62.9%] for those who used e-cigarettes monthly/semimonthly and 59.2% [95% CI, 49.9%–67.9%] for those who used e-cigarettes daily/nearly daily). Among all participants who used e-cigarettes, fewer than 1 in 5 reported no alcohol use (16.8% [95% CI, 11.1%–24.6%] among those who used e-cigarettes monthly/semimonthly and 13.8% [95% CI, 8.8%–20.9%] among those who used e-cigarettes daily/nearly daily). Stratifying NAS participants by age finds that, among people who used e-cigarettes, alcohol use exceeding guidelines was highest among those ages 18–29 (Appendix Table 2D.11).

Table 2.2 Past-Year Alcohol Use by Past-Year E-Cigarette Status in the 2019–2020 National Alcohol Survey (*N* = 8,308), Overall and by Sex *(Row Percents)*

E-cigarette use status (unweighted *n*)[a]	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]
	Alcohol use: Weighted % (95% CI)			
Overall				
No e-cigarette use in past year (7,927)	29.0 (27.7, 30.3)	42.6 (41.2, 44.1)	28.4 (27.1, 29.7)	<.001
Monthly or semimonthly use in past year (204)	16.8 (11.1, 24.6)	29.1 (21.5, 38.0)	54.1 (45.1, 62.9)	
Daily or nearly daily use in past year (177)	13.8 (8.8, 20.9)	27.1 (19.9, 35.7)	59.2 (49.9, 67.9)	

Table 2.2 (continued)

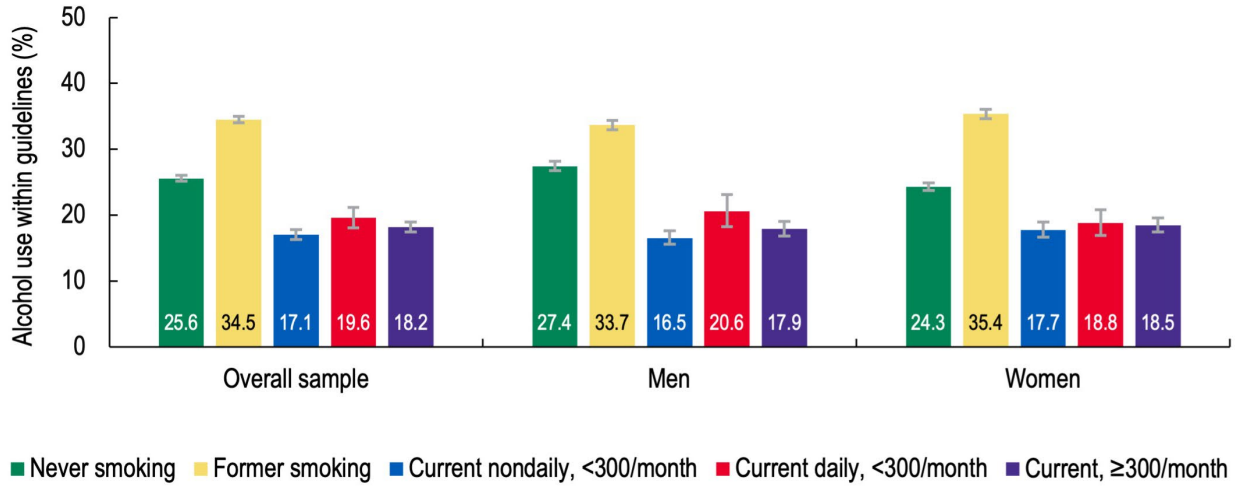
E-cigarette use status (unweighted *n*)[a]	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]
	Alcohol use: Weighted % (95% CI)			
Men				
No e-cigarette use in past year (3,200)	25.9 (24.0, 27.9)	43.4 (41.2, 45.7)	30.7 (28.6, 32.8)	<.001
Monthly or semimonthly use in past year (97)	13.7 (6.8, 25.6)	31.8 (21.1, 44.9)	54.5 (41.6, 66.7)	
Daily or nearly daily use in past year (83)	13.2 (6.5, 24.7)	29.0 (19.1, 41.5)	57.9 (44.8, 69.9)	
Women				
No e-cigarette use in past year (4,724)	31.7 (30.0, 33.6)	41.9 (40.1, 43.8)	26.3 (24.7, 28.0)	<.001
Monthly or semimonthly use in past year (107)	21.1 (13.0, 32.4)	25.2 (16.3, 36.7)	53.7 (41.8, 65.2)	
Daily or nearly daily use in past year (94)	14.5 (8.3, 24.1)	24.7 (15.5, 37.0)	60.8 (47.6, 72.6)	

Note. CI = confidence interval.
[a]*E-cigarette use* is measured within the past year. [b]Alcohol use is measured within the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66] [c]*p* value for bivariate association, obtained through a chi-squared test, between e-cigarette and alcohol use categories.
Source: 2019–2020 U.S. National Alcohol Survey.

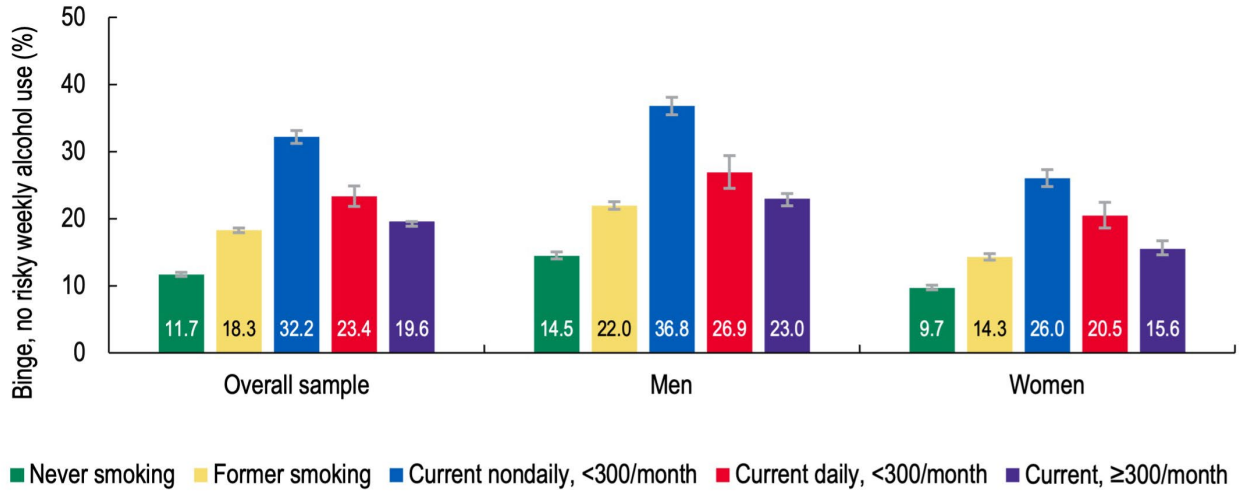
National Survey on Drug Use and Health Analysis: Past-30-Day Tobacco and Alcohol Co-Use. Figures 2.6, 2.7, and 2.8, as well as Appendix Tables 2D.12, 2D.13, and 2D.14, present the prevalence of alcohol use by cigarette smoking, smokeless tobacco use, and cigar smoking status for the 2015–2019 NSDUH sample. Similar to the findings of the NAS analyses, there was a statistically significant association between tobacco use and alcohol use ($p < .001$). People who never or formerly used cigarettes, smokeless tobacco, or cigars were the least likely to report engaging in either binge drinking (without risky weekly use) or risky weekly use. In contrast, people who used cigarettes, smokeless tobacco, or cigars in the past 30 days were the most likely to report engaging in these alcohol use behaviors (binge drinking or risky weekly alcohol use). For example, those who reported nondaily smoking <300 cigarettes/month were the most likely to report past month binge drinking (without risky weekly alcohol use) (32.2%, 95% CI = 31.3%–33.1%) or risky weekly alcohol use (19.8%, 95% CI = 19.0%–20.6%). Prevalence of alcohol use exceeding guidelines was somewhat lower among those who smoked daily. Among those who reported daily smoking <300 cigarettes/month, the prevalence of binge drinking (without risky weekly use) was 23.4% (95% CI, 21.9%–24.9%), and the prevalence of risky weekly use was 15.2% (95% CI, 14.0%–16.5%). Among those who smoked ≥300 cigarettes/ month, prevalence of binge drinking (without risky weekly use) was 19.6% (95% CI = 18.9%–20.3%) and prevalence of risky weekly use was 18.1% (95% CI = 17.4%–18.9%). Overall, among all NSDUH participants who reported past or current use of cigarettes, smokeless tobacco, or cigars, alcohol use exceeding guidelines (binge drinking and risky weekly use) was more prevalent than alcohol use within guidelines.

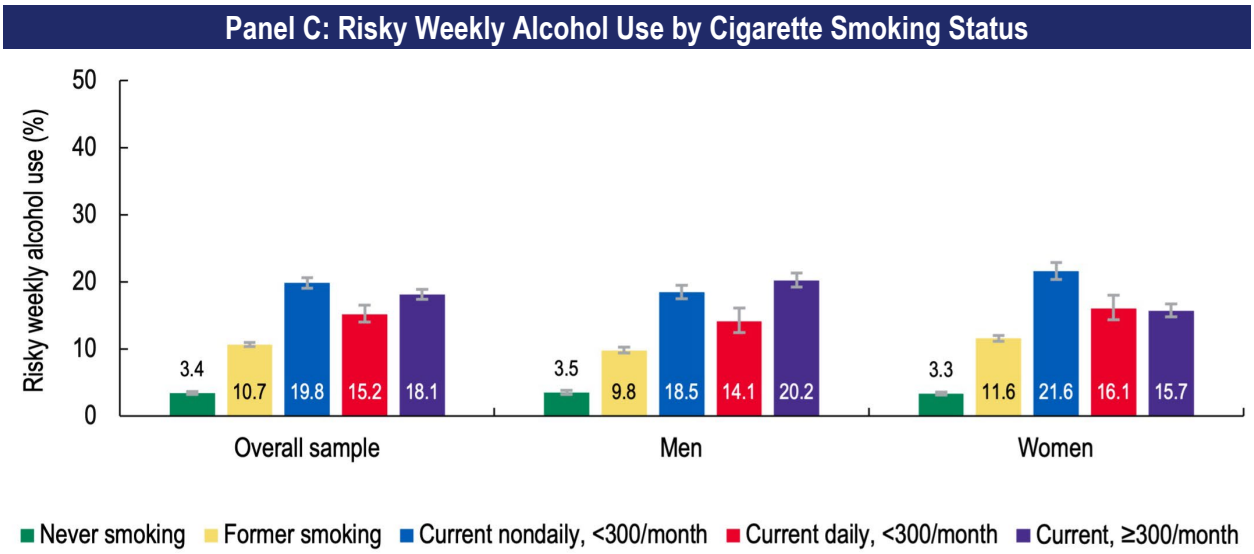
Figure 2.6 Past-30-Day Alcohol Use by Past-30-Day Cigarette Smoking Status, Overall and by Sex (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)

Panel A: Alcohol Use Within Guidelines by Cigarette Smoking Status



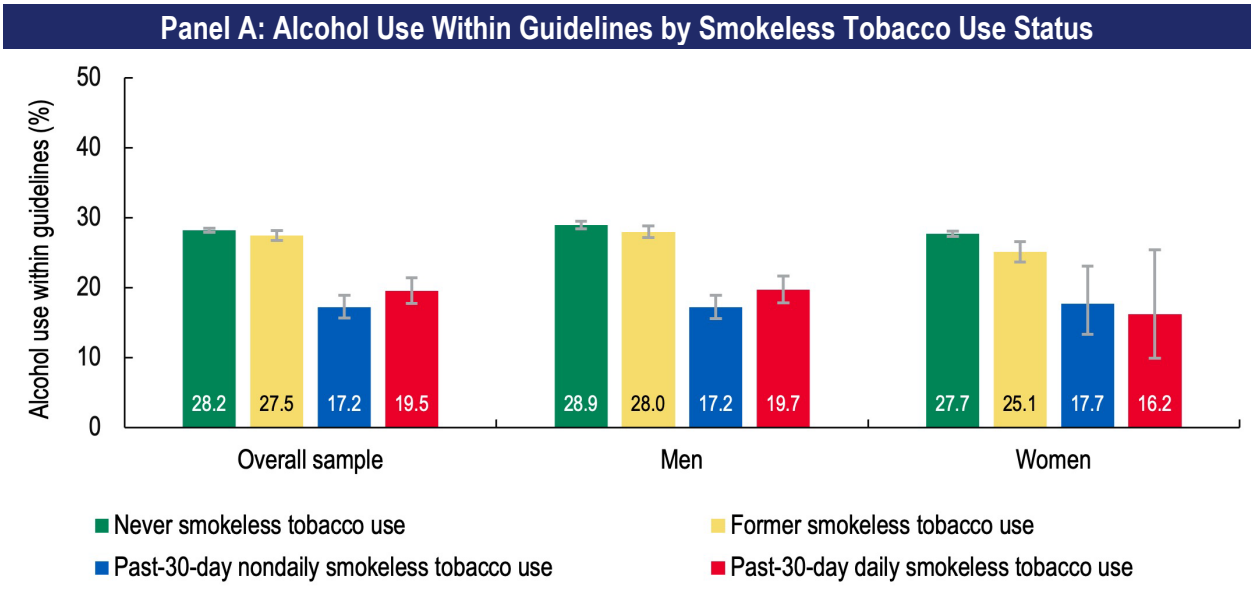
Panel B: Binge Drinking (Without Risky Weekly Use) by Cigarette Smoking Status



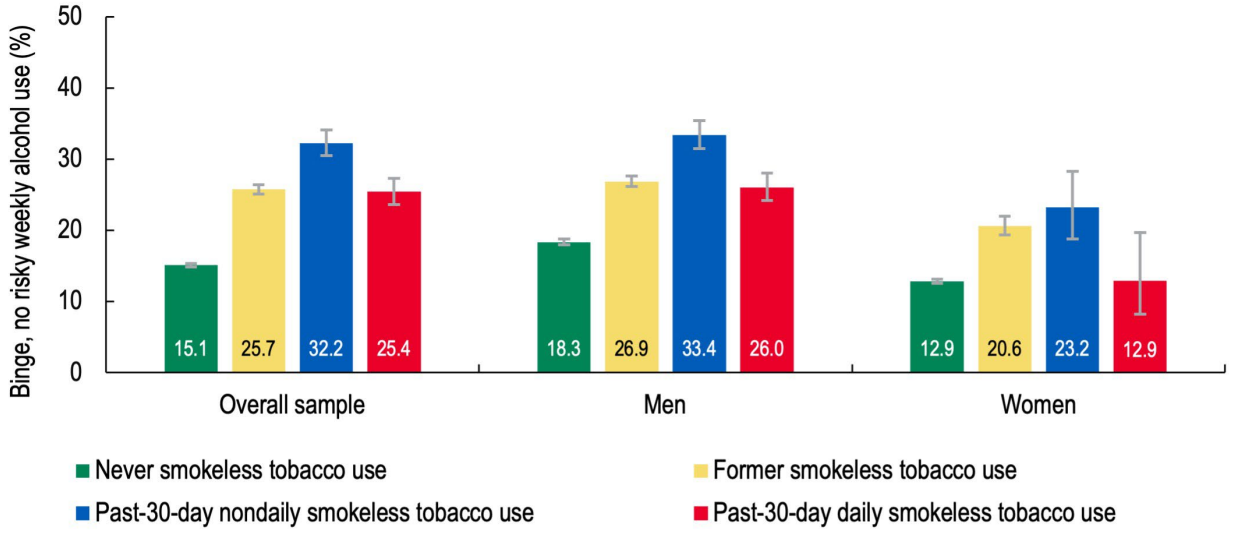


Note. For full data, see Appendix Table 2D.12. *Current cigarette smoking* is defined as use in the past 30 days and is presented by nondaily and daily frequency and number of cigarettes per month. Never and former alcohol use (not shown in the chart) are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

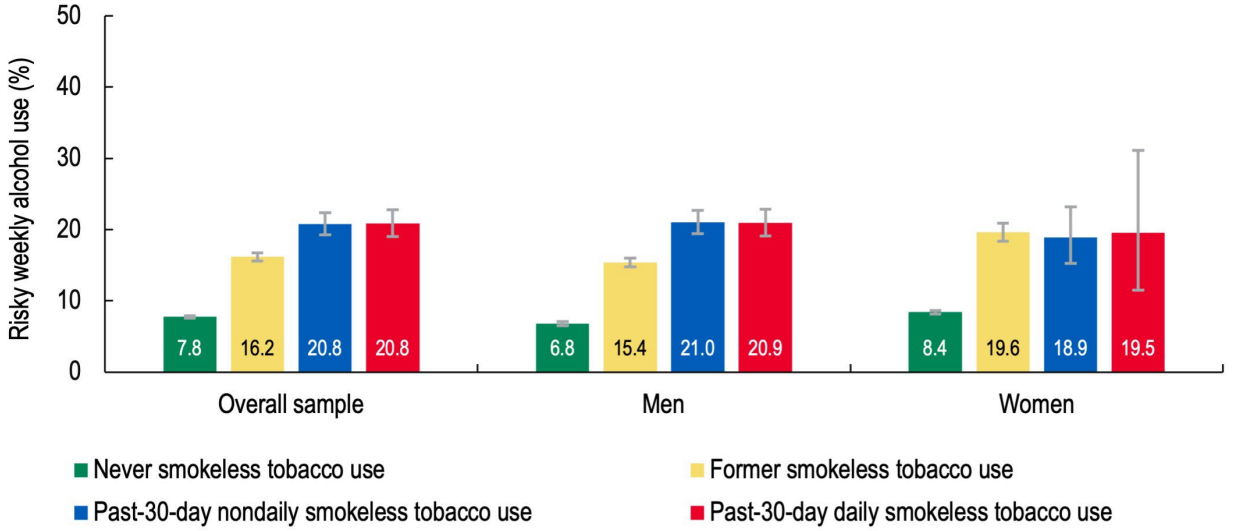
Figure 2.7 Past-30-Day Alcohol Use by Past-30-Day Smokeless Tobacco Use Status, Overall and by Sex (2015–2019 National Survey on Drug Use and Health; *N* = 210,931)



Panel B: Binge Drinking (Without Risky Weekly Use) by Smokeless Tobacco Use Status



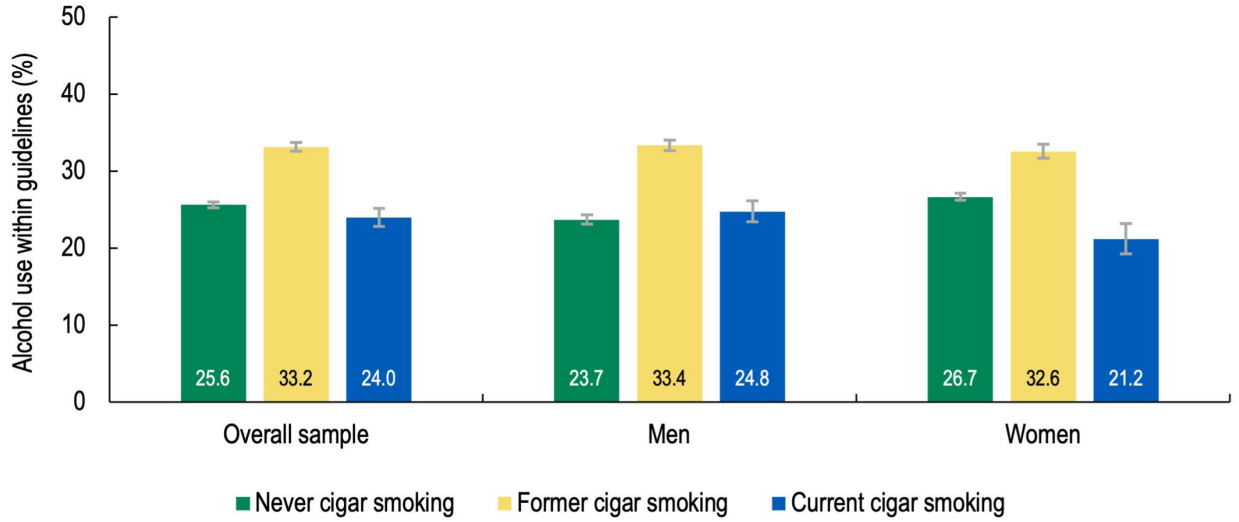
Panel C: Risky Weekly Alcohol Use by Smokeless Tobacco Use Status



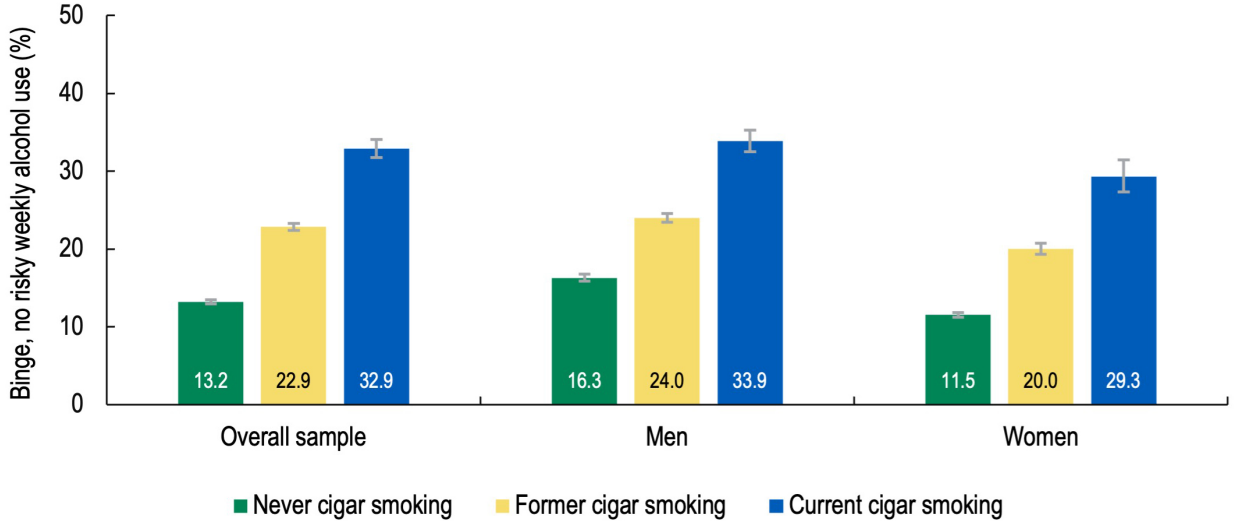
Note. For full data, see Appendix Table 2D.13. *Current use of smokeless tobacco* is defined as use in the past 30 days. Never and former alcohol use (not shown in the chart) are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Figure 2.8 Past-30-Day Alcohol Use by Past-30-Day Cigar Smoking Status, Overall and by Sex (2015–2019 National Survey on Drug Use and Health; *N* = 210,958)

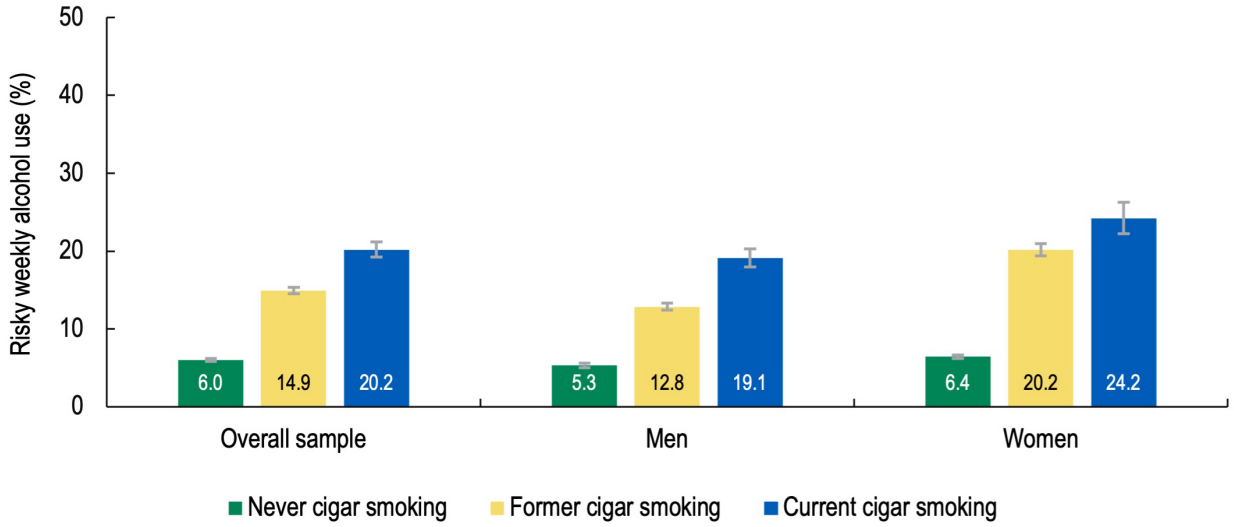
Panel A: Alcohol Use Within Guidelines by Cigar Smoking Status



Panel B: Binge Drinking (Without Risky Weekly Use) by Cigar Smoking Status



Panel C: Risky Weekly Alcohol Use by Cigar Smoking Status

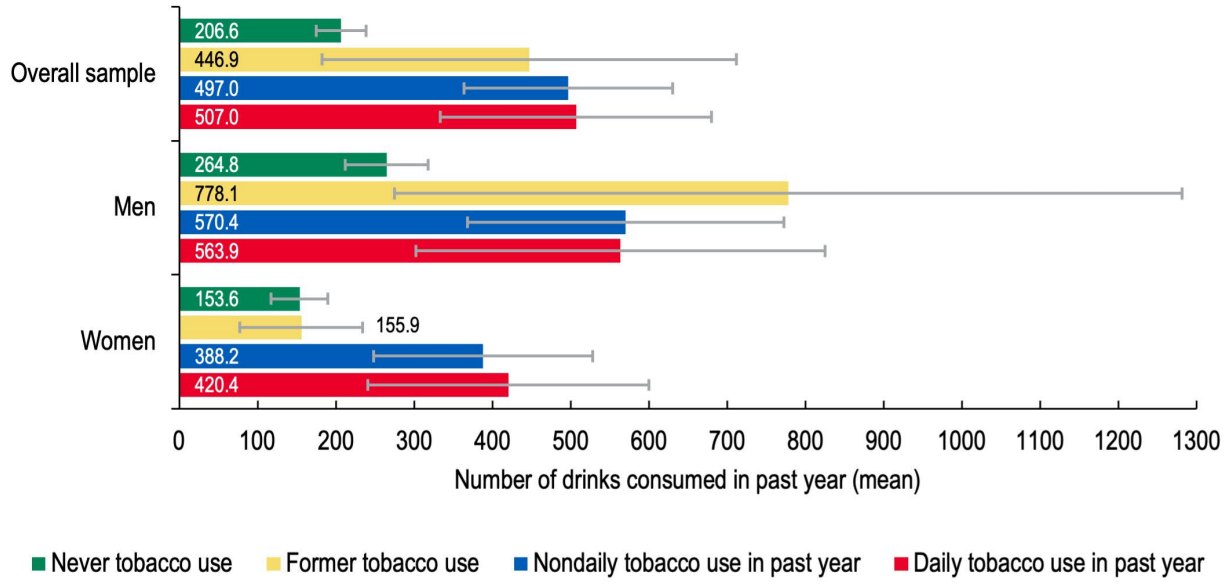


Note. For full data, see Appendix Table 2D.14. *Current cigar smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the chart) are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly* use is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

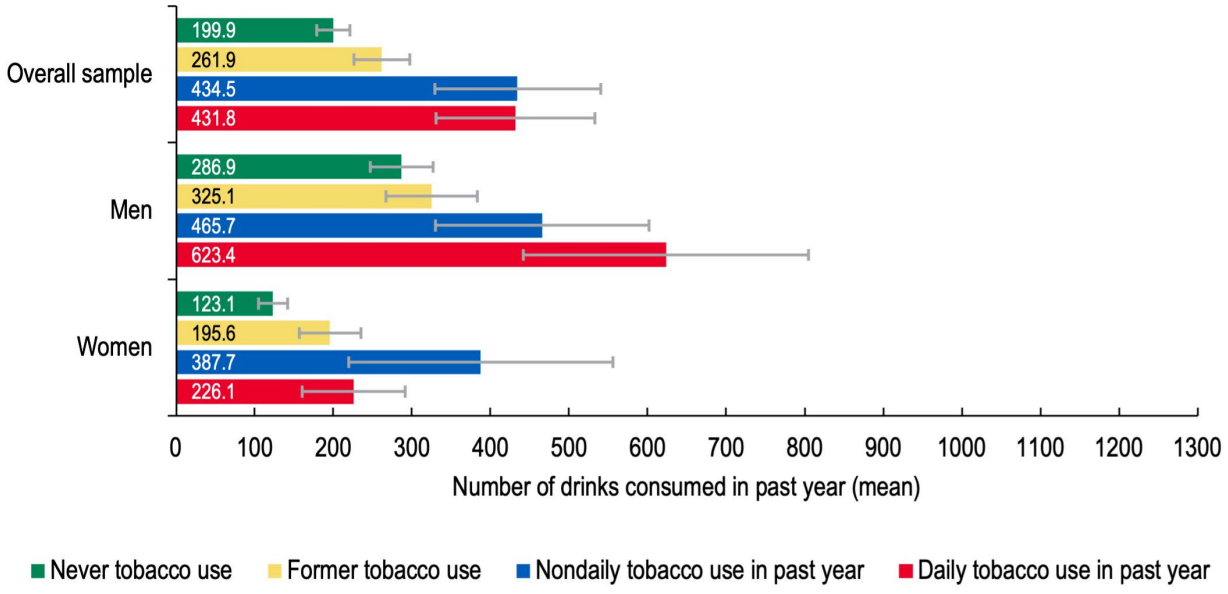
National Alcohol Surveys Analysis: Volume of Past-Year Alcohol Consumption.
This subsection summarizes past-year volume of alcohol consumption and tobacco use status using data from the pooled 2014–2015 and 2019–2020 NAS. Higher levels of alcohol consumption are associated with increased risk of cancer (see chapter 4). Among both men and women across age categories, people who reported past-year tobacco use had the highest overall past-year alcohol consumption (mean number of alcoholic drinks consumed), while those who reported never tobacco use had the lowest amount of alcohol consumption ($p < .01$; Figure 2.9 and Appendix Table 2D.15). Regardless of age or tobacco use status, overall past-year consumption was higher among men than among women. Alcohol consumption was largely driven by beer and spirits, especially among people younger than 60.

Figure 2.9 Average Number of Drinks Consumed in Past Year by Age, Past-Year Tobacco Use Status, and Sex (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

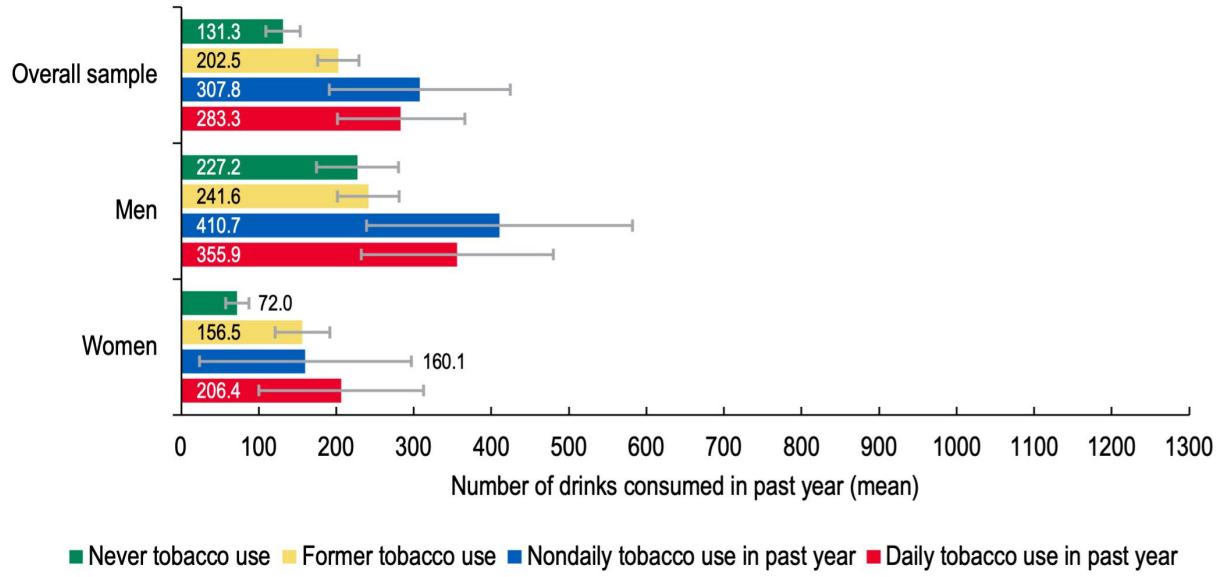
Panel A: Average Number of Drinks Consumed in Past Year, Ages 18–29



Panel B: Average Number of Drinks Consumed in Past Year, Ages 30–59



Panel C: Average Number of Drinks Consumed in Past Year, Ages 60 and Older

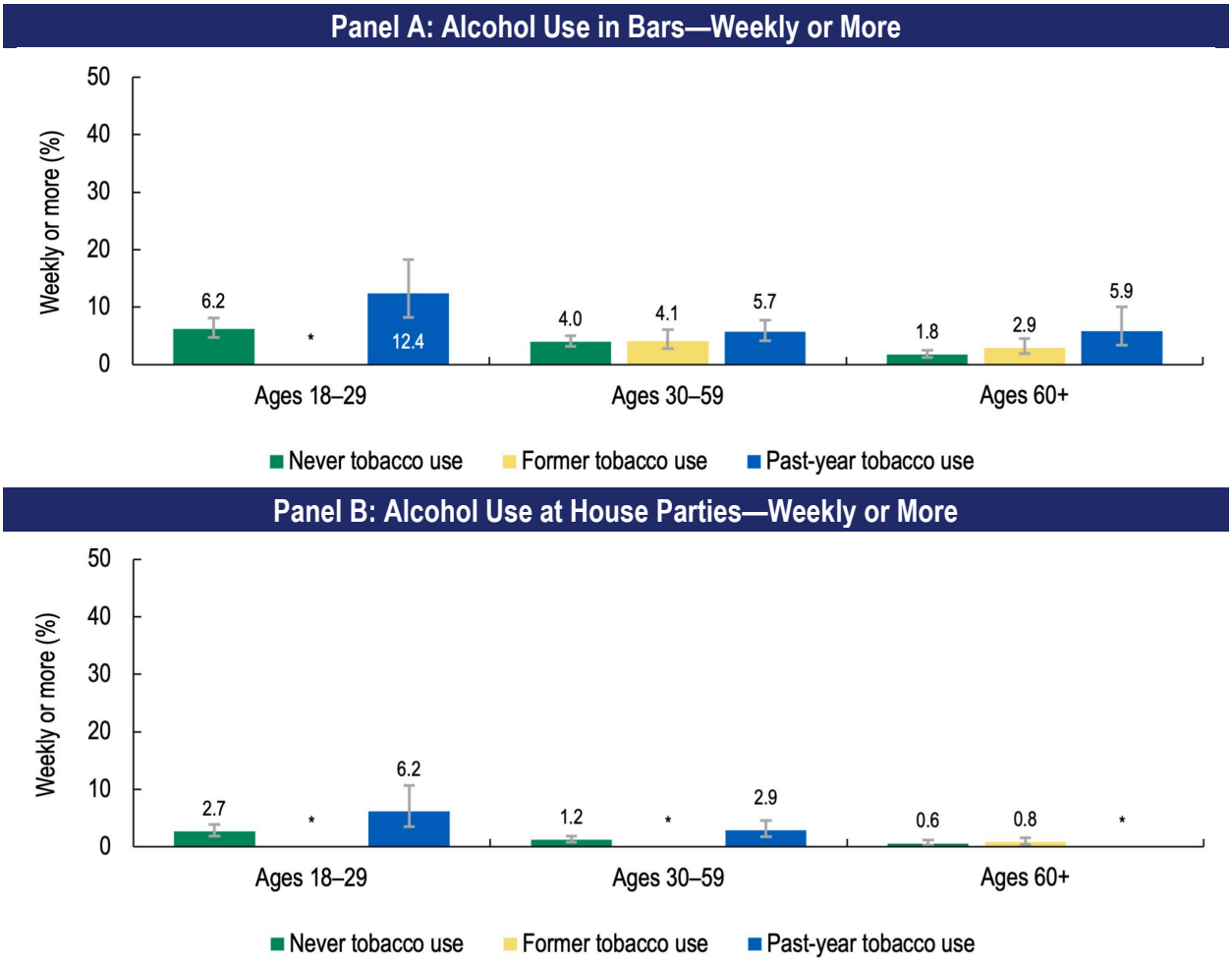


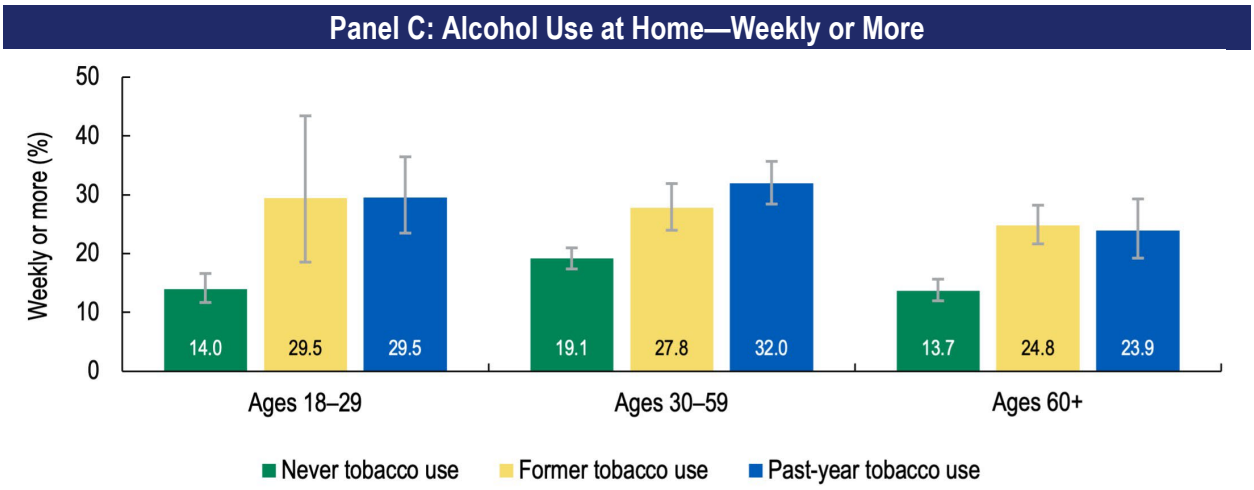
Note. For full data, see Appendix Table 2D.15. *Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured in the past year.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

National Alcohol Surveys Analysis: Drinking Context. This subsection summarizes past-year alcohol use by setting and tobacco use status using data from the pooled 2014–2015 and 2019–2020 NAS. Drinking setting (i.e., bars/taverns, parties, or in the home) can influence tobacco and alcohol use.[161-164]

Drinking context (bar, house party, or home) by tobacco use status is shown in Figure 2.10 and Appendix Table 2D.16. Regardless of tobacco use status, the home was the most common context for drinking weekly or more frequently, followed by bars and house parties. For all age groups, those who used tobacco in the past year had a greater likelihood of reporting weekly or more frequent alcohol use in the home than those who never used tobacco. Prevalence of weekly or more drinking at bars or house parties was low (<15% across age groups). Overall, there was a statistically significant bivariate association between tobacco use and drinking context categories ($p < .001$) except for drinking in bars among those ages 30–59).

Figure 2.10 Past-Year Alcohol Use by Setting on a Weekly Basis or More, by Past-Year Tobacco Use Status and Age (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)





Note. For full data, see Appendix Table 2D.16. *Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco* use is defined as never use in lifetime. Alcohol use is measured in the past year.
*Groups with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Lifetime Co-Use of Tobacco and Alcohol

The literature search identified several prospective studies of mortality that reported measures of lifetime tobacco or alcohol use. Data on lifetime smoking and alcohol use from three studies of prospective risks of alcohol use demonstrate a strong association between cigarette consumption and heavy alcohol use.[165-167] For example, in the 1984 and 1995 NAS, prevalence of current smoking (monthly or more in the past year) was 12.9% among lifetime alcohol abstainers, 24.3% among participants who formerly drank, and more than 50% among people who drank heavily (30–120 or more drinks per month for women and 60–120 or more drinks per month for men).[166] Among participants in the nationally representative 1988 NHIS who reported current drinking (12 or more drinks in the past year), current and former smoking were more strongly associated with increased frequency and quantity of drinking than never smoking.[165] In an analysis of pooled 1997–2014 NHIS data, participants who reported lifetime alcohol abstinence had the lowest prevalence of current smoking (9.0%), and those who reported current heavy drinking (>7 drinks/week for women and >14 drinks/week for men) had the highest prevalence of current smoking (46.0%).[167]

National Alcohol Surveys Analysis: Lifetime Tobacco Use and Heavy Alcohol Use. Data from the pooled 2014–2015 and 2019–2020 NAS were used to analyze lifetime tobacco use, cigarette pack years, and lifetime heavy drinking, defined as lifetime 5+ drinking days (i.e., the number of days in which ≥5 drinks were consumed). The average number of lifetime 5+ drinking days by tobacco use status, age, and sex is shown in Table 2.3. Never tobacco use is associated with the fewest 5+ drinking days for all age groups and for both men and women. Across age groups, those who used tobacco had more lifetime 5+ drinking days than those who never used tobacco. Among those ages 30 and older, the number of lifetime 5+ drinking days was also higher among those reporting daily or nondaily tobacco use than among those reporting former tobacco use, and men tended to report more 5+ drinking days than women. Additionally, those ages 60 and older tended to report fewer lifetime 5+ drinking days than those ages 30–59, despite being

older. This may reflect a combination of cohort effects (e.g., the older birth cohort having had less heavy lifetime drinking),[78] higher mortality among people with the highest level of tobacco use and alcohol consumption,[9,168] and age-related recall bias (i.e., older people may be less likely to recall or to accurately report any past heavy drinking).[169]

Data from the 2014–2015 NAS were used to analyze lifetime 5+ drinking days by lifetime cigarette consumption (among people who formerly smoked or smoked in the past year), expressed as cigarette pack years (Table 2.4). Overall and for both men and women, zero pack years was the most common category. For participants who smoked, 0.1–20 cigarette pack years was most common, followed by those who reported 40.1 or more cigarette pack years and those who reported 20.1–40 cigarette pack years. For all age groups and for both men and women, those who never smoked had the lowest likelihood of being in the highest category for number of lifetime 5+ drinking days (≥500 for those ages 18–29; ≥1,000 for those ages 30–59 and ages 60 or older). Across categories of cigarette pack years among those ages 30 and older, a greater percentage of men tended to reach the highest level of 5+ drinking days for their age group then women. Another notable feature of these data was the presence of a considerable proportion of adults, especially men, who reported 40.1 or more pack years of smoking and the highest level of 5+ drinking days for their age group.

Table 2.3 Average Number of Lifetime 5+ Drinking Days by Past-Year Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex, Stratified by Age

Tobacco use status[a]	Ages 18–29			Ages 30–59			Ages 60 and older		
	Overall	Men	Women	Overall	Men	Women	Overall	Men	Women
	Mean number of lifetime 5+ drinking days[b] (95% CI)								
Never (8,926)	140.5 (113.8, 167.5)	184.2 (134.1, 234.3)	100.8 (77.4, 124.1)	505.4 (448.3, 562.5)	746.0 (638.4, 853.6)	293.1 (248.2, 337.9)	333.8 (255.9, 411.7)	668.8 (487.0, 850.6)	126.6 (76.6, 176.7)
Former (2,537)	724.8** (409.6, 1,040.1)	1,047.2** (465.1, 1,629.3)	448.6** (269.0, 628.3)	1,109.8** (967.0, 1,252.6)	1,430.1** (1,184.3, 1,675.8)	774.6** (648.8, 900.3)	892.2** (755.0, 1,029.4)	1,434.0** (1,200.9, 1,667.1)	266.2** (185.8, 346.5)
Current (past year) (2,427)	550.1** (413.3, 686.9)	604.2** (402.8, 805.6)	468.4* (309.3, 627.4)	1,702.5** (1,510.5, 1,894.5)	2,085.6** (1,818.9, 2,352.3)	1,265.2* (992.9, 1,537.5)	1,827.1** (1,456.9, 2,197.3)	2,636.3** (2,047.2, 3,225.3)	934.9** (571.4, 1,298.3)
Nondaily (601)	413.0** (287.0, 539.0)	385.4** (248.6, 522.1)	454.0** (218.2, 689.7)	1,233.3** (1,001.9, 1,464.7)	1,358.9** (1,051.0, 1,666.9)	1,045.5** (687.6, 1,403.4)	780.3* (311.9, 1,249.8)	943.4 (233.4, 1,653.4)	546.0** (98.0, 994.0)
Daily (1,826)	616.6** (425.9, 807.2)	709.1** (424.6, 993.6)	475.5** (268.9, 682.0)	1,807.5** (1,579.9, 2,035.1	2,273.7** (1,953.1, 2,594.2)	1,306.1** (990.4, 1,621.8)	1,992.7** (1,574.0, 2,411.5)	2,943.5** (2,271.3, 3,615.7)	986.8** (580.5, 1,393.2)

Note. CI = confidence interval.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]*Lifetime 5+ drinking days* is defined as the number of days over lifetime in which participants reported drinking 5 or more drinks on 1 occasion.
*$p < .05$ for association comparing alcohol consumption among tobacco use categories (referent = Never), within each sex and age group. **$p < .01$ for association comparing alcohol consumption among tobacco use categories (referent = Never), within each sex and age group.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2.4 Lifetime Number of 5+ Drinking Days by Cigarette Smoking History (Pack Years) in the 2014–2015 National Alcohol Survey (*N* = 5,632), Overall and by Sex, Stratified by Age (*Row Percents*)

Cigarette pack years (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	0	0.1–499	≥500	*p* value[a]	0	0.1–999	≥1,000	*p* value	0	0.1–999	≥1,000	*p* value
	Lifetime number of 5+ drinking days[b]—Weighted % (95% CI)											
Overall												
0 (3,325)	50.8 (44.8, 56.7)	39.7 (33.9, 45.7)	9.6 (6.6, 13.8)	<.001	45.7 (42.1, 49.4)	39.6 (36.0, 43.4)	14.6 (12.0, 17.7)	<.001	77.6 (73.5, 81.1)	14.4 (11.5, 17.8)	8.1 (5.8, 11.2)	<.001
0.1–20 (1,204)	15.8 (9.8, 24.5)	49.3 (38.9, 59.7)	34.9 (25.6, 45.6)		19.6 (15.8, 24.0)	38.3 (32.9, 44.1)	42.1 (36.6, 47.9)		59.0 (51.4, 66.1)	18.9 (13.8, 25.4)	22.1 (16.2, 29.4)	
20.1–40 (382)	*	*	*		17.1 (10.5, 26.7)	35.3 (25.6, 46.5)	47.5 (37.1, 58.2)		34.6 (26.4, 43.8)	31.5 (23.1, 41.5)	33.9 (25.0, 44.1)	
≥40.1 (630)	27.0 (13.4, 47.0)	47.7 (29.9, 66.2)	25.3 (12.5, 44.4)		21.2 (14.8, 29.5)	35.8 (27.2, 45.5)	43.0 (33.7, 52.8)		41.6 (34.5, 49.1)	25.8 (20.0, 32.6)	32.6 (25.7, 40.3)	
Men												
0 (1,171)	47.0 (38.3, 55.9)	39.6 (31.2, 48.7)	13.4 (8.4, 20.7)	<.001	31.3 (26.3, 36.7)	45.8 (40.0, 51.7)	23.0 (18.3, 28.5)	<.001	60.1 (52.3, 67.5)	21.2 (15.9, 27.6)	18.7 (13.0, 26.2)	<.001
0.1–20 (514)	14.0 (6.7, 27.0)	46.9 (32.8, 61.5)	39.1 (26.0, 54.0)		11.5 (7.6, 17.1)	36.0 (28.3, 44.5)	52.5 (44.1, 60.7)		36.8 (26.4, 48.7)	21.9 (13.6, 33.2)	41.3 (30.1, 53.5)	
20.1–40 (189)	*	*	*		12.4 (5.5, 25.7)	29.6 (17.6, 45.1)	58.0 (42.9, 71.8)		18.0 (10.5, 29.2)	31.2 (20.3, 44.7)	50.8 (37.7, 63.8)	
≥40.1 (360)	*	53.9 (30.5, 75.7)	*		12.3 (6.5, 22.0)	39.2 (28.2, 51.5)	48.5 (36.6, 60.6)		28.7 (20.8, 38.3)	26.3 (18.9, 35.4)	44.9 (35.3, 55.0)	
Women												
0 (2,154)	53.8 (45.7, 61.8)	39.7 (32.1, 48.0)	6.4 (3.4, 11.9)	<.001	58.3 (53.5, 63.0)	34.3 (29.8, 39.1)	7.4 (5.2, 10.3)	<.001	86.7 (82.3, 90.1)	10.8 (7.7, 15.0)	2.5 (1.3, 4.9)	<.001
0.1–20 (690)	17.9 (9.5, 31.2)	52.0 (37.4, 66.4)	30.1 (18.1, 45.5)		27.5 (21.5, 34.4)	40.6 (33.1, 48.5)	31.9 (25.0, 39.7)		76.5 (67.7, 83.5)	16.6 (10.9, 24.5)	6.9 (3.2, 14.1)	
20.1–40 (193)	*	*	*		22.1 (11.9, 37.4)	41.4 (27.1, 57.3)	36.5 (23.5, 51.8)		58.7 (44.1, 71.9)	32.0 (20.0, 47.1)	9.3 (3.6, 22.1)	
≥40.1 (256)	*	*	*		39.7 (26.1, 55.1)	28.8 (16.9, 44.5)	31.5 (18.8, 47.9)		63.5 (52.4, 73.3)	25.0 (16.7, 35.6)	11.5 (6.4, 19.8)	

Note. CI = confidence interval.
[a]*p* value for bivariate association, obtained through a chi-squared test, between pack year and alcohol use categories, within each sex and age group. [b]*Lifetime 5+ drinking days* is defined as the number of days over lifetime in which participants reported drinking 5 or more drinks on 1 occasion.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 U.S. National Alcohol Survey.

Summary: Tobacco and Alcohol Co-Use Among Adults

New analyses of the pooled 2014–2015 and 2019–2020 NAS and 2015–2019 NSDUH find slightly lower levels of tobacco and alcohol co-use than found in earlier studies using 2001–2002 and 2012–2013 NESARC data (which reported co-use prevalence of more than 20%).[123,124] Overall, 15.6% (95% CI = 14.7%–16.6%) of NAS participants reported past-year co-use of tobacco and alcohol, and 11.8% (95% CI = 11.7%–12.0%) of NSDUH participants reported past-30-day co-use of cigarettes and alcohol. The lower estimate in NSDUH than in NAS could be due to differences survey year, measurement timeframes of use assessed (past 30-days vs. past year), types of tobacco products assessed (e.g., cigarettes vs. any non-e-cigarette tobacco product) and other factors.

The literature and analyses of 2014–2015 and 2019–2020 NAS data and 2015–2019 NSDUH data document a strong association between tobacco use and alcohol use. Similar to previous studies,[125,128,134,139] new analyses of the NAS and NSDUH found a higher prevalence of both any alcohol use and alcohol use exceeding guidelines among adults reporting use of tobacco, particularly those who used tobacco nondaily, than among those who reported never use of tobacco. More than one-half of NAS and NSDUH participants who reported nondaily tobacco use also reported hazardous alcohol use patterns (alcohol use exceeding guidelines, binge drinking, or risky weekly alcohol use). Among NSDUH participants reporting nondaily cigarette smoking, prevalence of past-30-day alcohol use exceeding guidelines was 52.0% (binge drinking, no risk weekly use: 32.2%, 95% CI = 31.3%–33.1%; risky weekly use: 19.8%, 95% CI = 19.0%–20.6%). Among NAS participants who reported using tobacco daily or nondaily in the past year, alcohol use was higher among adults ages 18–29 than among adults in other age groups and higher among men than among women. Overall prevalence of past-year use of alcohol exceeding guidelines was 56.1% (95% CI, 50.0%–61.9%) among participants who used tobacco nondaily in the past year. Specific thresholds of alcohol use exceeding guidelines, including 5+ drinking and high-intensity drinking, also reflect generally higher prevalence of these behaviors among adults reporting daily and nondaily tobacco use compared to adults reporting never using tobacco. Importantly, a fairly large proportion of adults, particularly men, report 40.1 or more pack years of smoking and the highest level of 5+ drinking days for their age group. This population is an especially important target for smoking cessation efforts.

A study of young adult participants in the 2002–2003 NESARC found that those who smoked nondaily had a higher likelihood of hazardous drinking than those who did not smoke,[128] aligning with the findings from the NSDUH and NAS data. Analysis of 2014–2015 and 2019–2020 NAS data finds that alcohol use at bars or house parties was particularly prevalent among adults ages 18–29 who reported past-year tobacco use. The association between nondaily tobacco use and alcohol use may be attributed in part to social smoking (i.e., smoking in the context of parties, bars, or nightclubs).

Analysis of the 2019–2020 NAS finds that alcohol use was common among adults who reported use of e-cigarettes in the past year. Compared to those who reported no e-cigarette use, adults who reported using e-cigarettes, whether monthly/semimonthly or daily/nearly daily, were less likely to report no past-year alcohol use and more likely to report past-year alcohol use exceeding guidelines. For example, among adults who reported daily or nearly daily e-cigarette

use, 59.2% reported alcohol use exceeding guidelines, compared with 28.4% of adults reporting no e-cigarette use. This analysis was limited by small sample size, but it does suggest that these tobacco-alcohol co-use patterns may extend across tobacco products.

Analyses of 2014–2015 and 2019–2020 NAS data found that, regardless of tobacco use status, men consume a higher average number of drinks per year than women. Among men ages 18–29, past-year drinks consumed was highest among those who reported former tobacco use. In contrast, among men ages 30 and older, past-year drinks consumed was highest among those who reported past-year tobacco use. Men also reported having a higher number of lifetime 5+ drinking days than women across all age groups and tobacco use categories except for women ages 18–29 who reported nondaily tobacco use.

Chapter Summary

Tobacco and alcohol products have long been among the most common addictive and carcinogenic substances used by adults and adolescents in the United States. Epidemiologic surveillance of patterns of use of these products, individually and together, contributes to the understanding of patterns of morbidity and mortality from cancer and serious diseases.

Nationally representative surveys provide self-reported data on alcohol use, tobacco use, and their co-use among U.S. adolescents, young adults, and older adults (this monograph defines co-use as use of tobacco and alcohol during the same reference period). Epidemiologic surveillance of tobacco and alcohol co-use is limited by reliance on self-reported data, which may underestimate use, and the fact that many surveys lack detailed measures of use for both tobacco and alcohol. Additionally, surveys do not always align with respect to the measures used; in particular, whether alcohol and/or tobacco use is defined as use in the past 30 days or past year, and the specific definition of hazardous alcohol use. Social desirability bias influences self-report of behaviors and could well influence self-report of tobacco and alcohol differently among different groups of people. The existing literature on tobacco and alcohol co-use is relatively limited, particularly as it regards nationally representative data. To address this gap, new analyses of three nationally representative surveys (Monitoring the Future, National Alcohol Surveys, and National Survey on Drug Use and Health) were conducted. The analysis of 2000–2023 Monitoring the Future study data examined co-use of tobacco and alcohol over time among U.S. students in 8th, 10th, and 12th grade. The 2015–2019 National Survey on Drug Use and Health analyses focused on past-30-day measures of co-use to estimate co-use prevalence and alcohol use prevalence by tobacco use status. The 2014–2015 and 2019–2020 National Alcohol Surveys analyses allowed estimates of co-use and alcohol use by tobacco use status that included more detailed information about past-year drinking, such as beverage-specific numbers of drinks consumed, frequency of drinking in different drinking contexts, and high-intensity drinking. Importantly, the National Alcohol Surveys were the only identified population survey that included detailed measures of lifetime accumulated cigarette smoking and alcohol use. Co-use of tobacco and alcohol is strongly influenced by short- and long-term trends in the use of each substance. Over many decades, overall adult cigarette smoking has declined significantly; among adults who smoke, the proportion who smoke nondaily (opposed to daily) has increased over time.

The marketplace for tobacco and alcohol products is more diverse than in the past, especially for tobacco. The tobacco product marketplace includes products such as e-cigarettes, heated tobacco products, and nicotine pouches, and can be expected to continue to evolve. The alcohol marketplace has always included products differing widely in alcohol content, but many new flavors and formulations across beer, wine, and spirits product types have been introduced in recent decades. This complicates efforts to measure the type and amount of alcohol used. Although overall alcohol consumption has declined over time, albeit at a far slower pace than cigarette smoking, alcohol is still the most common substance used by adults and adolescents.

Adolescence, commonly defined as the period from age 10 to age 19, is characterized by increasing autonomy, identification with peers, experimentation, and risk-taking; health risk behaviors often cluster during this important developmental period. Substance use, including tobacco and alcohol use, is commonly initiated in adolescence. (Although adolescents may also initiate cannabis use in conjunction with alcohol and/or tobacco use, cannabis use is beyond the scope of this monograph). Analysis of nationally representative surveys show that alcohol use and cigarette smoking, and their co-use among adolescents, have declined over the last few decades. However, the literature and a new analysis of Monitoring the Future study data confirm a continued close association between adolescent cigarette and alcohol use and extend this relationship to a wide variety of tobacco products, including cigarettes, e-cigarettes, cigars, and smokeless tobacco. Based on data from the 2023 Monitoring the Future study, more than two-thirds of 10th and 12th grade students who reported past-30-day cigarette smoking also reported past-30-day alcohol use and roughly one-half who reported past-30-day cigarette smoking also reported past-2-week binge drinking. Among 10th and 12th grade students who reported past-30-day nicotine vaping, more than one-half also reported past-30-day alcohol use, and about one-third reported past-2-week binge drinking. Additionally, use of more than one tobacco product appeared to place adolescents at higher risk for alcohol use and binge drinking than use of only one tobacco product.

The literature and new analysis of National Alcohol Surveys and National Survey on Drug Use and Health confirms the strong relationship between tobacco and alcohol use among adults. Data indicates that approximately 12%–16% of adults ages 18 and older engage in co-use of tobacco and alcohol products. This is lower than the percentage of adults who report use of only alcohol (roughly 42%–54%), higher than use of only cigarettes or tobacco products (roughly 5%–8%), and lower than no use of either substance (26%–38%). Nonetheless, 12%–16% represents as many as 1 in 8 U.S. adults, indicating that co-use of tobacco and alcohol products is not uncommon. Any past-year alcohol use was reported by 76% of National Alcohol Surveys participants who used tobacco in the past year, and any past-30-day alcohol use was reported by more than one-half of National Survey on Drug Use and Health participants who smoked cigarettes in the past 30 days.

The literature also finds a strong relationship between cigarette smoking and alcohol use disorders. Although the prevalence of smoking has decreased among adults with and without an alcohol use disorder, smoking is more than twice as common among people with an alcohol use disorder than among those without an alcohol use disorder. Smoking prevalence is also far

higher among adults with severe alcohol use disorder than among those with mild or moderate alcohol use disorder. Further, the literature finds a strong relationship between heavy alcohol consumption and use of diverse tobacco products, including cigarettes, e-cigarettes, and smokeless tobacco. New analysis of the pooled 2014–2015 and 2019–2020 National Alcohol Surveys and the 2015–2019 National Survey on Drug Use and Health provide more detailed information about patterns of co-use among adults ages 18 and older.

In particular, the analyses presented in this chapter show that individuals who do not use tobacco products are less likely to use alcohol or engage in hazardous drinking, whether defined as alcohol use exceeding guidelines, binge drinking, or other use patterns. Regardless of age, adults who reported never using tobacco products were among those most likely to report not using alcohol, and adults who reported never using tobacco products who did report using alcohol were the least likely to report exceeding guidelines. In contrast, adults who reported using tobacco products had the highest prevalence of alcohol use exceeding guidelines. A strong association was also found between tobacco use and hazardous alcohol use in the National Alcohol Surveys analysis, defined as either 5+ drinking days (days of drinking 5 or more drinks) or high intensity drinking (at least 8 drinks per day for women and 12 drinks per day for men). Overall, more than 40% of people who reported past-year tobacco use in the National Alcohol Surveys also reported past-year alcohol use exceeding guidelines. In the National Survey on Drug Use and Health, past-30-day binge drinking and/or risky weekly use was reported by more than one-half (52.0%) of people who smoked cigarettes nondaily <300 cigarettes/month, 38.6% of people who smoked daily <300 cigarettes/month, and 37.7% of people who smoked ≥300 cigarettes/month.
In general, alcohol use was lower among women than men and declined substantially across age groups regardless of tobacco use status. Although both tobacco and alcohol use decline considerably with age, co-use of these products persists. For example, among adults ages 60 and older who reported past-year daily use of tobacco, more than 20% of these individuals also reported past-year alcohol use exceeding guidelines.

The National Alcohol Surveys began measuring e-cigarette use in 2019–2020. Although limited by small sample size, analysis of this data finds that most adults (>80%) who used e-cigarettes in the past year also reported use of alcohol. Additionally, more than one-half of the adults who co-used alcohol and e-cigarettes engaged in alcohol use that exceeds guidelines. Thus, as with cigarette use, new analyses suggest that e-cigarette use is strongly associated with both alcohol use and hazardous alcohol use.

Analysis of the 2015–2019 National Survey on Drug Use and Health allows for examination of patterns of alcohol use with cigarettes, smokeless tobacco, and cigars. These data confirm that adults who never (or formerly) smoked cigarettes, used smokeless tobacco, or smoked cigars are the least likely to also engage in alcohol use and hazardous alcohol use (binge drinking or risky weekly alcohol use). Among adults who report use of tobacco products, those who use tobacco nondaily are at particular risk of hazardous alcohol use, which is consistent with analyses of the National Alcohol Surveys indicating that, overall, the highest prevalence of past-year alcohol use exceeding guidelines was among people who reported nondaily tobacco use. Nondaily smoking is more prevalent than in the past,[170] and major shifts in the availability and use of new tobacco products in the 2000s and of the use of multiple tobacco products may continue, complicating the understanding of tobacco and alcohol co-use.

Lifetime measures of cumulative tobacco and alcohol use are strongly associated with one another. Analysis of the 2014–2015 National Alcohol Surveys showed that nearly one-half of those ages 30–59 who reported 20 or more pack years of smoking also reported more than 1,000 5+ drinking days. Men reported more 5+ drinking days than women. Among adults ages 30 and older, the highest mean number of accumulated 5+ drinking days was among people who used tobacco in the past year; those who formerly used tobacco also reported more 5+ drinking days than those who never used tobacco.

Tobacco and alcohol co-use is a complex and changing aspect of substance use behavior. Product types, policies, and social norms change over time, sometimes rapidly, resulting in changes in behavior related to both tobacco and alcohol. Notable trends include the decline in adolescent alcohol and cigarette use, dramatic increases in use of e-cigarettes and other emerging tobacco products, rising use of alcohol by women, and the increased proportion of nondaily tobacco use. This dynamic landscape results in a variety of challenges for timely, accurate, and comprehensive surveillance.

Research Needs

Data on multiple types of products is necessary. The tobacco product marketplace has expanded over time and can be expected to continue to evolve. Studies to address the potential association between new and emerging tobacco products (e.g., e-cigarettes, heated tobacco products, and nicotine pouches) and alcohol and to track usage patterns over time are a high priority.

Validation of self-reported information could improve understanding of co-use patterns. All nationally representative surveys rely on self-report. Although prone to underestimate levels of tobacco and alcohol use, self-report is generally considered a good reflection of actual use patterns. However, periodic validation to assess the degree of measurement error inherent in self-report is needed, especially as the tobacco and alcohol product marketplace and social norms regarding use of these products continue to evolve. Furthermore, continued efforts to measure existing biomarkers, and identify new biomarkers, especially of long-term use characteristics, could benefit the accuracy of estimates from surveillance and epidemiological studies.

Further understanding of co-use patterns through measurement of simultaneous co-use and lifetime exposure would be informative. Few nationally representative studies include sufficiently detailed measures of tobacco and alcohol use to adequately describe co-use of these two substances. Efforts to define best practices for measures of co-use should be conducted that address appropriate time windows and account for age and sex-related variation in patterns of co-use. Simultaneous co-use is a common behavior pattern, which may influence some disease risks. Similarly, studies are needed to determine co-use patterns over the life course to better understand accumulated exposure, which is especially relevant to risks for cancer. Because the health effects of co-use may vary over the life course, it is also important to measure the developmental stage at which co-use occurs, including when co-use is heaviest. Measures may range from simple measures, such as years of drinking and average number of drinks per day, to those that are more sensitive to the timing of varying patterns of drinking and other details relevant to cancer risk. An example of varying life course alcohol use patterns is the decades measure approach used in the NAS, in which participants report the frequency of any drinking

and heavy (5+) drinking in each decade (e.g., teens, 20s, 30s) since their self-reported age of initiating alcohol use. Moving forward, any measure can be adapted to assess usage patterns over the lifetime including beverage- and context-specific measures and maximum drinks in a day. Measuring both tobacco and alcohol use by decades would facilitate assessment of co-use during each decade and allow for assessment of co-use with cannabis and hazardous substances.

Measurement of use intensity and use over time would help to further understand co-use patterns. There is also a need for tobacco and tobacco and alcohol use measures that capture varying intensity of use over the life course, as individuals often vary their use over time. Some diary studies (e.g., employing timeline followback or ecological momentary assessment) find that more cigarettes are smoked on days when alcohol drinking occurs.[14,15,17] However, to assess such patterns, measures must assess variability in the use of each product over time. One possibility is to follow the approach that has been used for alcohol and cannabis co-use and asking how often the substances are used together.[171] However, additional methodologic work is needed.

Modeling patterns of co-use of tobacco and alcohol and alcohol alone is needed to account for covariance of different demographic and behavioral variables. Few studies have conducted model-based estimation of patterns of co-use from national and regional data in the United States. Such efforts, along with models of changes in co-use patterns in recent years, could inform public health efforts, addressing demographic and geographic differences and evaluation of natural experiments.

Further understanding of contextual aspects of co-use is needed. Measures of context of and motivations/expectations for heavy tobacco and alcohol use episodes would be useful to understanding external drivers of heavy co-use and could inform the development of potential interventions to reduce the frequency and/or intensity of heavy co-use episodes. Such measures could include location of co-use (bar/club, house party, home), co-use patterns of people (e.g., partner, friends, strangers) in different contexts, and reasons for co-using.

Conclusions

1. **Tobacco and alcohol products are the most commonly used addictive and carcinogenic substances.** Adults who use tobacco (including cigarettes, smokeless tobacco, or cigars) are more likely to engage in alcohol use than those who do not use tobacco. Similarly, adults who use alcohol are more likely to use tobacco products than those who do not use alcohol. These associations are observed across all age groups, and for both men and women, although use prevalence varies among population groups.
2. **Co-use of tobacco and alcohol is common among adults.** A total of 15.6% of adults reported the co-use of tobacco and alcohol in the past year, representing approximately 39 million people, and a total of 11.8% of adults reported co-use of cigarettes and alcohol in the past month, representing nearly 30 million people. Co-use patterns vary across population groups based on age, sex, and other factors.
3. **Among adolescents, tobacco use and alcohol use often occur together.** Most adolescents who use a tobacco product also drink alcohol, and most adolescents who drink alcohol also use a tobacco product. This relationship extends beyond cigarettes to smokeless tobacco, cigars, and e-cigarettes. In 2023, co-use of nicotine-containing

e-cigarettes and alcohol was the most common tobacco and alcohol co-use pattern among adolescents. The literature strongly suggests that use of multiple tobacco products is an even stronger risk factor for alcohol use than use of one tobacco product or no use of tobacco products.

4. **Prevalence of alcohol use within guidelines and risky weekly alcohol use are similar in men and women, but prevalence of binge drinking without risky weekly alcohol consumption is greater in men than women.** These differences are seen in people across tobacco use categories.
5. **Tobacco and alcohol co-use is influenced by changes in the patterns of use of the individual substances.** Over the past 50 years, cigarette smoking among adults and adolescents has declined to historically low levels, but non-cigarette tobacco products such as e-cigarettes and nicotine pouches have entered the marketplace and are particularly prevalent among adolescents. In contrast, within the same period, the overall level of adult alcohol use has remained relatively stable while alcoholic beverage choice preferences have shifted away from beer and towards wine and spirits. In adolescents, alcohol use has declined steadily in both boys and girls since the 1970s.
6. **Adults who report nondaily use of tobacco in the past year or past month report the highest prevalence of any alcohol use and alcohol use exceeding guidelines compared to other tobacco use groups.** More than one-half of adults who use tobacco nondaily report alcohol use exceeding the 2020–2025 U.S. Dietary Guidelines for alcohol use, such as episodes of binge drinking. This is particularly true for young adults, which may reflect use of both substances in social settings. For example, among people ages 18–29 years, 72.5% (95% CI, 62.4%–80.8%) of people who used tobacco nondaily in the past year and 56.7% (95% CI, 47.8%–65.1%) of people who used tobacco daily in the past year reported alcohol use exceeding guidelines.
7. **Fewer older adults (age 60 and older) than younger adults use tobacco products, but those who do are also likely to use alcohol and to engage in alcohol use exceeding guidelines.** The co-use of tobacco and alcohol among older adults has important clinical implications, because this age group has the highest risk for cancer. Older men have higher prevalence of alcohol use and alcohol use exceeding guidelines than older women.
8. **Among both adults and adolescents, never use of tobacco is associated with a lower prevalence of alcohol use and lower levels of alcohol use exceeding guidelines.** People who report formerly using a tobacco product are at intermediate risk of alcohol use, falling between those who have never used tobacco and those who report current use.
9. **Assessment of trends and patterns of tobacco and alcohol co-use can be improved.** Although several nationally representative surveys measure tobacco and alcohol co-use, these measures rely on self-report, vary in the level of detail and time window with which they assess each substance, and tend not to assess use of tobacco and alcohol over the life course. Better alignment of these measures will allow for improved assessment and health outcomes due to co-use.

Appendix 2A National Surveys With Tobacco and Alcohol Use Measures

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Alcohol-focused surveys					
National Alcohol Survey (NAS)	**Overview**: The NAS is designed to assess trends in drinking in the national population, including drinking patterns and lifetime and current alcohol consumption. **Sponsors**: Alcohol Research Group, and the NIAAA	1964–present; conducted approximately every 5 years; 15 waves of data collection completed as of 2026	**Sample**: Nationally representative sample of U.S. noninstitutionalized adults **Design**: Cross-sectional **Administration**: Computer-assisted telephone interview	**Lifetime use** • Ever regular use of cigarettes, e-cigarettes, or other tobacco products ("tobacco cigarettes" or "any other kinds of tobacco;" e-cigarettes added in 2019–2020) • Age of initiation (not including e-cigarettes) • Use frequency when used tobacco most heavily • Time since last use **Past year use** • Use frequency in past 12 months (not including e-cigarettes in 2014–2015 and prior surveys) • Cigarettes per day	**Lifetime use** • Ever use • Largest number of drinks ever consumed at one time • Age of initiation • Frequency of drinking (overall and drinking 5+ drinks on an occasion) in 20s/30s/40s/50s/ **Past-year use** • Any use in past 12 months • Use frequency in past 12 months • Largest number of drinks consumed in a single day • Frequency of drinking 1 to ≥12 drinks in a single day • Frequency of drinking and typical number of drinks consumed at specific locations (bars, home, parties) **Current use** • Typical number of drinks consumed when drinking beer, wine, and liquor

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
National Epidemiologic Survey on Alcohol and Related Conditions (NESARC)	**Overview**: NESARC was designed to assess the prevalence of AUDs in the U.S. population. The survey includes questions about tobacco products including cigarettes, ENDS, cigars, pipes, and smokeless tobacco. **Sponsor**: NIAAA	Three waves: 2001–2002; 2004–2005; 2012–2013	**Sample**: Nationally representative sample of U.S. civilian, noninstitutionalized adults **Design**: Waves 1 and 2 longitudinal; Wave 3 cross-sectional **Administration**: Home-based, in-person interviews; saliva samples collected from Wave 3 respondents	**Lifetime use** • Ever use and ever daily use • Age of initiation • Time since last use • Typical use frequency and amount used in the year before quitting **Past-year use** • Any use in past 12 months • Typical use frequency and amount used in past 12 months **Cessation behavior** • Lifetime quit attempts • Quit attempts in past 12 months	**Lifetime use** • Ever use • Age of initiation • Time since last use • Typical frequency of drinking • Typical and largest number of drinks consumed during period when drank the most • Largest number of drinks consumed in a single day before age 18, and frequency of consuming 5+ drinks in a single day • Frequency of drinking 5+, 8+, and 12+ drinks in a single day for periods when drank the most **Past-year use** • Any use in past 12 months • Use frequency in past 12 months • Largest number of drinks consumed in a single day • Number of drinks consumed on drinking days • Frequency of drinking 4+ and 5+ drinks in <2 hours • Frequency of drinking 4+, 5+ 8+, and 12+ drinks in a single day • Frequency of drinking beer, wine, and liquor and typical drinking location (own home, friends'/relatives' homes, or public places)

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Tobacco-focused surveys					
Population Assessment of Tobacco and Health (PATH)	**Overview**: PATH is a national longitudinal study of tobacco use and how it affects the health of people in the United States. The survey includes questions about use and perceptions of cigarettes, ENDS, hookah, cigars, and smokeless tobacco. **Sponsors**: NIDA, FDA	2013–present; 8 waves of data collection completed as of 2026	**Sample**: Nationally representative sample of U.S. civilian, noninstitutionalized population age 12 or older **Design**: Longitudinal cohort study with 2 cohorts **Administration**: Home-based, in-person interviews, telephone, and biospecimen collection	**Lifetime use** • Ever use • Ever fairly regular use and amount used • Age of initiation • Time since last use **Past-year use** • Any use in past 12 months • Every or some day use 12 months ago **Past-30-day use** • Any use in past 30 days • Number of days used • Amount used per day **Current use** • Every day or some day use • Amount used per day **Cessation behavior** • Complete quitting • Quit attempts in past 12 months • Use of other tobacco products to quit smoking cigarettes	**Lifetime use** • Ever use • Age of initiation **Past year use** • Any use in past 12 months **Past-30-day use** • Any use in past 30 days • Number of drinking days • Number of drinks consumed on drinking days **Family alcohol use** • Alcoholism/problem drinking among blood relatives

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
National Youth Tobacco Survey (NYTS)	**Overview**: NYTS provides nationally representative data about middle and high school youths' tobacco-related beliefs, attitudes, behaviors, and exposure to pro- and anti-tobacco influences. The survey includes questions about cigarettes, ENDS, hookah, cigars, smokeless tobacco, pipes, HTPs (added in 2020), and nicotine pouches (added in 2021). **Sponsor**: CDC, FDA beginning in 2011.	1999–2009: Biennial 2011–present: Annual, ongoing	**Sample:** Nationally representative sample of middle and high school students (6th–12th grade) **Design:** Cross-sectional **Administration**: School-based through 2020; web-based and completed at school, home, or elsewhere starting in 2021	**Lifetime use** • Ever use • Age of initiation • Time since last use **Past-30-day use** • Any use in past 30 days • Number of days used • Amount used per day **Cessation behavior** • Quit attempts in past 12 months	None

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Tobacco Use Supplement to the Current Population Survey (TUS-CPS)	**Overview**: TUS-CPS is a survey of tobacco use that is administered as part of the U.S. Census Bureau's Current Population Survey every 3–4 years. The survey includes questions about cigarettes, cigars, pipes, smokeless tobacco, ENDS (added in 2011), HTPs (added in 2022), and nicotine pouches (added in 2022). **Sponsors**: NCI, U.S. Census Bureau, FDA (beginning in 2014)	1992–present; conducted every 3 to 4 years	**Sample**: Nationally representative sample of U.S. civilian, noninstitutionalized adults (adolescents ages 15–17 were included in 1992–2006 cycles) **Design**: Cross-sectional **Administration**: Telephone survey or in-person interviews	**Lifetime use** • Age of initiation • Amount smoked when smoked every day • Years used • Time since quitting **Past-year use** • Every day or some day use in past 12 months • Days smoked per month and cigarettes per day in past 12 months **Past-30-day use** • Number of days used **Current use** • Every day or some day use • Cigarettes per day **Cessation behavior** • Quit attempts in past 12 months	None

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Health behavior surveys					
National Longitudinal Study of Adolescent to Adult Health (Add Health)	**Overview**: Add Health is a longitudinal, nationally representative study of adolescents who were in 7th–12th grade in 1994–1995. It includes data on respondents' social, economic, psychological, and physical well-being, as well as contextual data on families, neighborhoods, communities, and schools. **Sponsors**: NICHD, with co-funding from other agencies and foundations	5 waves, data collected approximately every 6–7 years: Wave I (1994–1995), Wave II (1996), Wave III (2001–2002), Wave IV (2008–2009), Wave V (2016–2018)	**Sample**: Nationally representative sample of adolescents in 7th–12th grade **Design**: Longitudinal **Administration**: School- and home-based interviews and biospecimen collection	**Lifetime use** • Ever use • Ever regular use of cigarettes • Age of initiation **Past-30-day use** • Number of days used (cigarettes, cigar, pipe, smokeless tobacco, ENDS) • Cigarettes per day **Cessation behavior** • Ever tried to quit or cut down use • Ever quit or cut down use for at least 1 month	**Lifetime use** • Ever use • Age of initiation **Past year use** • Number of drinking days • Number of drinks consumed on drinking days • Number of binge drinking days (drinking at least 5 drinks in a row) **Past-30-day use** • Number of drinking days • Number of drinks consumed on drinking days **Cessation behavior** • Ever tried to quit or cut down use • Ever quit or cut down use for at least 1 month
Behavioral Risk Factor Surveillance System (BRFSS)	**Overview**: BRFSS collects state data annually about U.S. residents regarding their health-related risk behaviors, chronic health conditions, and use of preventive services. **Sponsors**: CDC, state health departments	1984–present: Annual; ongoing	**Sample**: Representative sample of U.S. adults **Design**: Cross-sectional **Administration**: Telephone survey	**Lifetime use** • Ever use (cigarettes, ENDS) **Current use** • Every day or some day use (cigarettes, smokeless tobacco, ENDS)	**Past-30-day use** • Number of drinking days per week/month • Number of drinks consumed on drinking days • Number of binge drinking days (drinking at least 4/5 drinks on an occasion for women/men) • Largest number of drinks consumed on an occasion

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Health Information National Trends Survey (HINTS)	**Overview**: HINTS collects nationally representative data about the American public's use of cancer-related information **Sponsor**: NCI	2003–2009: Biennial (2009 was Puerto Rico only) 2011–2015; 2017–2020: Annual 2022–present: Biennial	**Sample**: Nationally representative sample of U.S. civilian, noninstitutionalized adults **Design**: Cross-sectional **Administration**: 2003, 2005: Telephone only; 2007: Telephone and mailed questionnaires; 2009–present: Mailed questionnaires only	**Lifetime use** • Ever use of cigarettes • Ever use of cigars, smokeless tobacco, pipe, hookah (2015, 2017 surveys) • Ever use of ENDS (2017–present surveys) • Ever use of heated tobacco products (2020 survey) • Time since quit smoking cigarettes (2003, 2005, 2007, 2013, 2015, 2017 surveys) • Ever smoked cigarettes every day for 6 months (2005, 2007 surveys) **Past-year use** • Every day or some day use of cigarettes 12 months ago (2005 survey) **Past-30-day use** • Number of days smoked cigarettes (2005, 2007 surveys) • Number of cigarettes per day (current smokers–2003, 2005, 2007, 2013 surveys; former smokers–2003, 2005, 2007 surveys) • Number of days used e-cigarettes (2019 survey)	**Past-30-day use** • Number of drinking days per week (2011, 2018–2020, 2022 surveys) • Number of drinks per drinking day (2011, 2020, 2022 surveys) • Average number of drinks per week (2018–2020, 2022 surveys) • Number of binge drinking days (drinking at least 4/5 drinks on an occasion for women/men) (2020, 2022 surveys)

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
				Current use • Every day or some day use of cigarettes • Every day or some day use of cigars, smokeless tobacco (2015, 2017 surveys) • Every day or some day use of ENDS (2017–present surveys) • Current use of heated tobacco products (2020 survey) • Cigarettes per day (2003, 2005, 2007, 2013 surveys) **Cessation behavior** • Quit smoking for ≥1 day in past 12 months (2005, 2012–2014, 2017, 2019 surveys)	
Monitoring the Future (MTF)	**Overview**: MTF is an annual, ongoing study of beliefs, attitudes, and behavior of American secondary school students, college students, and young adults. The survey includes questions about cigarettes, ENDS, and smokeless tobacco. **Sponsors**: NIDA, University of Michigan	1975–present; Annual, ongoing	**Sample:** Nationally representative sample of 8th, 10th, and 12th grade students **Design**: Cross-sectional and longitudinal **Administration:** Group-administered school-based survey Follow-up surveys are mailed to a randomly selected sample from each senior class, with biennial follow-up until age 30, and every 5 years thereafter.	**Lifetime use** • Ever use **Past-year use** • Any use in past year **Past-30-day use** • Any use in past 30 days • Daily use of cigarettes or smokeless tobacco • Daily use of ENDS (added in 2017) • Smoking 1/2 pack of cigarettes or more per day	**Lifetime use** • Ever use **Past-year use** • Any use in past year **Past-30-day use** • Any use in past 30 days • Daily use • Drinking ≥5, 10, and 15 drinks in a row in the past 2 weeks

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
National Health and Nutrition Examination Survey (NHANES)	**Overview**: NHANES is a program of studies to assess the health and nutritional status of adults and children in the United States. The survey combines interviews and physical examinations. The survey includes questions about cigarettes, pipes, cigars, smokeless tobacco, hookah, and ENDS. **Sponsor**: CDC	1999–present; biennial	**Sample:** Nationally representative sample of the U.S. civilian, noninstitutionalized population (newborns and older) **Design**: Cross-sectional **Administration:** Home-based interviews and physical examination by physicians in mobile examination centers	**Lifetime use** • Ever use (cigarettes, cigars, ENDS, smokeless tobacco) • Age of initiation (cigarettes) • Time since quitting (cigarettes) • Cigarettes per day before quitting **Past-30-day use** • Number of days used (cigarettes) • Cigarettes per day **Current use** • Every day or some day use • Use in past 5 days (cigarettes, cigars, ENDS, smokeless tobacco) • Number of days used in past 5 days • Number of times used in past 5 days	**Lifetime use** • Ever use • Ever drinking ≥5 drinks every day **Past year use** • Use frequency in past 12 months (days per week, month, or year) • Average number of drinks per day on drinking days • Frequency of binge drinking (drinking at least 5 drinks in a day) • Frequency of binge drinking (≥5 drinks) within a 2-hour period • Frequency of drinking 8 or more drinks in a single day • Frequency of drinking 12 or more drinks in a single day **Past-30-day use** • Frequency of binge drinking days (drinking at least 5 drinks on an occasion)

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
National Health Interview Survey (NHIS)	**Overview**: NHIS is conducted annually to monitor the health of the U.S. population. The survey collects and analyzes data on a broad range of health topics, including tobacco use, by various demographic and socioeconomic characteristics. Tobacco use questions include questions about cigarettes, cigars, pipes, smokeless tobacco, hookah, and ENDS. **Sponsor**: CDC	1957–present; annual, ongoing Tobacco use questions have been asked annually since 1997. Questions about smoking history and cessation are included on a rotating basis. Alcohol questions were asked annually from 1997 to 2018. Alcohol questions will be asked on a rotating basis from 2019 onward.	**Sample**: Representative sample of the U.S. civilian, noninstitutionalized population. In the 2019 NHIS and later, a sample adult and sample child are selected from each household; information about the child is collected from a parent or knowledgeable adult. **Design**: Cross-sectional **Administration**: Home-based interviews	**Lifetime use** • Ever use • Age of initiation of cigarette smoking (rotating question) • Time since quitting (rotating question) **Past-30-day use** • Number of days smoked (cigarettes, cigars, ENDS) • Number of cigarettes per day **Current use** • Every day or some day use (cigarettes, cigars, pipes, smokeless tobacco, ENDS) **Cessation behavior** • Tried to quit in past year (rotating question) • Use of ENDS to quit smoking (rotating question)	*Rotating questions:* **Lifetime use** • Ever use • Drinking ≥12 drinks in any year **Past year use** • Number of drinking days (per week, month, or year) • Number of drinks consumed on drinking days • Any past year binge drinking (drinking ≥4/5 drinks in a day for women/men) **Past-30-day use** • Any past-30-day use • Number of binge drinking days (drinking ≥4/5 drinks on an occasion for women/men)

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
National Longitudinal Surveys (NLS)	**Overview**: The NLS are a set of surveys designed to gather information about labor market activities and other life events. The surveys include questions about lifetime and current cigarette smoking. **Sponsor**: Bureau of Labor Statistics	1967–present; 5 total cohorts; data collected annually or biennially depending on survey year Surveys ongoing for 2 cohorts: Studies of the 1980–1984 birth cohort [NLSY97], 1957–1964 birth cohort [NLSY79], and children of the 1957–1964 cohort [NLSY79 Child and Young Adult] are still ongoing.	**Sample**: Nationally representative sample of youth or mature adults representing specific birth cohorts. **Design**: Longitudinal **Administration**: In-person and/or telephone-based interviews	**Lifetime use** • Ever use of cigarettes • Age of initiation (cigarettes) • Use of cigarettes since date of last interview • Months or years since last smoked daily (NLSY79 only) **Past-30-day use** • Number of days smoked cigarettes • Number of cigarettes per day	**Lifetime use** • Ever use • Age of initiation • Alcohol use since date of last interview **Past-30-day use** • Any use in past month • Number of drinking days • Number of drinks consumed on drinking days • Number of days consumed ≥5 or 6 drinks on an occasion
National Survey on Drug Use and Health (NSDUH)	**Overview**: NSDUH is an annual survey that provides national and state-level data on the use of tobacco, alcohol, illicit drugs (including nonmedical use of prescription drugs) and mental health in the United States. The survey includes questions about cigarettes, ENDS, smokeless tobacco, cigars, and pipes. **Sponsor**: SAMHSA	1971–present; Annual, ongoing	**Sample**: Nationally representative sample of U.S. civilian, noninstitutionalized population age 12 or older **Design**: Cross-sectional **Administration:** Before 2020: home-based interviews; 2020–present: Home-based and web-based interviews	**Lifetime use** • Ever use • Ever daily use of cigarettes • Age of initiation (tobacco product use; daily use of cigarettes) • Time since last use **Past-30-day use** • Number of days used • Number of cigarettes per day	**Lifetime use** • Ever use • Age of initiation • Ever binge drinking (drinking at least 4/5 drinks on an occasion for women/men) **Past year use** • Number of drinking days **Past-30-day use** • Number of drinking days • Number of drinks consumed on drinking days • Number of binge drinking days (drinking at least 4/5 drinks on an occasion for women/men)

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Pregnancy Risk Assessment Monitoring System (PRAMS)	**Overview**: PRAMS collects state-specific data on maternal attitudes, behaviors, and experiences before, during, and after pregnancy. The system collects data from questionnaires as well as birth certificates. **Sponsors**: CDC, state health departments	1988–present; ongoing	**Sample**: Population-based sample of people who delivered live infants, representing more than 80% of U.S. live births. Data are weighted by jurisdiction and are not nationally representative. **Design**: Cross-sectional **Administration**: Self-administered, mailed questionnaire; interviewer-administered telephone survey; online through a web survey module (starting 2023)	**Lifetime use** • Cigarette smoking, ENDS use in past 2 years **Past-year use** • Number of cigarettes per day and ENDS use frequency (every day/some day) in 3 months before pregnancy • Number of cigarettes per day and ENDS use frequency (every day/some day) in last 3 months of pregnancy **Current use** • Number of cigarettes now smoked per day **Cessation behavior** • Use of ENDS to quit or reduce smoking	**Past year use** • Any alcohol use during first, second, and third trimester of pregnancy • Any consumption of ≥4 drinks in a 2-hour period during first, second, and third trimester of pregnancy

Appendix 2A (continued)

Dataset	Description	Frequency	Population and methodology	Selected tobacco use measures	Selected alcohol use measures
Youth Risk Behavior Survey (YRBS)	**Overview**: YRBS is conducted biennially to monitor priority health-risk behaviors and the prevalence of obesity and asthma among adolescents and young adults. The survey includes questions about cigarettes, cigars, smokeless tobacco, and ENDS. **Sponsor**: CDC	1991–present; Biennially	**Sample**: Nationally representative sample of high school students (9th–12th grade) **Design:** Cross-sectional **Administration**: Self-administered school-based survey	**Lifetime use** • Ever use • Age of initiation **Past-30-day use** • Number of days used • Number of cigarettes per day	**Lifetime use** • Ever use • Total drinking days (only 2017 and earlier surveys) • Age of initiation **Past-30-day use** • Number of drinking days • Number of binge drinking days • Number of drinks on the occasion the respondent drank the most

Note. AUD = alcohol use disorder; CDC = Centers for Disease Control and Prevention; ENDS = electronic nicotine delivery systems; FDA = Food and Drug Administration; HTPs = heated tobacco products; NCI = National Cancer Institute; NIAAA = National Institute on Alcohol Abuse and Alcoholism; NICHD = National Institute of Child Health and Human Development; NIDA = National Institute on Drug Abuse; SAMHSA = Substance Abuse and Mental Health Services Administration.
Source: Alcohol Research Group n.d., 2014,[62,172] Bureau of Labor Statistics n.d., 2023,[173-177] CDC 2013, 2017, 2018, 2022, 2023, 2024,[178-200] Hyland et al. 2017,[201] CSR Incorporated 2022,[31] Inter-University Consortium for Political and Social Research 2021, 2023, 2024,[202-207] NCI n.d., 2023, 2024,[208-212] NIAAA n.d., 2022, 2024,[213,214] NIDA n.d.,[215] Office of Disease Prevention and Health Promotion n.d.,[216] Office on Smoking and Health 2020, 2021, 2022,[217-220] Piesse et al. 2021,[221] SAMHSA n.d.,[63,222,223] University of Michigan 2024,[61,224] University of North Carolina, Carolina Population Center n.d.[225-227]

Appendix 2B Literature Search Strategy

The following search terms were used in PubMed, Google Scholar, Ovid, and Scopus between June 2023 and September 2024 to identify literature regarding co-use of tobacco and alcohol in the general population.

- Alcohol, drinking, beer, wine, spirits, drinker, drinking patterns
- Tobacco, cigarette, smoking, smoker, co-use, simultaneous use, co-use, co-user, tobacco product, e-cigarette, vaping, polysubstance use

Additional studies published after 2024 were incorporated into the chapter based on their relevance.

Appendix 2C Methods—Monitoring the Future Study, National Alcohol Surveys, and National Survey on Drug Use and Health Analyses

The Monitoring the Future Study

Monitoring the Future (MTF) is a nationally representative, classroom-based survey of U.S. students in 8th, 10th, and 12th grade. The survey uses multistage random sampling to select a sample of students in approximately 100 public and private high schools and middle schools. The survey is conducted annually and began with collecting data from high school seniors in 1975 and expanded to students in 8th and 10th grade in 1991. Additional information regarding the MTF methodology is available from the University of Michigan.[228]

Monitoring the Future Tobacco Use Measures

Tobacco use measures in the MTF focused on past-30-day use of cigarettes, large cigars, smokeless tobacco, and nicotine vaping.

Cigarette Smoking Status. Past-30-day cigarette smoking status was based on responses to the question "How frequently have you smoked cigarettes during the past 30 days?" with answer categories "not at all," "less than one cigarette per day," "one to five cigarettes per day," "about one-half pack per day," "one pack per day," "one and one-half packs per day," and "two packs or more per day."

Large Cigar Smoking Status. Past-30-day large cigar smoking status was based on a question parallel to the cigarette smoking question.

Smokeless Tobacco Use Status. Past-30-day smokeless tobacco use status was based on a question parallel to the cigarette question.

Nicotine Vaping Status. Past-30-day nicotine vaping status was based on a question parallel to the cigarette question.

Monitoring the Future Alcohol Use Measures

Alcohol use measures in the MTF focused on past-30-day alcohol use and binge drinking in the past 2 weeks.

Alcohol Use Status. Past-30-day alcohol use was based on responses to the question, "On how many occasions (if any) have you used alcohol during the last 30 days?" with answer categories of 0, 1–2, 3–5, 6–9, 10–19, 20–39, and 40 or more occasions.

Binge Drinking. Binge drinking was defined as drinking 5 or more drinks in a row over the past 2 weeks.

The National Alcohol Surveys

The National Alcohol Survey (NAS) is a population-based survey, representative of noninstitutionalized adults ages 18 or older within all 50 states and the District of Columbia. Conducted at approximately 5-year intervals, NAS has the longest time frame of any nationally

representative survey, with more detailed alcohol use pattern and heavy alcohol use measures than other surveys. NAS is unique in including life course measures of both tobacco and alcohol use as well as measures of current alcohol use in specific contexts, such as bars and the frequencies of high-intensity drinking (HID) occasions. The 2014–2015 and 2019–2020 NAS were conducted by the Alcohol Research Group (ARG) with funding from the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The 2014–2015 NAS was conducted through telephone interviews of participants recruited through dual-frame landline and mobile phone random digit dialing (RDD). The 2019–2020 NAS consisted of two probability samples: a mobile phone RDD sample with telephone interviews and an address-based sample with online surveys. These probability samples were supplemented with a nonprobability sample drawn from an existing web panel. Additionally, targeted oversampling of Black and Hispanic/Latino participants allowed for subgroup comparisons. Additional information regarding the NAS methodology is available from the ARG.[62]

National Alcohol Surveys Tobacco Use Measures

The NAS analyses included a measure of tobacco use status (based on lifetime and past 12-month use) and a measure of lifetime cigarette pack years.

Current Tobacco Use. Current tobacco use was categorized by tobacco use status and frequency. Participants were classified as never using tobacco if they responded "no" to the question, "Have you ever smoked cigarettes or used any other kind of tobacco, including e-cigarettes, on a regular basis in your life?" Frequency of tobacco use was asked only of those who reported ever tobacco use in lifetime. Participants were asked how often they smoked cigarettes or used any other kinds of tobacco (not including e-cigarettes) in the past 12 months with the options: daily or nearly daily, 1 to 4 days per week, once every 2 to 3 weeks, once every month or so, less often than that, or never within the past 12 months. Former tobacco use was a "yes" response to the lifetime use question and no reported tobacco use within the past 12 months. Current tobacco use (i.e., past-year tobacco use) was defined as reporting tobacco use within the past 12 months. Due to small cell sizes, categories were collapsed into nondaily current tobacco use (those who reported using tobacco 1 to 4 days per week, once every 2 to 3 weeks, once every month or so, or less often than that), and daily current tobacco use (those who reported using tobacco daily or nearly daily).

Frequency of e-cigarette use was assessed only in the 2019–2020 NAS and was asked separately from use of cigarettes or other tobacco products. Use of e-cigarettes in 2019–2020 NAS was categorized as daily or nearly daily, monthly, or semimonthly (monthly or less, or weekly or nearly weekly), and no use in the past year.

Lifetime Cigarette Smoking. Pack years were calculated to measure lifetime cigarette exposure (among people who formerly smoked or smoked in the past year). This measure was created using participants' current age, age of smoking onset, and average number of cigarettes per day (standardized to packs by dividing by 20). To calculate pack years, the number of packs smoked per day was multiplied by the number of years smoked. Based on previously established cutoff points,[229] participants were categorized into 1 of 4 groups: 0 pack years, 0.1–20 pack years, 20.1–40 pack years, and ≥40.1 pack years. As all NAS analyses were age stratified, alternative

cutoff points were examined for those ages 18–29 as these previously established thresholds likely translate to different risk levels among younger individuals. Due to limited data availability, this measure was only calculated for the 2014–2015 NAS.

National Alcohol Surveys Alcohol Use Measures

The NAS analyses included several measures of alcohol use in the past 12 months, and 1 measure of lifetime alcohol use.

Past-Year Alcohol Use. The NAS analyses included three primary measures of past-year alcohol use:

- **Drinking within guidelines**: 3 categories—no alcohol use in past year, alcohol use within guidelines (consuming ≤7 drinks per week for women and ≤14 drinks per week for men), and alcohol use exceeding guidelines (consuming >7 drinks per week for women and >14 drinks per week for men) per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use[66]
- **5+ drinking**: Consuming 5 or more drinks on any given day (binary yes/no variable)
- **HID**: Consuming ≥8 drinks on any given day for women and ≥12 drinks on any given day for men (binary yes/no variable)

In addition, past-year volume of consumption and frequency of drinking by context were also analyzed.

- **Volume**. Volume of consumption was represented as the number of standard drinks consumed within the past year. This was calculated using the average number and type of drinks (beer, wine, or spirits) consumed each drinking day and the frequency of drinking days in the past year.
- **Frequency of drinking by context**. Participants reported frequency of drinking (never, monthly or less, weekly or more) within the past year for the following drinking contexts: bars, taverns, or cocktail lounges; party in someone's home; and quiet evening at home.

Lifetime Alcohol Use. Lifetime number of 5+ drinking days was estimated using decades questions about the frequency of 5+ drinking days (for example, "How often did you have five or more drinks on one occasion during your teens/20s/30s/40s/50s?" Response options were every day or nearly every day, at least once a week, at least once a month (but less than 1/week), at least once a year (but less than 1/month), less than once a year, and never).

The National Survey on Drug Use and Health

The National Survey on Drug Use and Health (NSDUH) is a nationally representative survey of the U.S. noninstitutionalized civilian population, not including people with no permanent residence or living in college dormitories, group homes, or on military bases. The survey uses multistage probability sampling methods for each of the 50 states and the District of Columbia to select a representative sample of participants ages 12 or older. Adolescents ages 12–17 and adults 18–25 are oversampled. The NSDUH uses a combination of computer-assisted personal

interviewing and audio-computer–assisted self-interviewing. The large size of the NSDUH allows for analyses of population subgroups. Chapter 3 presents analyses of subgroups by race and/or ethnicity, educational attainment, income, sexual minority status, presence of chronic health conditions, and military veteran status. A detailed description of NSDUH survey methodology is available from the Substance Abuse and Mental Health Services Administration.[230]

The 2015–2019 NSDUH included only limited information on lifetime use of tobacco and alcohol but have information on the frequency and quantity of alcohol consumption and cigarette smoking in the past 30 days as well as the frequency of binge drinking days in the past 30 days. The 2015–2019 survey years were the most recent comparable 5-year series available at the time these analyses were conducted due to methodological changes during the COVID-19 period that prevent comparisons with earlier years. Pooled data were necessary for less represented subgroups (e.g., people from sexual minority groups, people with health conditions).

National Survey on Drug Use and Health Tobacco Use Measures

Tobacco use measures in the NSDUH analyses focused on current (past-30-day) use and former and never lifetime use of cigarettes, cigars, and smokeless tobacco.

Cigarette Smoking Status. Cigarette smoking status measures were based on questions on lifetime, past-year, and past-30-day use. Current smoking was defined as having smoked on at least 1 day in the past 30 days. Lifetime use, use frequency, and typical number of cigarettes per day were used to categorize cigarette smoking as never smoking (no lifetime use), former smoking (did not smoke in the past 30 days), or current smoking. Among those who reported current smoking, 3 groups were created: nondaily smoking of <300 cigarettes per month (<10 cigarettes per day on average), daily smoking of <300 cigarettes per month, and ≥300 cigarettes per month (≥10 cigarettes per day on average, whether daily or nondaily smoking). The average number of cigarettes per month was calculated as the number of days participant reported smoking in the past 30 days (ranging from 1 to 30, in response to the question "During the past 30 days, on how many days did you smoke part or all of a cigarette?") multiplied by the middle value of the average number of cigarettes smoked per day ("On the days you smoked cigarettes during the past 30 days, how many cigarettes did you smoke per day, on average"), as indicated below:

1. Less than one cigarette per day; middle value: 0.5 cigarettes
2. 1 cigarette per day; middle value: 1 cigarette
3. 2 to 5 cigarettes per day; middle value: 3 cigarettes
4. 6 to 15 cigarettes per day (about ½ pack); middle value: 10 cigarettes
5. 16 to 25 cigarettes per day (about 1 pack); middle value: 20 cigarettes
6. 26 to 35 cigarettes per day (about 1½ pack); middle value: 30 cigarettes
7. More than 35 cigarettes per day (about 2 packs or more); middle value: 40 cigarettes

Cigar Smoking Status. Current cigar smoking was defined as use of cigars (any kind, including big cigars, cigarillos, and little cigars) in the past 30 days. Lifetime use and use frequency were used to categorize cigar smoking status as never use (no lifetime use), former use (did not smoke cigars in the past 30 days), and current use (smoked cigars 1 or more days in the past 30 days).

Smokeless Tobacco Use Status. Current smokeless tobacco use was defined as use of smokeless tobacco (snuff, dip, chewing tobacco, or "snus") in the past 30 days. Lifetime use and use frequency were used to categorize current smokeless tobacco use as never use (no lifetime use), former use (did not use smokeless tobacco in the past 30 days), nondaily use (used smokeless tobacco 1 to 29 days in the past 30 days), and daily use (used smokeless tobacco all 30 days in the past 30 days).

National Survey on Drug Use and Health Alcohol Use Measures

Alcohol use measures in the NSDUH analyses focused on past-30-day use and former and never lifetime use.

Alcohol Use Status. A measure of current drinking was based on self-reported alcoholic beverage consumption in the past 30 days. Drinking volume was calculated using drinking frequency (number of days having consumed at least one drink of any alcoholic beverage), usual quantity (average number of drinks on days when drank), and binge drinking days (frequency of more than four or five drinks for women and men, respectively). As discussed in the "Measurement of Tobacco and Alcohol Co-Use" section, given participants' tendencies to underestimate their own consumption, binge drinking days were incorporated into the volume calculation. For participants whose usual quantity was fewer than five drinks (for men) or four drinks (for women), volume was calculated using *(frequency days – binge days) * usual quantity + binge days * 6 drinks (women) or 7 drinks (men)*. For participants whose usual quantity was equal to or more than five or four drinks, volume was calculated by multiplying frequency days and usual quantity. This volume calculation was used to derive average weekly and daily consumption, and indicate whether participants exceeded U.S. risky weekly drinking guidelines, defined as more than 7 drinks/week for women or more than 14 drinks/week for men.[65,66] To include participants who did not have information for usual binge drinking days, volume was measured using only frequency and usual quantity (n = 660). Current alcohol use was based on participants having reported use on at least 1 day in the past 30 days. Based on the calculations of daily and weekly consumption, participants were grouped into the following categories: "alcohol use within guidelines" (use within past 30 days, but without binge drinking and without exceeding risky weekly drinking guidelines); "binge, no risky weekly use" (binge drinking without exceeding risky weekly drinking guidelines); and "risky weekly use" (exceeding weekly drinking guidelines). Participants who responded "no" to the question "Have you ever, even once, had a drink of any type of alcoholic beverage?" were categorized to "never alcohol use." Participants who responded "yes" to this lifetime use question but did not report use in the past 30 days were classified as "former alcohol use."

Statistical Analysis

Weighted prevalence estimates were calculated for overall tobacco and alcohol use patterns in the MTF, NAS, and NSDUH analyses. Statistical analyses tested differences in alcohol use between tobacco use (NAS) and cigarette smoking (NSDUH) groups. For categorical variables, chi-squared tests were conducted to assess differences in alcohol use patterns among those with different tobacco-use patterns (NAS) or cigarette smoking patterns (NSDUH). For continuous variables in the NAS (i.e., lifetime number of 5+ drinking days, overall and beverage-specific volume consumption), Poisson regression was used to test for statistically significant differences, adjusting for survey cycle. To examine differences by subgroups, chi-squared tests and Poisson regressions were conducted separately for each subgroup. All analyses were conducted in Stata, version 17, and significance testing was conducted at the $a = .05$ level.

Appendix 2D Monitoring the Future, National Alcohol Surveys, and National Survey on Drug Use and Health Tables

Table 2D.1 Past-30-Day Tobacco and Alcohol Use Among 8th Grade Students in the Monitoring the Future Study, 2000–2023

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2000 (17,311)	22.4 (20.7, 24.1)	11.7 (10.5, 12.9)	N/A	N/A	N/A	14.6 (13.2, 16.2)	9.6 (8.7, 10.7)	6.1 (5.4, 6.9)	N/A	N/A	N/A	4.2 (3.4, 5.2)	3.0 (2.4, 3.8)	2.3 (1.8, 3.0)
2001 (16,756)	21.5 (20.1, 23.0)	11.0 (10.0, 12.1)	N/A	N/A	N/A	12.2 (11.0, 13.5)	7.9 (7.1, 8.8)	5.1 (4.5, 5.7)	N/A	N/A	N/A	4.0 (3.1, 5.2)	2.3 (1.8, 3.0)	1.8 (1.4, 2.3)
2002 (15,489)	19.6 (18.2, 21.1)	10.3 (9.3, 11.4)	N/A	N/A	N/A	10.7 (9.5, 12.0)	7.0 (6.2, 8.0)	5.0 (4.3, 5.7)	N/A	N/A	N/A	3.3 (2.5, 4.3)	2.0 (1.4, 2.5)	1.4 (0.9, 2.1)
2003 (17,023)	19.7 (18.4, 21.2)	9.8 (8.9, 10.9)	N/A	N/A	N/A	10.2 (9.1, 11.5)	7.0 (6.1, 7.9)	4.8 (4.2, 5.6)	N/A	N/A	N/A	4.1 (3.5, 4.9)	2.6 (2.1, 3.2)	2.0 (1.5, 2.5)
2004 (17,413)	18.6 (17.5, 19.7)	9.4 (8.6, 10.3)	N/A	N/A	N/A	9.2 (8.2, 10.3)	5.9 (5.2, 6.7)	4.1 (3.5, 4.8)	N/A	N/A	N/A	4.1 (3.2, 5.3)	2.4 (1.8, 3.2)	1.6 (1.2, 2.1)
2005 (17,258)	17.1 (16.0, 18.3)	8.4 (7.7, 9.2)	N/A	N/A	N/A	9.3 (8.3, 10.4)	5.9 (5.2, 6.7)	3.8 (3.3, 4.3)	N/A	N/A	N/A	3.3 (2.5, 4.3)	1.9 (1.4, 2.5)	1.4 (1.0, 1.8)
2006 (17,026)	17.2 (15.9, 18.6)	8.7 (7.9, 9.6)	N/A	N/A	N/A	8.7 (7.7, 9.7)	5.6 (5.0, 6.4)	3.7 (3.3, 4.3)	N/A	N/A	N/A	3.7 (3.0, 4.4)	2.0 (1.6, 2.5)	1.5 (1.2, 1.9)
2007 (16,495)	15.9 (14.7, 17.1)	8.3 (7.5, 9.2)	N/A	N/A	N/A	7.1 (6.3, 7.9)	4.6 (4.0, 5.2)	3.3 (2.9, 3.8)	N/A	N/A	N/A	3.2 (2.6, 3.8)	2.1 (1.7, 2.6)	1.4 (1.1, 1.8)
2008 (16,253)	15.9 (14.6, 17.4)	8.1 (7.2, 9.0)	N/A	N/A	N/A	6.8 (6.1, 7.6)	4.6 (4.1, 5.1)	3.2 (2.9, 3.7)	N/A	N/A	N/A	3.5 (2.8, 4.3)	2.0 (1.5, 2.6)	1.4 (1.0, 1.8)
2009 (15,509)	14.9 (13.4, 16.6)	7.8 (6.8, 8.9)	N/A	N/A	N/A	6.5 (5.7, 7.3)	4.2 (3.6, 4.9)	2.7 (2.3, 3.3)	N/A	N/A	N/A	3.7 (3.0, 4.7)	2.2 (1.6, 3.1)	1.4 (1.0, 1.9)
2010 (15,769)	13.8 (12.5, 15.1)	7.2 (6.4, 8.1)	N/A	N/A	N/A	7.1 (6.2, 8.1)	4.1 (3.5, 4.7)	2.7 (2.2, 3.2)	N/A	N/A	N/A	4.1 (3.2, 5.1)	2.0 (1.5, 2.7)	1.5 (1.1, 2.1)
2011 (16,496)	12.7 (11.6, 13.9)	6.4 (5.7, 7.3)	N/A	N/A	N/A	6.1 (5.3, 7.0)	3.7 (3.2, 4.3)	2.4 (2.1, 2.9)	N/A	N/A	N/A	3.5 (2.8, 4.2)	1.9 (1.5, 2.4)	1.2 (0.9, 1.5)
2012 (15,678)	11.0 (10.0, 12.1)	5.1 (4.4, 5.9)	N/A	N/A	N/A	4.9 (4.2, 5.8)	2.9 (2.4, 3.5)	1.9 (1.6, 2.2)	N/A	N/A	N/A	2.8 (2.2, 3.4)	1.4 (1.0, 1.8)	0.9 (0.7, 1.2)
2013 (15,233)	10.2 (9.3, 11.3)	5.1 (4.5, 5.8)	N/A	N/A	N/A	4.5 (3.8, 5.3)	2.6 (2.2, 3.1)	1.8 (1.5, 2.2)	N/A	N/A	N/A	2.8 (2.2, 3.6)	1.6 (1.1, 2.2)	1.1 (0.8, 1.6)
2014 (15,195)	9.0 (8.2, 9.9)	4.1 (3.7, 4.7)	N/A	N/A	N/A	4.0 (3.4, 4.7)	2.2 (1.8, 2.7)	1.3 (1.0, 1.7)	1.9 (1.4, 2.5)	0.9 (0.6, 1.3)	*	3.0 (2.4, 3.6)	1.6 (1.2, 2.1)	0.9 (0.6, 1.4)
2015 (15,015)	9.7 (8.7, 10.8)	4.6 (4.1, 5.3)	N/A	N/A	N/A	3.6 (3.1, 4.2)	2.1 (1.7, 2.5)	1.3 (1.1, 1.6)	2.4 (1.9, 3.1)	1.2 (0.8, 1.6)	0.8 (0.6, 1.2)	3.2 (2.6, 3.9)	1.6 (1.2, 2.1)	0.9 (0.7, 1.3)
2016 (17,643)	7.3 (6.6, 8.0)	3.4 (2.9, 4.0)	N/A	N/A	N/A	2.6 (2.2, 3.2)	1.3 (1.0, 1.6)	0.8 (0.6, 1.1)	1.5 (1.1, 2.1)	0.6 (0.4, 0.8)	*	2.5 (2.0, 3.1)	1.1 (0.8, 1.5)	0.7 (0.5, 1.0)
2017 (16,010)	8.0 (7.2, 8.9)	3.7 (3.2, 4.2)	3.5 (2.9, 4.2)	1.5 (1.1, 2.0)	1.1 (0.8, 1.6)	1.9 (1.6, 2.2)	1.1 (0.9, 1.3)	0.7 (0.6, 0.9)	1.5 (1.1, 1.9)	0.6 (0.4, 0.9)	*	1.7 (1.4, 2.2)	0.9 (0.6, 1.2)	0.5 (0.4, 0.8)

Table 2D.1 (continued)

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2018 (14,836)	8.2 (7.3, 9.1)	3.7 (3.2, 4.2)	6.1 (5.1, 7.4)	2.5 (1.9, 3.3)	1.4 (1.0, 2.0)	2.2 (1.7, 2.7)	1.1 (0.8, 1.4)	0.6 (0.5, 0.9)	1.7 (1.2, 2.3)	0.8 (0.5, 1.3)	*	2.1 (1.6, 2.7)	0.9 (0.7, 1.3)	0.6 (0.4, 0.9)
2019 (14,223)	7.9 (6.9, 9.1)	3.8 (3.2, 4.6)	9.6 (8.3, 11.1)	3.6 (2.9, 4.4)	2.1 (1.6, 2.6)	2.3 (1.7, 3.0)	1.1 (0.8, 1.7)	0.8 (0.5, 1.3)	1.3 (0.9, 2.0)	0.7 (0.5, 1.2)	*	2.5 (1.9, 3.2)	0.9 (0.6, 1.4)	0.7 (0.4, 1.2)
2020 (3,161)	9.9 (7.8, 12.5)	4.5 (3.2, 6.4)	10.5 (7.7, 14.1)	5.1 (3.4, 7.6)	3.0 (1.9, 4.6)	2.2 (1.5, 3.1)	1.5 (1.0, 2.3)	1.0 (0.7, 1.5)	1.5 (0.8, 2.9)	*	*	2.3 (1.6, 3.3)	*	*
2021 (11,446)	7.3 (6.3, 8.5)	2.8 (2.2, 3.4)	7.6 (6.4, 9.1)	3.2 (2.6, 3.9)	1.8 (1.3, 2.3)	1.1 (0.8, 1.6)	0.6 (0.4, 1.0)	0.4 (0.3, 0.7)	1.1 (0.7, 1.7)	*	*	1.6 (1.2, 2.3)	*	*
2022 (9,889)	6.0 (5.0, 7.2)	2.2 (1.8, 2.7)	7.1 (6.0, 8.3)	2.3 (1.8, 3.0)	1.1 (0.8, 1.5)	0.8 (0.6, 1.1)	0.4 (0.3, 0.6)	*	0.5 (0.2, 1.0)	*	*	1.2 (0.8, 1.6)	*	*
2023 (6,240)	5.9 (4.7, 7.4)	2.0 (1.4, 2.8)	7.0 (5.4, 9.0)	2.3 (1.7, 3.2)	1.1 (0.7, 1.7)	1.1 (0.8, 1.7)	*	*	1.0 (0.6, 1.7)	*	*	1.6 (1.0, 2.5)	*	*

Note. CI = confidence interval; N/A = not applicable. Data are visualized in Figure 2.2. *Alcohol use* and *tobacco product use* are defined as use in the past 30 days. *Binge drinking* is defined as drinking ≥5 drinks in a row over the past 2 weeks. The large cigar question was first asked in 2014. The nicotine vaping question was first asked in 2017. Questions about certain products were only asked of some students. For large cigars, *N* is one-third of *N* indicated across years. For smokeless tobacco, *N* is one-half of *N* indicated across years. For nicotine vaping, *N* is one-third of *N* indicated for 2017 and 2018, and two-thirds of *N* indicated in 2019.
*Cells with $n < 30$ are suppressed.
Source: 2000–2023 Monitoring the Future study.

Table 2D.2 Past-30-Day Tobacco and Alcohol Use Among 10th Grade Students in the Monitoring the Future Study, 2000–2023

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2000 (14,576)	41.0 (39.0, 43.0)	24.1 (22.4, 25.9)	N/A	N/A	N/A	23.9 (22.3, 25.6)	18.3 (16.9, 19.7)	12.8 (11.7, 14.0)	N/A	N/A	N/A	6.1 (5.0, 7.3)	4.7 (3.9, 5.6)	3.7 (3.0, 4.5)
2001 (14,286)	39.0 (37.4, 40.5)	22.8 (21.4, 24.3)	N/A	N/A	N/A	21.3 (19.6, 23.1)	16.5 (15.1, 18.0)	11.8 (10.7, 13.0)	N/A	N/A	N/A	6.9 (5.7, 8.2)	5.4 (4.5, 6.4)	4.1 (3.4, 5.0)
2002 (14,683)	35.4 (33.6, 37.3)	20.3 (19.0, 21.7)	N/A	N/A	N/A	17.7 (16.3, 19.1)	13.1 (12.1, 14.3)	9.2 (8.5, 10.1)	N/A	N/A	N/A	6.1 (5.1, 7.3)	4.3 (3.5, 5.2)	3.2 (2.6, 3.9)
2003 (16,244)	35.4 (33.5, 37.3)	20.0 (18.5, 21.6)	N/A	N/A	N/A	16.6 (15.4, 18.0)	12.6 (11.6, 13.8)	8.8 (8.0, 9.7)	N/A	N/A	N/A	5.3 (4.4, 6.4)	3.7 (3.0, 4.6)	3.0 (2.4, 3.8)
2004 (16,839)	35.2 (33.2, 37.3)	19.9 (18.3, 21.5)	N/A	N/A	N/A	16.0 (14.6, 17.5)	12.1 (10.9, 13.3)	8.5 (7.5, 9.5)	N/A	N/A	N/A	4.9 (4.0, 6.0)	3.7 (3.0, 4.5)	3.0 (2.4, 3.8)
2005 (16,711)	33.2 (31.4, 35.1)	19.0 (17.6, 20.5)	N/A	N/A	N/A	14.9 (13.7, 16.2)	11.0 (10.1, 12.0)	7.7 (7.0, 8.5)	N/A	N/A	N/A	5.6 (4.7, 6.7)	4.0 (3.3, 4.9)	3.2 (2.6, 3.9)
2006 (16,620)	33.8 (32.1, 35.6)	19.9 (18.6, 21.3)	N/A	N/A	N/A	14.5 (13.3, 15.8)	11.1 (10.2, 12.1)	8.0 (7.3, 8.8)	N/A	N/A	N/A	5.7 (4.9, 6.7)	4.4 (3.7, 5.2)	3.5 (2.9, 4.1)
2007 (16,398)	33.4 (31.7, 35.2)	19.6 (18.2, 21.2)	N/A	N/A	N/A	14.0 (12.9, 15.2)	10.8 (9.8, 11.8)	7.8 (7.0, 8.6)	N/A	N/A	N/A	6.1 (5.2, 7.2)	4.7 (3.9, 5.6)	3.5 (2.9, 4.2)
2008 (15,518)	28.8 (27.3, 30.3)	16.0 (14.7, 17.3)	N/A	N/A	N/A	12.3 (11.2, 13.4)	9.1 (8.3, 10.0)	6.6 (6.0, 7.4)	N/A	N/A	N/A	5.0 (4.1, 6.0)	3.2 (2.6, 4.0)	2.4 (1.9, 3.1)
2009 (16,320)	30.4 (28.8, 32.0)	17.5 (16.2, 18.7)	N/A	N/A	N/A	13.1 (12.0, 14.4)	9.7 (8.9, 10.7)	7.0 (6.3, 7.8)	N/A	N/A	N/A	6.5 (5.4, 7.7)	4.9 (4.0, 5.9)	3.8 (3.1, 4.6)
2010 (15,586)	28.9 (27.4, 30.5)	16.3 (15.3, 17.5)	N/A	N/A	N/A	13.6 (12.4, 14.8)	9.7 (8.8, 10.6)	6.8 (6.2, 7.5)	N/A	N/A	N/A	7.5 (6.3, 8.9)	5.3 (4.4, 6.4)	4.0 (3.3, 4.8)
2011 (15,382)	27.2 (25.6, 28.8)	14.7 (13.6, 15.9)	N/A	N/A	N/A	11.8 (10.7, 13.0)	8.5 (7.6, 9.4)	6.1 (5.4, 6.8)	N/A	N/A	N/A	6.6 (5.6, 7.7)	4.4 (3.8, 5.3)	3.3 (2.8, 3.9)
2012 (15,428)	27.6 (25.9, 29.2)	15.6 (14.3, 16.9)	N/A	N/A	N/A	10.8 (9.6, 12.0)	8.0 (7.1, 9.0)	5.6 (4.9, 6.3)	N/A	N/A	N/A	6.4 (5.4, 7.5)	4.2 (3.5, 5.1)	2.9 (2.4, 3.6)
2013 (13,262)	25.7 (24.0, 27.4)	13.7 (12.4, 15.1)	N/A	N/A	N/A	9.1 (8.0, 10.4)	6.3 (5.4, 7.2)	4.2 (3.6, 4.9)	N/A	N/A	N/A	6.4 (5.5, 7.5)	4.1 (3.4, 4.9)	2.9 (2.3, 3.5)
2014 (13,341)	23.5 (21.8, 25.3)	12.6 (11.4, 14.0)	N/A	N/A	N/A	7.2 (6.3, 8.1)	5.1 (4.4, 5.9)	3.7 (3.1, 4.3)	3.9 (3.2, 4.8)	3.0 (2.4, 3.8)	2.4 (1.8, 3.1)	5.3 (4.3, 6.4)	3.5 (2.8, 4.4)	2.7 (2.0, 3.4)
2015 (16,147)	21.5 (19.9, 23.2)	10.9 (9.8, 12.1)	N/A	N/A	N/A	6.3 (5.5, 7.2)	4.2 (3.6, 5.0)	2.9 (2.4, 3.4)	3.4 (2.8, 4.2)	2.3 (1.8, 3.0)	1.7 (1.3, 2.3)	4.9 (4.1, 5.8)	3.2 (2.6, 3.8)	2.3 (1.9, 2.9)
2016 (15,230)	19.9 (18.4, 21.6)	9.7 (8.8, 10.8)	N/A	N/A	N/A	4.9 (4.2, 5.7)	3.1 (2.6, 3.7)	2.0 (1.7, 2.4)	2.3 (1.7, 3.0)	1.5 (1.1, 2.1)	1.2 (0.8, 1.7)	3.5 (2.8, 4.5)	2.2 (1.6, 3.0)	1.5 (1.1, 2.1)
2017 (14,171)	19.7 (18.0, 21.5)	9.8 (8.8, 11.0)	8.2 (6.6, 10.2)	5.8 (4.6, 7.3)	3.8 (3.0, 4.9)	5.0 (4.3, 5.9)	3.1 (2.5, 3.8)	2.0 (1.6, 2.6)	2.6 (2.1, 3.3)	1.8 (1.3, 2.5)	1.3 (0.9, 1.9)	3.8 (3.1, 4.6)	2.3 (1.8, 2.9)	1.8 (1.4, 2.4)
2018 (15,144)	18.6 (17.0, 20.4)	8.7 (7.6, 9.9)	16.1 (13.9, 18.6)	9.1 (7.6, 10.9)	5.2 (4.2, 6.4)	4.2 (3.5, 5.2)	2.9 (2.4, 3.6)	2.0 (1.6, 2.6)	2.8 (2.2, 3.6)	1.9 (1.4, 2.5)	1.5 (1.1, 2.0)	3.9 (3.2, 4.8)	2.3 (1.8, 2.9)	1.5 (1.1, 2.0)

Table 2D.2 (continued)

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2019 (14,595)	18.4 (16.5, 20.5)	8.5 (7.3, 9.8)	19.9 (17.8, 22.3)	10.3 (8.8, 12.0)	5.6 (4.8, 6.7)	3.4 (2.7, 4.3)	2.5 (1.9, 3.4)	1.5 (1.1, 2.0)	2.1 (1.5, 2.9)	1.7 (1.1, 2.6)	1.3 (0.8, 2.1)	3.2 (2.6, 4.1)	1.9 (1.4, 2.7)	1.3 (0.9, 2.0)
2020 (4,890)	20.3 (16.7, 24.4)	9.6 (7.7, 11.9)	19.3 (15.9, 23.2)	10.6 (8.4, 13.2)	5.7 (4.4, 7.2)	3.2 (2.5, 4.2)	2.0 (1.4, 2.7)	1.5 (1.1, 2.2)	1.2 (0.7, 2.2)	*	*	3.5 (2.4, 5.1)	*	*
2021 (11,792)	13.1 (11.4, 15.0)	5.9 (5.0, 6.9)	13.1 (11.2, 15.4)	6.5 (5.5, 7.7)	3.7 (3.0, 4.4)	1.8 (1.5, 2.2)	1.1 (0.8, 1.4)	0.8 (0.6, 1.1)	1.3 (0.8, 2.1)	1.0 (0.6, 1.8)	*	1.7 (1.3, 2.2)	0.7 (0.5, 1.1)	*
2022 (11,950)	13.6 (12.0, 15.3)	5.9 (5.1, 6.9)	14.2 (12.1, 16.6)	6.7 (5.7, 7.9)	3.5 (2.9, 4.3)	1.7 (1.2, 2.3)	1.0 (0.7, 1.4)	0.7 (0.5, 1.0)	0.8 (0.5, 1.2)	*	*	2.5 (2.0, 3.1)	1.0 (0.7, 1.4)	0.7 (0.5, 1.1)
2023 (8,494)	13.7 (12.1, 15.4)	5.4 (4.6, 6.5)	11.9 (10.6, 13.4)	6.2 (5.2, 7.3)	3.2 (2.5, 4.0)	2.3 (1.8, 3.1)	1.5 (1.0, 2.1)	1.0 (0.7, 1.4)	0.3 (0.2, 0.5)	*	*	2.3 (1.7, 3.2)	1.1 (0.6, 2.0)	*

Note. CI = confidence interval; N/A = Not applicable. Data are visualized in Figure 2.2. *Alcohol use* and *tobacco product use* are defined as use in the past 30 days. *Binge drinking* is defined as drinking ≥5 drinks in a row over the past 2 weeks. The large cigar question was first asked in 2014. The nicotine vaping question was first asked in 2017. Questions about certain products were only asked of some students. For large cigars, *N* is one-third of *N* indicated across years. For smokeless tobacco, *N* is one-half of *N* indicated across years. For nicotine vaping, *N* is one-third of *N* indicated for 2017 and 2018, and two-thirds of *N* indicated in 2019.
*Cells with $n < 30$ are suppressed.
Source: 2000–2023 Monitoring the Future study.

Table 2D.3 Past-30-Day Tobacco and Alcohol Use Among 12th Grade Students in the Monitoring the Future Study, 2000–2023

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2000 (13,286)	50.0 (47.6, 52.5)	30.0 (27.7, 32.4)	N/A	N/A	N/A	31.4 (29.2, 33.7)	25.5 (23.6, 27.5)	18.4 (16.9, 20.1)	N/A	N/A	N/A	7.6 (6.0, 9.7)	6.3 (4.9, 8.1)	5.1 (3.9, 6.8)
2001 (13,304)	49.8 (47.2, 52.3)	29.7 (27.6, 32.0)	N/A	N/A	N/A	29.5 (27.3, 31.7)	24.0 (22.1, 26.2)	17.1 (15.5, 18.9)	N/A	N/A	N/A	7.8 (6.5, 9.4)	6.0 (4.9, 7.4)	4.9 (3.9, 6.2)
2002 (13,544)	48.6 (46.1, 51.0)	28.6 (26.4, 30.9)	N/A	N/A	N/A	26.7 (24.8, 28.6)	21.8 (20.0, 23.7)	15.5 (14.0, 17.1)	N/A	N/A	N/A	6.5 (5.2, 8.0)	5.2 (4.1, 6.6)	4.7 (3.7, 6.1)
2003 (15,200)	47.5 (45.4, 49.7)	27.9 (25.9, 30.0)	N/A	N/A	N/A	24.4 (22.6, 26.3)	20.1 (18.5, 21.8)	14.2 (12.9, 15.7)	N/A	N/A	N/A	6.7 (5.3, 8.3)	5.4 (4.4, 6.7)	4.2 (3.3, 5.3)
2004 (15,222)	48.0 (45.8, 50.3)	29.2 (27.2, 31.2)	N/A	N/A	N/A	25.0 (23.4, 26.7)	20.2 (18.8, 21.8)	14.6 (13.4, 15.9)	N/A	N/A	N/A	6.7 (5.4, 8.2)	5.1 (4.0, 6.5)	4.0 (3.0, 5.3)
2005 (15,378)	47.0 (44.6, 49.4)	27.1 (25.0, 29.2)	N/A	N/A	N/A	23.2 (21.5, 25.0)	18.4 (16.9, 20.1)	13.3 (12.0, 14.8)	N/A	N/A	N/A	7.6 (6.2, 9.3)	6.3 (5.0, 7.8)	4.9 (3.8, 6.2)
2006 (14,814)	45.3 (42.3, 48.3)	25.4 (23.1, 27.9)	N/A	N/A	N/A	21.6 (19.9, 23.3)	17.3 (15.9, 18.8)	11.7 (10.5, 13.0)	N/A	N/A	N/A	6.1 (4.9, 7.6)	5.0 (3.9, 6.3)	3.6 (2.7, 4.7)
2007 (15,132)	44.4 (41.3, 47.5)	25.9 (23.4, 28.5)	N/A	N/A	N/A	21.6 (19.9, 23.4)	17.6 (16.0, 19.3)	12.5 (11.2, 14.0)	N/A	N/A	N/A	6.6 (5.4, 8.0)	5.6 (4.5, 6.8)	4.2 (3.3, 5.3)
2008 (14,577)	43.1 (41.0, 45.3)	24.6 (22.9, 26.5)	N/A	N/A	N/A	20.4 (19.0, 21.9)	16.1 (14.9, 17.5)	11.6 (10.5, 12.7)	N/A	N/A	N/A	6.5 (5.2, 8.2)	5.2 (4.2, 6.5)	4.3 (3.3, 5.4)
2009 (14,268)	43.5 (40.7, 46.3)	25.2 (23.1, 27.5)	N/A	N/A	N/A	20.1 (18.5, 21.9)	15.9 (14.6, 17.3)	11.5 (10.5, 12.6)	N/A	N/A	N/A	8.4 (6.9, 10.1)	7.2 (5.8, 9.0)	6.4 (5.0, 8.1)
2010 (15,127)	41.2 (38.6, 43.9)	23.2 (21.1, 25.4)	N/A	N/A	N/A	19.2 (17.8, 20.7)	14.9 (13.7, 16.2)	10.5 (9.5, 11.7)	N/A	N/A	N/A	8.5 (7.1, 10.2)	6.7 (5.4, 8.3)	5.4 (4.3, 6.9)
2011 (14,855)	40.0 (38.1, 42.0)	21.6 (20.0, 23.3)	N/A	N/A	N/A	18.7 (17.0, 20.4)	14.1 (12.8, 15.5)	9.7 (8.7, 10.9)	N/A	N/A	N/A	8.3 (6.6, 10.3)	6.4 (5.0, 8.1)	5.2 (4.0, 6.7)
2012 (14,343)	41.5 (39.4, 43.6)	23.7 (21.9, 25.6)	N/A	N/A	N/A	17.1 (15.5, 18.9)	13.4 (12.1, 14.9)	9.6 (8.5, 10.8)	N/A	N/A	N/A	7.9 (6.4, 9.7)	6.4 (5.2, 8.0)	5.0 (3.9, 6.4)
2013 (13,180)	39.2 (36.7, 41.7)	22.1 (20.2, 24.1)	N/A	N/A	N/A	16.3 (14.9, 17.8)	11.8 (10.8, 12.9)	8.3 (7.5, 9.2)	N/A	N/A	N/A	8.1 (6.6, 9.9)	6.8 (5.5, 8.4)	4.9 (3.9, 6.1)
2014 (13,015)	37.4 (35.1, 39.8)	19.4 (17.9, 21.1)	N/A	N/A	N/A	13.6 (12.4, 14.9)	9.9 (8.8, 11.0)	6.6 (5.8, 7.4)	6.4 (5.4, 7.6)	5.0 (4.1, 6.0)	3.9 (3.1, 4.8)	8.4 (6.5, 10.7)	6.0 (4.6, 7.8)	4.4 (3.1, 6.2)
2015 (13,730)	35.3 (32.9, 37.9)	17.2 (15.4, 19.2)	N/A	N/A	N/A	11.4 (10.3, 12.5)	8.0 (7.1, 8.9)	5.3 (4.7, 6.1)	7.0 (6.0, 8.2)	5.2 (4.3, 6.2)	4.0 (3.2, 5.0)	6.1 (4.9, 7.6)	4.7 (3.7, 6.0)	3.0 (2.2, 4.2)
2016 (12,600)	33.2 (30.9, 35.6)	15.5 (14.1, 17.1)	N/A	N/A	N/A	10.5 (9.4, 11.7)	7.3 (6.5, 8.3)	4.5 (3.9, 5.3)	6.5 (5.4, 7.7)	4.7 (3.8, 5.9)	3.4 (2.7, 4.3)	6.6 (5.3, 8.2)	4.3 (3.2, 5.7)	2.8 (2.0, 4.1)
2017 (13,522)	33.2 (31.0, 35.5)	16.6 (14.8, 18.6)	11.0 (9.2, 13.0)	8.6 (7.1, 10.4)	5.4 (4.3, 6.6)	9.7 (8.5, 11.0)	7.0 (6.0, 8.1)	4.9 (4.1, 5.8)	5.6 (4.5, 6.9)	4.3 (3.4, 5.4)	3.3 (2.6, 4.2)	4.9 (3.8, 6.4)	3.3 (2.4, 4.7)	2.6 (1.8, 3.9)
2018 (14,502)	30.2 (28.1, 32.3)	13.8 (12.3, 15.5)	20.9 (17.7, 24.5)	13.9 (11.6, 16.7)	7.4 (5.8, 9.3)	7.6 (6.7, 8.8)	5.3 (4.5, 6.2)	3.3 (2.8, 4.0)	5.2 (4.4, 6.2)	3.5 (2.8, 4.4)	2.6 (2.1, 3.4)	4.2 (3.2, 5.4)	2.9 (2.1, 4.0)	2.2 (1.5, 3.2)

Table 2D.3 (continued)

Year (unweighted *N*)	Alcohol		Nicotine vaping			Cigarette			Large cigar			Smokeless tobacco		
	Past-30-day alcohol use	Past-2-week binge drinking	Past-30-day nicotine vaping	Co-use with alcohol	Co-use with binge drinking	Past-30-day cigarette smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day large cigar smoking	Co-use with alcohol	Co-use with binge drinking	Past-30-day smokeless tobacco use	Co-use with alcohol	Co-use with binge drinking
	Use prevalence: Weighted % (95% CI)													
2019 (13,713)	29.3 (27.2, 31.5)	14.4 (12.8, 16.1)	25.5 (22.7, 28.4)	16.2 (14.1, 18.5)	9.7 (8.2, 11.4)	5.7 (4.9, 6.7)	3.7 (3.1, 4.5)	2.7 (2.2, 3.3)	4.5 (3.7, 5.6)	3.5 (2.7, 4.4)	2.7 (2.1, 3.6)	3.5 (2.6, 4.8)	2.7 (1.9, 3.9)	1.9 (1.2, 2.9)
2020 (3,770)	33.6 (28.0, 39.7)	16.8 (12.7, 21.8)	24.7 (19.4, 30.9)	17.5 (13.2, 22.8)	11.0 (7.9, 15.2)	7.5 (4.4, 12.5)	5.5 (3.1, 9.5)	4.2 (2.2, 7.7)	2.5 (1.4, 4.5)	*	*	*	*	*
2021 (9,022)	25.8 (22.3, 29.8)	11.8 (9.8, 14.2)	19.6 (16.9, 22.6)	12.1 (10.0, 14.6)	7.2 (5.8, 9.0)	4.1 (2.7, 6.2)	2.7 (1.8, 3.9)	1.8 (1.2, 2.7)	1.9 (1.3, 2.7)	1.4 (0.9, 2.1)	0.9 (0.6, 1.5)	2.2 (1.5, 3.3)	*	*
2022 (9,599)	28.4 (25.1, 31.9)	12.6 (10.7, 14.8)	20.7 (18.2, 23.5)	13.5 (11.4, 15.9)	7.4 (6.1, 9.0)	4.0 (3.1, 5.1)	2.8 (2.1, 3.8)	2.0 (1.5, 2.7)	2.1 (1.6, 2.9)	1.7 (1.2, 2.2)	1.2 (0.8, 1.7)	3.2 (2.2, 4.7)	2.6 (1.8, 3.9)	*
2023 (7,584)	24.3 (21.1, 27.7)	10.2 (8.3, 12.4)	16.9 (13.9, 20.3)	9.9 (7.9, 12.3)	5.5 (4.2, 7.0)	2.9 (2.2, 3.9)	2.1 (1.3, 3.2)	1.3 (0.9, 2.0)	1.8 (1.0, 3.0)	1.2 (0.6, 2.4)	1.1 (0.5, 2.6)	2.5 (1.4, 4.2)	*	*

Note. CI = confidence interval; N/A = not applicable. Data are visualized in Figure 2.2. *Alcohol use* and *tobacco product use* are defined as use in the past 30 days. *Binge drinking* is defined as drinking ≥5 drinks in a row over the past 2 weeks. The large cigar question was first asked in 2014. The nicotine vaping question was first asked in 2017. Questions about certain products were only asked of some students. For large cigars and smokeless tobacco, *N* is one-sixth of *N* indicated across years. For nicotine vaping, *N* is one-third of *N* indicated in 2017 and 2018, and two-thirds of *N* indicated in 2019.

*Cells with $n < 30$ are suppressed.

Source: 2000–2023 Monitoring the Future study.

Table 2D.4 Past-30-Day Alcohol Use by Tobacco Use Status and Grade Among Students in the Monitoring the Future Study, 2023

Tobacco use status	Past-30-day alcohol use	Past-2-week binge drinking
	Use prevalence: Weighted % (95% CI)	
Nicotine vaping status		
8th Grade		
No past-30-day nicotine vaping	3.8 (2.8, 5.0)	0.9 (0.5, 1.5)
Past-30-day nicotine vaping	34.1 (29.5, 39.0)	16.0 (12.0, 21.1)
10th Grade		
No past-30-day nicotine vaping	8.5 (7.5, 9.6)	2.5 (2.1, 3.1)
Past-30-day nicotine vaping	53.1 (47.8, 58.3)	28.0 (24.0, 32.5)
12th Grade		
No past-30-day nicotine vaping	17.4 (14.7, 20.4)	5.6 (4.4, 7.1)
Past-30-day nicotine vaping	58.4 (53.7, 62.9)	32.8 (28.8, 37.1)
Cigarette smoking status		
8th Grade		
No past-30-day cigarette smoking	5.5 (4.4, 7.0)	1.7 (1.1, 2.4)
Past-30-day cigarette smoking	*	*
10th Grade		
No past-30-day cigarette smoking	12.5 (11.1, 14.0)	4.6 (3.8, 5.4)
Past-30-day cigarette smoking	69.8 (59.9, 78.1)	46.8 (39.3, 54.4)
12th Grade		
No past-30-day cigarette smoking	22.8 (20.1, 25.9)	9.0 (7.4, 11.0)
Past-30-day cigarette smoking	74.1 (59.8, 84.7)	50.7 (42.2, 59.1)
Cigar smoking status		
8th Grade		
No past-30-day cigar smoking	4.3 (3.4, 5.5)	*
Past-30-day cigar smoking	*	*
10th Grade		
No past-30-day cigar smoking	12.7 (10.4, 15.3)	4.7 (3.8, 5.9)
Past-30-day cigar smoking	*	*

Table 2D.4 (continued)

Tobacco use status	Past-30-day alcohol use	Past-2-week binge drinking
	Use prevalence: Weighted % (95% CI)	
12th Grade		
No past-30-day cigar smoking	23.5 (20.0, 27.4)	10.6 (8.5, 13.2)
Past-30-day cigar smoking	78.4 (57.9, 90.6)	66.1 (42.7, 83.6)
Smokeless tobacco use status		
8th Grade		
No past-30-day smokeless tobacco use	4.8 (3.7, 6.2)	1.3 (0.8, 2.0)
Past-30-day smokeless tobacco use	*	*
10th Grade		
No past-30-day smokeless tobacco use	13.3 (11.5, 15.4)	4.4 (3.3, 5.7)
Past-30-day smokeless tobacco use	47.1 (29.2, 65.8)	*
12th Grade		
No past-30-day smokeless tobacco use	24.3 (19.6, 29.6)	9.7 (7.5, 12.5)
Past-30-day smokeless tobacco use	*	*

Note. CI = confidence interval. Data are visualized in Figure 2.3. *Alcohol use* and *tobacco product use* are defined as use in the past 30 days. *Binge drinking* is defined as drinking ≥5 drinks in a row over the past 2 weeks.
*Cells with $n < 30$ are suppressed.
Source: 2023 Monitoring the Future study.

Table 2D.5 Characteristics of Participants in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125)

	Overall		Men		Women	
	N	Weighted % (95% CI)	*n*	Weighted % (95% CI)	*n*	Weighted % (95% CI)
Total	14,125	100.0	5,702	48.2 (47.0, 49.4)	8,417	51.8 (50.6, 53.0)
Age						
18–29	2,433	19.6 (18.6, 20.7)	1,032	20.4 (18.9, 22.0)	1,401	18.9 (17.6, 20.3)
30–59	7,153	52.8 (51.5, 54.0)	2,791	53.6 (51.8, 55.5)	4,361	52.0 (50.4, 53.6)
≥60	4,438	27.6 (26.6, 28.7)	1,853	26.0 (24.5, 27.5)	2,583	29.1 (27.7, 30.6)
Race or ethnicity[a]						
White	7,183	64.7 (63.6, 65.8)	3,003	65.1 (63.4, 66.8)	4,180	64.4 (62.9, 65.9)
Black or African American	3,096	12.5 (11.9, 13.3)	1,119	11.7 (10.7, 12.8)	1,977	13.4 (12.4, 14.3)
Hispanic, Latino/a, or of Spanish origin	2,795	14.9 (14.1, 15.7)	1,102	15.3 (14.1, 16.6)	1,692	14.5 (13.5, 15.5)
Other	1,051	7.9 (7.2, 8.6)	478	7.8 (6.8, 9.0)	568	7.8 (6.9, 8.7)
Education						
High school or less	4,156	35.2 (34.0, 36.4)	1,631	34.7 (32.9, 36.6)	2,524	35.6 (34.0, 37.2)
Some college	4,245	34.9 (33.7, 36.1)	1,621	33.8 (32.1, 35.7)	2,623	35.8 (34.3, 37.4)
College degree	5,690	30.0 (28.9, 31.0)	2,438	31.5 (29.9, 33.1)	3,252	28.6 (27.2, 29.9)
Tobacco use status[b]						
Never	8,998	60.4 (59.2, 61.6)	3,347	56.4 (54.5, 58.2)	5,648	64.2 (62.6, 65.7)
Former	2,559	18.9 (17.9, 19.8)	1,157	20.2 (18.7, 21.7)	1,401	17.6 (16.4, 18.9)
Current (past-year)	2,432	20.7 (19.7, 21.8)	1,137	23.4 (21.8, 25.2)	1,295	18.2 (16.9, 19.6)
Nondaily	601	4.2 (3.7, 4.7)	311	5.2 (4.4, 6.1)	290	3.3 (2.7, 3.9)
Daily	1,831	16.5 (15.6, 17.5)	826	18.2 (16.7, 19.9)	1,005	14.9 (13.7, 16.2)
Cigarette pack years[c]						
0 pack years	3,392	56.7 (54.7, 58.7)	1,188	51.2 (48.1, 54.2)	2,204	61.9 (59.3, 64.5)
0.1–20 pack years	1,204	23.5 (21.8, 25.2)	514	23.9 (21.4, 26.7)	690	23.0 (20.8, 25.4)
20.1–40 pack years	382	7.2 (6.3, 8.3)	189	8.5 (7.0, 10.3)	193	6.1 (4.9, 7.5)
≥40.1 pack years	654	12.5 (11.3, 14.0)	368	16.4 (14.3, 18.8)	286	8.9 (7.5, 10.6)

Table 2D.5 (continued)

	Overall		Men		Women	
	N	Weighted % (95% CI)	*n*	Weighted % (95% CI)	*n*	Weighted % (95% CI)
Alcohol use (past year)[d]						
No alcohol use	4,531	31.2 (30.0, 32.3)	1,610	27.4 (25.7, 29.0)	2,918	34.7 (33.1, 36.2)
Alcohol use within guidelines	5,564	38.9 (37.7, 40.1)	2,337	39.1 (37.3, 40.9)	3,227	38.7 (37.2, 40.3)
Alcohol use exceeding guidelines	3,676	30.0 (28.8, 31.2)	1,635	33.6 (31.8, 35.4)	2,041	26.6 (25.1, 28.1)
Lifetime 5+ drinking days[e]						
0.00	7,231	44.8 (43.6, 46.0)	2,122	31.8 (30.2, 33.5)	5,104	56.8 (55.1, 58.4)
0.1–500	3,832	30.1 (28.1, 31.2)	1,780	33.1 (31.3, 34.8)	2,102	27.4 (25.9, 28.9)
500+	3,012	25.1 (24.1, 26.3)	1,800	35.1 (33.3, 36.9)	1,211	15.9 (14.7, 17.1)
Hazardous alcohol use (past-year)						
No past-year 5+ drinking[f]	11,513	78.3 (77.2, 79.3)	4,194	69.4 (67.6, 71.1)	7,314	86.6 (85.4, 87.7)
Past-year 5+ drinking	2,608	21.7 (20.7, 22.8)	1,505	30.6 (28.9, 32.4)	1,102	13.4 (12.3, 14.6)
No past-year HID[g]	13,378	94.0 (93.3, 94.6)	5,400	93.3 (92.3, 94.3)	7,978	94.6 (93.8, 95.3)
Past-year HID	741	6.0 (5.5, 6.7)	302	6.7 (5.7, 7.7)	439	5.4 (4.7, 6.2)

Note. CI = confidence interval; HID = high intensity drinking.
[a]White and Black participants are non-Hispanic. [b]*Tobacco use* is defined as use of products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [c]Pack years data are available only for the 2014–2015 NAS. [d]Alcohol use is measured in the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking ≥4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66] [e]*Lifetime 5+ drinking days* is defined as the number of days over lifetime in which participants reported drinking 5 or more drinks on 1 occasion. [f]Alcohol use is measured in the past year. *5+ drinks* is defined as consuming 5 or more drinks in 1 day. [g]Alcohol use is measured in the past year. *HID* is defined as drinking ≥8/12 drinks in one day for women/men.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2D.6 Characteristics of Participants in the 2015–2019 National Survey on Drug Use and Health (*N* = 214,505)

	Overall		Men		Women	
	N	Weighted % (95 % CI)	*n*	Weighted % (95% CI)	*n*	Weighted % (95% CI)
Total	214,505	100.0	99,769	48.3 (47.9, 48.6)	114,736	51.8 (51.4, 52.1)
Race or ethnicity[a]						
White	128,924	63.9 (63.6, 64.2)	60,618	64.3 (63.9, 64.8)	68,306	63.4 (63.0, 63.9)
Black	27,081	11.9 (11.7, 12.1)	11,885	11.1 (10.9, 11.4)	15,196	12.5 (12.3, 12.8)
Hispanic	37,015	16.0 (15.8, 16.3)	17,053	16.5 (16.2, 16.9)	19,962	15.6 (15.3, 15.9)
Multirace	7,041	1.7 (1.6, 1.8)	3,286	1.6 (1.5, 1.7)	3,755	1.8 (1.7, 1.9)
American Indian, Alaska Native, Native Hawaiian, or Other Pacific Islander	4,169	0.9 (0.9, 1.0)	1,981	0.9 (0.8, 1.0)	2,188	0.9 (0.9, 1.0)
Asian	10,275	5.6 (5.4, 5.8)	4,946	5.5 (5.2, 5.7)	5,329	5.7 (5.5, 6.0)
Education						
High school or less	84,913	37.6 (37.3, 37.9)	43,401	39.9 (39.5, 40.4)	41,512	35.3 (34.9, 35.7)
Some college	71,981	30.8 (30.6, 31.1)	31,091	28.8 (28.4, 29.2)	40,890	32.8 (32.3, 33.2)
College degree	57,611	31.6 (31.3, 31.9)	25,277	31.3 (30.8, 31.7)	32,334	31.9 (31.5, 32.3)
Age category						
18–25	69,916	13.9 (13.8, 14.1)	33,739	14.5 (14.3, 14.7)	36,177	13.4 (13.2, 13.6)
26–34	44,016	16.0 (15.8, 16.2)	20,219	16.4 (16.2, 16.7)	23,797	15.4 (15.3, 15.8)
35–49	56,566	24.7 (24.4, 24.9)	25,740	25.1 (24.7, 25.4)	30,826	24.3 (24.0, 24.6)
50–64	25,213	25.3 (25.0, 25.6)	11,605	25.3 (24.8, 25.8)	13,608	25.3 (24.9, 25.7)
65+	18,794	20.2 (19.9, 20.5)	8,466	18.8 (18.3, 19.2)	10,328	21.5 (21.1, 21.9)
Income						
<$20,000	42,934	16.3 (16.1, 16.5)	17,661	14.1 (13.8, 14.4)	25,273	18.3 (18.0, 18.6)
$20,000–$49,999	66,946	29.5 (29.2, 29.7)	30,479	28.3 (27.9, 28.7)	36,467	30.5 (30.1, 30.9)
$50,000–$74,999	33,515	16.0 (15.8, 16.2)	16,006	16.2 (15.9, 16.6)	17,509	15.8 (15.5, 16.1)
≥$75,000	71,110	38.3 (38.0, 38.6)	35,623	41.3 (40.9, 41.8)	35,487	35.4 (35.0, 35.8)
Metropolitan status[b]						
Nonmetropolitan	42,006	14.2 (14.0, 14.4)	19,601	14.1 (13.8, 14.4)	22,405	14.2 (13.9, 14.5)
Metropolitan	172,499	85.9 (85.7, 86.1)	80,168	85.9 (85.6, 86.2)	92,331	85.8 (85.5, 86.1)

Table 2D.6 (continued)

	Overall		Men		Women	
	N	Weighted % (95 % CI)	*n*	Weighted % (95% CI)	*n*	Weighted % (95% CI)
Sexual orientation						
Heterosexual	195,385	95.0 (94.8, 95.1)	93,506	95.9 (95.8, 96.1)	101,879	94.1 (83.9, 94.2)
Lesbian, gay, or bisexual	15,007	5.0 (4.9, 5.2)	4,698	4.1 (3.9, 4.2)	10,309	6.0 (5.8, 6.1)
Veteran status[c]						
Not a veteran	202,629	91.4 (91.2, 91.6)	89,391	83.6 (83.3, 84.0)	113,238	98.6 (98.5, 98.7)
Veteran	11,876	8.6 (8.4, 8.8)	10,378	16.4 (16.0, 16.8)	1,498	1.4 (1.3, 1.5)
Health condition[d]						
Cancer	7,635	6.3 (6.1, 6.5)	2,831	5.4 (5.2, 5.6)	4,804	7.2 (6.9, 7.4)
Hypertension	26,188	19.6 (19.3, 19.9)	11,531	18.2 (17.8, 18.6)	14,657	20.9 (20.5, 21.3)
Heart condition	14,151	10.5 (10.3, 10.8)	7,008	11.7 (11.4, 12.1)	7,143	9.5 (9.2, 9.7)
Diabetes	14,539	10.7 (10.4, 10.9)	6,379	10.7 (10.3, 11.0)	8,160	10.7 (10.4, 11.0)
Mental distress[e]						
None/low (Score of 0–4 on Kessler K6 nonspecific distress scale)	131,014	68.1 (67.8, 68.4)	64,351	70.9 (70.5, 71.3)	66,663	65.5 (65.1, 65.9)
Moderate (Score of 5–12 on Kessler K6 nonspecific distress scale)	66,045	26.1 (25.8, 26.3)	28,823	24.2 (23.8, 24.6)	37,222	27.8 (27.4, 28.2)
Severe (Score of 13–24 on Kessler K6 nonspecific distress scale)	17,446	5.8 (5.7, 6.0)	6,595	4.9 (4.7, 5.1)	10,851	6.8 (6.6, 6.9)
Cigarette smoking status[f]						
Never	86,348	38.8 (38.5, 39.1)	35,154	33.2 (32.7, 33.6)	51,194	44.0 (43.5, 44.4)
Former	79,438	41.8 (41.5, 42.1)	39,236	45.0 (44.5, 45.4)	40,202	38.8 (38.4, 39.3)
Current (past-30-day)	48,321	19.5 (19.2, 19.7)	25,148	21.9 (21.5, 22.2)	23,173	17.2 (16.9, 17.5)
Current nondaily smoking, <300 cigarettes/ month	21,015	7.7 (7.5, 7.8)	11,664	9.1 (8.8, 9.3)	9,351	6.4 (6.2, 6.5)
Current daily smoking, <300 cigarettes/month	6,105	2.4 (2.3, 2.4)	2,605	2.2 (2.1, 2.3)	3,500	2.5 (2.4, 2.6)
Current daily or nondaily smoking, ≥300 cigarettes/month	21,201	9.4 (9.3, 9.6)	10,879	10.6 (10.3, 10.9)	10,322	8.3 (8.1, 8.6)

Table 2D.6 (continued)

	Overall		Men		Women	
	N	Weighted % (95 % CI)	*n*	Weighted % (95% CI)	*n*	Weighted % (95% CI)
Smokeless tobacco use status						
Never	173, 048	83.0 (82.8, 83.2)	66,928	70.5 (70.1, 70.9)	106,120	94.7 (94.5, 94.8)
Former	32,054	13.7 (13.5, 13.9)	24,390	23.2 (22.8, 23.5)	7,664	4.8 (4.6, 5.0)
Current (past-30-day) nondaily use	5,304	1.7 (1.7, 1.8)	4,626	3.2 (3.1, 3.4)	678	0.4 (0.3, 0.4)
Current (past-30-day) daily use	4,013	1.6 (1.5, 1.7)	3,789	3.2 (3.1, 3.3)	224	0.1 (0.1, 0.1)
Cigar smoking status						
Never	138,181	65.6 (65.3, 65.9)	49,260	48.4 (47.9, 48.8)	88,921	81.6 (81.3, 81.9)
Former	63,395	29.7 (29.4, 30.0)	41,161	43.9 (43.5, 44.4)	22,234	16.4 (16.1, 16.7)
Current (past-30-day) cigar smoking	12,867	4.7 (4.6, 4.8)	9,319	7.7 (7.5, 8.0)	3,548	2.0 (1.9, 2.1)
Alcohol use (past 30 days)[g]						
Never	30,427	14.2 (14.0, 14.5)	12,697	11.3 (11.0, 11.6)	17,730	17.0 (16.7, 17.3)
Former	63,147	31.6 (31.3, 31.9)	27,158	30.0 (29.5, 30.4)	35,989	33.2 (32.8, 33.6)
Past-30-day alcohol use within guidelines	52,558	27.8 (27.5, 28.1)	23,930	28.1 (27.6, 28.5)	28,628	27.5 (27.1, 27.9)
Past-30-day binge, no risky weekly use	43,212	17.0 (16.8, 17.2)	23,958	21.0 (20.7, 21.4)	19,254	13.3 (13.0, 13.5)
Past-30-day risky weekly use	21,666	9.3 (9.2, 9.5)	10,195	9.7 (9.4, 9.9)	11,471	9.0 (8.8, 9.3)

Note. CI = confidence interval.
[a]Categories other than Hispanic are non-Hispanic. *Multirace* is defined as two or more races. [b]NSDUH categorizes participants' counties of residence as large metropolitan, small metropolitan, and nonmetropolitan; large and small metropolitan counties were defined as metropolitan in this analysis. [c]*Veterans* are participants who indicated they are "now separated or retired from the military". [d]*Health conditions* are defined as a condition that a doctor or other health care professional has ever told participants that they had, including "cancer," "any kind of heart condition or heart disease," "diabetes or sugar diabetes," and "hypertension, also called high blood pressure." [e]*Mental distress* levels are based on scores on the Kessler K6 nonspecific distress scale, where a score of 0–4 = none/low, 5–12 = moderate, and 13–24 = severe. [f]*Cigarette smoking*, *smokeless tobacco use*, and *cigar smoking* are defined as use in the past 30 days. *Former smoking*, *smokeless tobacco use*, and *cigar smoking* are defined as use within lifetime but not in the past 30 days. *Never smoking, smokeless tobacco use*, and *cigar smoking* are defined as never use in lifetime. [g]*Never and former alcohol use* are defined based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 2D.7 Co-Use Prevalence in the Past Year in 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125) and in the Past Month in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684)

Co-use status (unweighted *n*)	Weighted % (95% CI)
NAS 2014–2015 and 2019–2020	
No past-year tobacco or alcohol use (3,938)	25.6 (24.6, 26.7)
Alcohol use only in past year (7,755)	53.9 (52.7, 55.1)
Tobacco use only in past year (593)	4.8 (4.3, 5.4)
Co-use of tobacco and alcohol in past year (1,839)	15.6 (14.7, 16.6)
NSDUH 2015–2019	
No past-30-day cigarette smoking or alcohol use (76,540)	38.4 (38.0, 38.7)
Alcohol use only in the past month (86,934)	42.3 (42.0, 42.6)
Cigarette smoking only in the past month (16,814)	7.5 (7.3, 7.6)
Co-use of cigarettes and alcohol in the past month (30,396)	11.8 (11.7, 12.0)

Note. CI = confidence interval; NAS = National Alcohol Surveys; NSDUH = National Survey on Drug Use and Health. Data are visualized in Figure 2.4. *Tobacco use* in NAS is defined as use of products other than e-cigarettes (due to survey question wording). *Tobacco* and *alcohol use* in the NAS are defined as use in the past year. *Cigarette smoking* and *alcohol use* in the NSDUH are defined as use in the past 30 days.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys, 2015–2019 National Survey on Drug Use and Health.

Table 2D.8 Past-Year Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]
	Alcohol use: Weighted % (95% CI)			
Overall				
Overall (13,639)	31.2 (30.0, 32.3)	38.9 (37.7, 40.1)	30.0 (28.8, 31.2)	
Never (8,768)	35.1 (33.7, 36.6)	39.1 (37.6, 40.6)	25.8 (24.4, 27.2)	<.001
Former (2,489)	26.9 (24.6, 29.4)	46.2 (43.4, 49.0)	26.9 (24.4, 29.6)	
Current (past-year) (2,382)	24.0 (21.6, 26.6)	31.9 (29.2, 34.7)	44.1 (41.2, 47.1)	
Nondaily (592)	13.8 (10.5, 18.0)	30.1 (24.9, 35.9)	56.1 (50.0, 61.9)	
Daily (1,790)	26.6 (23.8, 29.6)	32.3 (29.3, 35.5)	41.1 (37.7, 44.4)	
Men				
Overall (5,523)	27.4 (25.7, 29.0)	39.1 (37.3, 40.9)	33.6 (31.8, 35.4)	
Never (3,280)	31.0 (28.8, 33.3)	39.0 (36.7, 41.4)	30.0 (27.7, 32.4)	<.001
Former (1,130)	25.9 (22.6, 29.6)	47.4 (43.4, 51.4)	26.7 (23.1, 30.5)	
Current (past-year) (1,113)	20.4 (17.3, 23.8)	32.2 (28.4, 36.3)	47.4 (43.2, 51.6)	
Nondaily (305)	12.1 (8.0, 17.9)	32.9 (25.6, 41.1)	55.0 (46.7, 63.0)	
Daily (808)	22.7 (19.0, 26.9)	32.1 (27.7, 36.8)	45.2 (40.4, 50.1)	
Women				
Overall (8,061)	34.7 (33.1, 36.2)	38.7 (37.2, 40.3)	26.6 (25.1, 28.1)	
Never (5,487)	38.6 (36.6, 40.5)	39.2 (37.3, 41.1)	22.3 (20.6, 24.0)	<.001
Former (1,359)	27.9 (24.8, 31.3)	44.9 (41.0, 48.8)	27.2 (23.8, 30.9)	
Current (past-year) (1,269)	28.3 (24.8, 32.2)	31.4 (27.8, 35.3)	40.2 (36.2, 44.4)	
Nondaily (287)	16.3 (11.2, 23.0)	26.1 (19.7, 33.9)	57.6 (48.9, 65.8)	
Daily (982)	31.0 (26.9, 35.4)	32.6 (28.5, 37.0)	36.4 (32.0, 40.9)	

Note. CI = confidence interval. Data are visualized in Figure 2.5.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]Alcohol use is measured in the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking ≥4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66] [c]*p* value for bivariate association, obtained through a chi-squared test, between tobacco use categories (never, former, and current [past-year]) and alcohol use categories within each sex and age group.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2D.9 Past-Year Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex, Stratified by Age *(Row Percents)*

Tobacco use status[a] (weighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
Overall												
Overall (13,548)	27.7 (25.2, 30.4)	26.4 (23.8, 29.0)	45.9 (42.9, 48.9)		27.6 (26.1, 29.2)	40.1 (38.4, 41.8)	32.3 (30.6, 34.0)		40.2 (38.1, 42.3)	45.6 (43.5, 47.7)	14.2 (12.8, 15.8)	
Never (8,699)	33.2 (30.1, 36.5)	26.4 (23.5, 29.5)	40.4 (37.1, 43.8)	<.001	30.6 (28.6, 32.6)	42.9 (40.8, 45.1)	26.5 (24.5, 28.5)	<.001	47.3 (44.4, 50.3)	43.1 (40.2, 45.9)	9.6 (8.1, 11.4)	<.001
Former (2,470)	15.6 (8.3, 27.5)	34.0 (22.5, 47.8)	50.3 (37.0, 63.6)		22.5 (19.0, 26.5)	42.3 (38.0, 46.8)	35.1 (31.0, 39.5)		31.7 (28.4, 35.1)	50.9 (47.2, 54.6)	17.5 (14.7, 20.6)	
Current (past-year) (2,379)	14.1 (9.9, 19.6)	24.1 (18.6, 30.5)	61.9 (54.9, 68.4)		23.5 (20.5, 26.8)	31.1 (27.6, 34.8)	45.4 (41.6, 49.3)		35.6 (30.1, 41.5)	42.2 (36.5, 48.2)	22.2 (17.8, 27.4)	
Nondaily (592)	8.7 (4.9, 15.0)	18.8 (11.9, 28.3)	72.5 (62.4, 80.8)		14.4 (9.8, 20.8)	29.9 (23.0, 37.9)	55.7 (47.5, 63.6)		23.6 (14.0, 37.0)	58.3 (44.4, 71.0)	18.1 (10.5, 29.4)	
Daily (1,787)	16.7 (11.0, 24.5)	26.7 (19.6, 35.1)	56.7 (47.8, 65.1)		25.5 (22.0, 29.4)	31.3 (27.4, 35.5)	43.1 (38.8, 47.5)		37.5 (31.4, 44.0)	39.6 (33.4, 46.1)	22.9 (18.0, 28.7)	
Men												
Overall (5,500)	26.5 (23.0, 30.4)	25.5 (21.9, 29.4)	48.0 (43.7, 52.4)		24.9 (22.7, 27.2)	38.4 (35.9, 41.0)	36.7 (34.1, 39.3)		33.0 (30.0, 36.2)	51.4 (48.2, 54.7)	15.5 (13.4, 17.9)	
Never (3,263)	34.5 (29.9, 39.5)	24.3 (20.3, 28.8)	41.2 (36.3, 46.3)	<.001	27.2 (24.3, 30.3)	41.6 (38.3, 45.0)	31.2 (28.0, 34.6)	<.001	37.1 (32.6, 41.7)	50.9 (46.3, 55.5)	12.1 (9.5, 15.2)	.01
Former (1,125)	*	31.9 (16.9, 51.9)	67.3 (47.4, 82.4)		22.8 (17.6, 29.0)	41.8 (35.4, 48.4)	35.4 (29.3, 42.1)		30.7 (26.2, 35.6)	53.5 (48.3, 58.5)	15.8 (12.5, 19.9)	
Current (past-year) (1,112)	11.7 (7.0, 18.8)	27.2 (19.6, 36.5)	61.1 (51.5, 69.9)		21.0 (17.0, 25.6)	28.9 (24.1, 34.2)	50.1 (44.6, 55.6)		28.7 (21.3, 37.4)	47.8 (39.3, 56.5)	23.5 (17.2, 31.1)	
Nondaily (305)	*	18.2 (9.9, 31.0)	74.1 (60.6, 84.2)		15.5 (9.2, 25.2)	30.9 (21.3, 42.5)	53.6 (42.3, 64.6)		*	76.2 (59.9, 87.2)	*	
Daily (807)	13.6 (7.4, 23.7)	31.6 (21.4, 43.8)	54.8 (42.7, 66.5)		22.4 (17.8, 27.8)	28.4 (23.0, 34.5)	49.2 (42.9, 55.5)		32.3 (23.8, 42.1)	42.6 (33.4, 52.2)	25.2 (18.1, 33.9)	

Table 2D.9 (continued)

Tobacco use status[a] (weighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
Women												
Overall (8,048)	29.0 (25.4, 32.8)	27.3 (23.9, 31.0)	43.8 (39.8, 47.8)		30.2 (28.2, 32.3)	41.8 (39.6, 44.0)	28.0 (26.0, 30.1)		46.2 (43.4, 49.1)	40.7 (37.9, 43.5)	13.1 (11.2, 15.3)	
Never (5,436)	32.0 (27.9, 36.5)	28.3 (24.4, 32.6)	39.6 (35.2, 44.2)	<.001	33.7 (31.1, 36.4)	44.1 (41.4, 46.8)	22.2 (20.0, 24.6)	<.001	53.7 (49.9, 57.4)	38.2 (34.7, 41.8)	8.1 (6.3, 10.4)	<.001
Former (1,345)	28.2 (15.2, 46.2)	35.8 (20.3, 54.9)	36.1 (21.8, 53.3)		22.3 (17.9, 27.3)	42.9 (37.1, 49.0)	34.8 (29.5, 40.6)		32.7 (28.3, 37.5)	47.9 (42.7, 53.2)	19.3 (15.1, 24.4)	
Current (past-year) (1,267)	17.6 (10.8, 27.4)	19.3 (13.1, 27.6)	63.0 (52.8, 72.1)		26.3 (21.9, 31.3)	33.5 (28.7, 38.7)	40.1 (35.0, 45.5)		43.2 (35.5, 51.2)	36.0 (28.8, 43.9)	20.8 (15.1, 28.1)	
Nondaily (287)	*	19.6 (9.7, 35.7)	70.2 (53.8, 82.7)		12.7 (7.3, 21.3)	28.5 (19.9, 39.0)	58.7 (47.6, 69.1)		43.8 (25.7, 63.7)	32.6 (18.5, 50.7)	23.6 (11.7, 41.8)	
Daily (980)	21.4 (12.2, 34.8)	19.2 (11.9, 29.5)	59.4 (46.8, 70.9)		28.9 (23.8, 34.6)	34.5 (29.0, 40.3)	36.6 (31.1, 42.6)		43.1 (34.8, 51.8)	36.4 (28.6, 45.1)	20.5 (14.3, 28.5)	

Note. CI = confidence interval. Data are visualized in Figure 2.5.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]Alcohol use is measured in the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking at least 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66] [c]*p* value for bivariate association, obtained through a chi-squared test, between tobacco use categories (never, former, and current [past-year]) and alcohol use categories within each sex and age group.
*Cells with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2D.10 Past-Year Hazardous Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex, Stratified by Age *(Row Percents)*

Tobacco use status[a] (weighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
Overall																		
Overall (13,887)	63.7 (60.8, 66.5)	36.3 (33.5, 39.2)		87.6 (85.5, 89.5)	12.4 (10.5, 14.5)		76.0 (74.5, 77.5)	24.0 (22.5, 25.5)		93.7 (92.8, 94.5)	6.3 (5.5, 7.2)		92.7 (91.5, 93.7)	7.3 (6.3, 8.5)		98.8 (98.3, 99.2)	1.2 (0.8, 1.7)	
Never (8,925)	69.4 (66.2, 72.5)	30.6 (27.5, 33.8)	<.001	90.9 (88.8, 92.6)	9.1 (7.4, 11.2)	<.001	80.8 (78.9, 82.6)	19.2 (17.4, 21.1)	<.001	95.4 (94.3, 96.3)	4.6 (3.7, 5.7)	<.001	95.5 (94.3, 96.5)	4.5 (3.5, 5.7)	<.001	99.2 (98.7, 99.5)	0.8 (0.5, 1.3)	<.001
Former (2,536)	61.2 (47.8, 73.2)	38.8 (26.8, 52.2)		92.3 (86.1, 95.8)	7.7 (4.2, 13.9)		75.0 (71.0, 78.6)	25.0 (21.4, 29.0)		95.3 (93.1, 96.8)	4.7 (3.2, 6.9)		91.3 (89.0, 93.1)	8.7 (6.9, 11.0)		99.1 (98.2, 99.5)	0.9 (0.5, 1.8)	
Current (past-year) (2,426)	46.5 (39.7, 53.6)	53.5 (46.4, 60.3)		76.9 (70.5, 82.3)	23.1 (17.7, 29.5)		64.1 (60.3, 67.7)	35.9 (32.3, 39.7)		88.2 (85.6, 90.4)	11.8 (9.6, 14.4)		86.2 (81.7, 89.6)	13.8 (10.4, 18.3)		97.0 (94.4, 98.4)	3.0 (1.6, 5.6)	
Nondaily (601)	35.2 (25.5, 46.3)	64.8 (53.7, 74.5)		79.8 (69.7, 87.2)	20.2 (12.8, 30.3)		57.0 (48.8, 64.8)	43.0 (35.2, 51.2)		87.7 (81.2, 92.2)	12.3 (7.8, 18.8)		90.4 (80.6, 95.5)	9.6 (4.5, 19.4)		98.9 (95.3, 99.8)	*	
Daily (1,825)	52.0 (43.3, 60.7)	48.0 (39.3, 56.7)		75.5 (67.1, 82.3)	24.5 (17.7, 32.9)		65.7 (61.4, 69.7)	34.3 (30.3, 38.6)		88.3 (85.4, 90.8)	11.7 (9.2, 14.6)		85.5 (80.5, 89.3)	14.5 (10.7, 19.5)		96.7 |(93.7, 98.3)	3.3 (1.7, 6.3)	
Men																		
Overall (5,613)	55.6 (51.3, 59.8)	44.4 (40.2, 48.7)		88.4 (85.2, 91.0)	11.6 (9.0, 14.8)		65.8 (63.2, 68.4)	34.2 (31.6, 36.8)		92.8 (91.2, 94.1)	7.2 (5.9, 8.8)		87.4 (85.2, 89.4)	12.6 (10.6, 14.8)		98.2 (97.2, 98.9)	1.8 (1.1, 2.8)	
Never (3,330)	61.7 (56.7, 66.5)	38.3 (33.5, 43.3)	<.001	91.1 (87.5, 93.7)	8.9 (6.3, 12.5)	.002	70.3 (67.0, 73.4)	29.7 (26.6, 33.0)	<.001	94.1 (92.0, 95.7)	5.9 (4.3, 8.0)	<.001	90.8 (88.0, 93.0)	9.2 (7.0, 12.0)	.002	98.4 (97.0, 99.1)	1.6 (0.9, 3.0)	.049
Former (1,149)	38.0 (21.9, 57.3)	62.0 (42.7, 78.1)		95.1 (84.2, 98.6)	*		67.8 (61.4, 73.7)	32.2 (26.3, 38.6)		95.9 (92.1, 98.0)	4.1 (2.0, 7.9)		87.1 (83.2, 90.1)	12.9 (9.9, 16.8)		99.0 (97.5, 99.6)	*	
Current (past-year) (1,134)	43.4 (34.3, 53.0)	56.6 (47.0, 65.7)		80.5 (72.0, 87.0)	19.5 (13.0, 28.0)		54.2 (48.7, 59.6)	45.8 (40.4, 51.3)		87.9 (84.1, 90.9)	12.1 (9.1, 15.9)		80.1 (72.7, 85.9)	19.9 (14.1, 27.3)		96.2 (91.3, 98.4)	3.8 (1.6, 8.7)	
Nondaily (311)	27.7 (17.4, 41.2)	72.3 (58.8, 82.6)		86.2 (75.9, 92.6)	13.8 (7.4, 24.1)		48.4 (37.4, 59.4)	51.6 (40.6, 62.6)		89.7 (81.5, 94.5)	10.3 (5.5, 18.5)		87.5 (71.8, 95.1)	*		99.4 (95.6, 99.9)	*	
Daily (823)	50.9 (39.0, 62.7)	49.1 (37.3, 61.0)		77.8 (66.0, 86.4)	22.2 (13.6, 34.0)		55.8 (49.5, 61.9)	44.2 (38.1, 50.5)		87.4 (82.9, 90.8)	12.6 (9.2, 17.1)		78.7 (70.3, 85.3)	21.3 (14.7, 29.7)		95.6 (89.9, 98.1)	4.4 (1.9, 10.1)	

Table 2D.10 (continued)

Tobacco use status[a] (weighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
Women																		
Overall (8,272)	71.9 (68.2, 75.4)	28.1 (24.6, 31.8)		86.8 (83.9, 89.3)	13.2 (10.7, 16.1)		85.9 (84.2, 87.3)	14.1 (12.7, 15.8)		94.6 (93.5, 95.5)	5.4 (4.5, 6.5)		97.2 (96.2, 97.9)	2.8 (2.1, 3.8)		99.3 (98.9, 99.6)	0.7 (0.4, 1.1)	
Never (5,594)	76.4 (72.4, 80.0)	23.6 (20.0, 27.6)	<.001	90.7 (88.0, 92.8)	9.3 (7.2, 12.0)	<.001	90.1 (88.4, 91.6)	9.9 (8.4, 11.6)	<.001	96.6 (95.4, 97.4)	3.4 (2.6, 4.6)	<.001	98.5 (97.1, 99.2)	1.5 (0.8, 2.9)	<.001	99.8 (99.4, 99.9)	*	.001
Former (1,386)	81.1 (67.2, 90.0)	18.9 (10.0, 32.8)		89.9 (80.3, 95.0)	10.1 (5.0, 19.7)		82.5 (77.9, 86.3)	17.5 (13.7, 22.1)		94.6 (91.6, 96.6)	5.4 (3.4, 8.4)		96.6 (94.8, 97.8)	3.4 (2.2, 5.2)		99.1 (97.8, 99.7)	*	
Current (past-year) 1,292)	51.3 (41.2, 61.3)	48.7 (38.7, 58.8)		71.4 (61.2, 79.8)	28.6 (20.2, 38.8)		75.4 (70.5, 79.7)	24.6 (20.3, 29.5)		88.6 (84.8, 91.6)	11.4 (8.4, 15.2)		92.8 (88.3, 95.7)	7.2 (4.3, 11.7)		97.9 (95.3, 99.1)	*	
Nondaily (290)	46.3 (29.5, 64.0)	53.7 (36.0, 70.5)		70.3 (51.3, 84.2)	29.7 (15.8, 48.7)		69.9 (58.3, 79.4)	30.1 (20.6, 41.7)		84.7 (72.7, 92.0)	15.3 (8.0, 27.3)		94.5 (82.5, 98.4)	*		98.2 (88.1, 99.8)	*	
Daily (1,002)	53.7 (41.5, 65.6)	46.3 (34.4, 58.5)		72.0 (59.4, 81.8)	28.0 (18.2, 40.6)		76.4 (70.9, 81.1)	23.6 (18.9, 29.1)		89.4 (85.3, 92.4)	10.6 (7.6, 14.7)		92.6 (87.5, 95.7)	7.4 (4.3, 12.5)		97.8 (94.9, 99.1)	*	

Note. CI = confidence interval; HID = high intensity drinking.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]Alcohol use is measured in the past year. *5+ drinks* is defined as consuming 5 or more drinks in 1 day. [c]Alcohol use is measured in the past year. *HID* is defined as drinking at ≥ 8/12 drinks in one day for women/men. [d]*p* value for bivariate association, obtained through a chi-squared test, between tobacco use categories (never, former, and current [past-year]) and alcohol use categories within each sex and age group.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2D.11 Past-Year Alcohol Use by E-Cigarette Status in the 2019–2020 National Alcohol Survey (*N* = 8,096), Overall and by Sex, Stratified by Age *(Row Percents)*

E-cigarette use status[a] (weighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
Overall												
No e-cigarette use in past year (7,918)	29.7 (26.8, 32.7)	30.9 (27.9, 34.0)	39.5 (36.4, 42.7)	<.001	26.3 (24.6, 28.1)	42.9 (40.9, 44.9)	30.8 (29.0, 32.7)	<.001	33.1 (30.5, 35.8)	49.5 (46.7, 52.3)	17.3 (15.3, 19.6)	.02
Monthly or semi-monthly use in past year(204)	*	23.1 (11.9, 40.0)	67.6 (51.7, 80.3)		18.8 (11.4, 29.6)	33.2 (23.0, 45.2)	48.0 (36.4, 59.8)		*	*	*	
Daily or nearly daily use in past year (177)	*	23.1 (13.1, 37.6)	66.1 (51.6, 78.1)		15.0 (7.8, 26.9)	30.8 (20.9, 42.9)	54.2 (41.8, 66.2)		*	*	*	
Men												
No e-cigarette use in past year (3,199)	30.3 (25.9, 35.1)	29.8 (25.2, 34.7)	40.0 (35.2, 45.0)	<.001	24.9 (22.3, 27.8)	41.6 (38.5, 44.8)	33.5 (30.5, 36.6)	.19	24.7 (21.5, 28.2)	56.0 (52.0, 59.9)	19.3 (16.3, 22.7)	.35
Monthly or semi-monthly use in past year (97)	*	*	69.2 (43.6, 86.7)		*	36.0 (22.4, 52.3)	47.5 (32.0, 63.4)		*	*	*	
Daily or nearly daily use in past year (83)	*	*	69.6 (49.2, 84.4)		*	33.6 (19.8, 50.9)	49.1 (31.8, 66.6)		*	*	*	

Table 2D.11 (continued)

E-cigarette use status[a] (weighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Alcohol use within guidelines	Alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
Women												
No e-cigarette use in past year (4,718)	29.1 (25.5, 32.9)	32.0 (28.2, 36.0)	39.0 (35.1, 43.0)	.01	27.6 (25.4, 29.9)	44.1 (41.6, 46.5)	28.4 (26.2, 30.6)	.002	40.2 (36.5, 44.1)	44.1 (40.3, 47.9)	15.7 (13.1, 18.7)	.002
Monthly or semi-monthly in past year (107)	*	15.7 (7.3, 30.6)	65.7 (46.9, 80.6)		22.4 (11.5, 39.2)	28.8 (15.6, 46.8)	48.8 (32.6, 65.2)		*	*	*	
Daily or nearly daily use in past year (94)	*	23.3 (10.9, 43.0)	60.1 (40.7, 76.8)		*	27.6 (14.8, 45.5)	60.1 (42.9, 75.1)		*	*	*	

Note. CI = confidence interval.
[a]E-cigarette use is measured within the past year. [b]Alcohol use is measured in the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking ≥4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[65] and NIAAA's definition of heavy alcohol use.[66] [c]*p* value for bivariate association, obtained through a chi-squared test, between e-cigarette and alcohol use categories within each sex and age group.
*Cells with $n < 10$ are suppressed.
Source: 2019–2020 U.S. National Alcohol Survey.

Table 2D.12 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), Overall and by Sex *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall						
Overall (210,684)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (85,223)	29.6 (29.1, 30.1)	29.7 (29.3, 30.2)	25.6 (25.1, 26.0)	11.7 (11.4, 12.0)	3.4 (3.2, 3.6)	<.001
Former (78,251)	3.9 (3.7, 4.1)	32.7 (32.3, 33.2)	34.5 (34.0, 35.0)	18.3 (17.9, 18.7)	10.7 (10.3, 11.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month, (20,406)	6.3 (5.8, 6.8)	24.6 (23.7, 25.6)	17.1 (16.3, 17.8)	32.2 (31.3, 33.1)	19.8 (19.0, 20.6)	
Current (past-30-day) daily smoking, <300 cigarettes/month (5,964)	6.7 (5.8, 7.8)	35.1 (33.3, 37.0)	19.6 (18.1, 21.2)	23.4 (21.9, 24.9)	15.2 (14.0, 16.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (20,840)	5.1 (4.6, 5.6)	39.0 (38.1, 40.0)	18.2 (17.4, 18.9)	19.6 (18.9, 20.3)	18.1 (17.4, 18.9)	
Men						
Overall (97,753)	11.2 (11.0, 11.5)	29.9 (29.5, 30.4)	28.1 (27.7, 28.5)	21.0 (20.7, 21.4)	9.7 (9.4, 10.0)	
Never (34,639)	25.9 (25.3, 26.6)	28.6 (27.9, 29.4)	27.4 (26.7, 28.2)	14.5 (14.0, 15.0)	3.5 (3.2, 3.8)	<.001
Former (38,592)	3.4 (3.1, 3.7)	31.2 (30.5, 31.9)	33.7 (33.0, 34.4)	22.0 (21.4, 22.5)	9.8 (9.4, 10.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (11,312)	5.6 (5.0, 6.3)	22.6 (21.4, 23.8)	16.5 (15.6, 17.6)	36.8 (35.5, 38.1)	18.5 (17.5, 19.5)	
Current (past-30-day) daily smoking, <300 cigarettes/month (2,537)	5.8 (4.6, 7.4)	32.5 (29.9, 35.3)	20.6 (18.3, 23.1)	26.9 (24.6, 29.4)	14.1 (12.4, 16.0)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (10,673)	4.5 (3.9, 5.2)	34.4 (33.1, 35.7)	17.9 (16.8, 19.0)	23.0 (21.9, 24.1)	20.2 (19.2, 21.3)	

Table 2D.12 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Women						
Overall (112,931)	17.0 (16.7, 17.3)	33.2 (32.8, 33.6)	27.6 (27.2, 28.0)	13.3 (13.0, 13.5)	9.0 (8.8, 9.3)	
Never (50,584)	32.2 (31.5, 32.8)	30.5 (29.9, 31.1)	24.3 (23.7, 24.8)	9.7 (9.4, 10.1)	3.3 (3.1, 3.6)	<.001
Former (39,659)	4.4 (4.0, 4.7)	34.4 (33.7, 35.1)	35.4 (34.7, 36.1)	14.3 (13.9, 14.8)	11.6 (11.1, 12.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (9,094)	7.3 (6.5, 8.2)	27.4 (26.0, 28.8)	17.7 (16.6, 18.9)	26.0 (24.8, 27.3)	21.6 (20.4, 22.9)	
Current (past-30-day) daily smoking, <300 cigarettes/month (3,427)	7.5 (6.1, 9.1)	37.2 (34.8, 39.7)	18.8 (16.9, 20.8)	20.5 (18.6, 22.4)	16.1 (14.3, 18.0)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (10,167)	5.8 (5.1, 6.6)	44.4 (43.1, 45.8)	18.5 (17.4, 19.6)	15.6 (14.7, 16.5)	15.7 (14.8, 16.7)	

Note. CI = confidence interval. Data are visualized in Figure 2.6.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never and former alcohol use* are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each sex group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 2D.13 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), Overall and by Sex *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall						
Overall (210,931)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (170,331)	16.5 (16.3, 16.8)	32.4 (32.1, 32.7)	28.2 (27.9, 28.5)	15.1 (14.9, 15.3)	7.8 (7.6, 7.9)	<.001
Former (31,502)	2.4 (2.2, 2.7)	28.2 (27.5, 29.0)	27.5 (26.7, 28.2)	25.7 (25.1, 26.4)	16.2 (15.6, 16.7)	
Current (past-30-day) nondaily use (5,167)	6.8 (5.7, 8.1)	23.0 (21.3, 24.8)	17.2 (15.7, 18.9)	32.2 (30.5, 34.1)	20.8 (19.3, 22.4)	
Current (past-30-day) daily use (3,931)	3.9 (3.0, 5.0)	30.3 (28.3, 32.51)	19.5 (17.7, 21.4)	25.4 (23.6, 27.3)	20.8 (19.1, 22.8)	
Men						
Overall (97,905)	11.3 (11.0, 11.5)	30.0 (29.5, 30.4)	28.1 (27.6, 28.5)	21.1 (20.7, 21.4)	9.7 (9.4, 9.9)	
Never (65,717)	14.8 (14.5, 15.2)	31.1 (30.6, 31.6)	28.9 (28.4, 29.5)	18.3 (17.9, 18.8)	6.8 (6.5, 7.1)	<.001
Former (23,976)	2.2 (1.9, 2.5)	27.6 (26.8, 28.4)	28.0 (27.1, 28.8)	26.9 (26.1, 27.7)	15.4 (14.8, 16.0)	
Current (past-30-day) nondaily use (4,503)	6.1 (5.0, 7.4)	22.3 (20.5, 24.2)	17.2 (15.6, 18.9)	33.4 (31.5, 35.4)	21.0 (19.4, 22.7)	
Current (past-30-day) daily use (3,709)	3.3 (2.5, 4.4)	30.1 (27.9, 32.3)	19.7 (17.9, 21.7)	26.0 (24.2, 28.0)	20.9 (19.1, 22.9)	
Women						
Overall (113,026)	17.0 (16.7, 17.3)	33.2 (32.8, 33.6)	27.5 (27.1, 27.9)	13.3 (13.0, 13.5)	9.0 (8.8, 9.3)	
Never (104,614)	17.7 (17.3, 18.0)	33.3 (32.9, 33.8)	27.7 (27.3, 28.1)	12.9 (12.6, 13.1)	8.4 (8.2, 8.7)	<.001
Former (7,526)	3.7 (3.0, 4.6)	30.9 (29.3, 32.6)	25.1 (23.6, 26.6)	20.6 (19.4, 22.0)	19.6 (18.4, 20.9)	
Current (past-30-day) nondaily use (664)	12.2 (8.4, 17.4)	28.0 (22.7, 33.9)	17.7 (13.4, 23.1)	23.2 (18.8, 28.3)	18.9 (15.3, 23.2)	
Current (past-30-day) daily use (222)	15.2 (8.3, 26.1)	36.2 (26.5, 47.2)	16.2 (9.9, 25.4)	12.9 (8.2, 19.7)	19.5 (11.6, 31.1)	

Note. CI = confidence interval. Data are visualized in Figure 2.7.
[a]*Current smokeless tobacco use* is defined as use in the past 30 days. [b]*Never and former alcohol use* are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories within each sex group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 2D.14 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), Overall and by Sex *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall						
Overall (210,958)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (136,021)	20.5 (20.2, 20.8)	34.6 (34.2, 35.0)	25.6 (25.3, 26.0)	13.2 (13.0, 13.5)	6.0 (5.8, 6.2)	<.001
Former (62,392)	2.1 (1.9, 2.3)	27.0 (26.4, 27.5)	33.2 (32.6, 33.7)	22.9 (22.4, 23.3)	14.9 (14.5, 15.3)	
Current (past-30-day) cigar smoking (12,545)	3.3 (2.8, 3.7)	19.7 (18.6, 20.8)	24.0 (22.8, 25.2)	32.9 (31.7, 34.1)	20.2 (19.2, 21.2)	
Men						
Overall (97,915)	11.3 (11.0, 11.6)	30.0 (29.5, 30.4)	28.1 (27.6, 28.5)	21.1 (20.7, 21.4)	9.7 (9.4, 9.9)	
Never (48,324)	20.8 (20.3, 21.4)	33.8 (33.2, 34.5)	23.7 (23.1, 24.3)	16.3 (15.8, 16.8)	5.3 (5.0, 5.6)	<.001
Former (40,493)	2.2 (1.9, 2.4)	27.6 (27.0, 28.3)	33.4 (32.7, 34.1)	24.0 (23.4, 24.6)	12.8 (12.4, 13.3)	
Current (past-30-day) cigar smoking (9,098)	3.2 (2.7, 3.8)	19.1 (17.9, 20.3)	24.8 (23.4, 26.2)	33.9 (32.5, 35.3)	19.1 (18.0, 20.3)	
Women						
Overall (113,043)	17.0 (16.7, 17.3)	33.2 (32.8, 33.6)	27.5 (27.1, 27.9)	13.3 (13.0, 13.5)	9.0 (8.8, 9.3)	
Never (87,697)	20.3 (19.9, 20.8)	35.0 (34.6, 35.5)	26.7 (26.2, 27.1)	11.5 (11.3, 11.8)	6.4 (6.2, 6.7)	<.001
Former (21,899)	1.9 (1.7, 2.3)	25.3 (24.4, 26.1)	32.6 (31.7, 33.5)	20.0 (19.3, 20.8)	20.2 (19.4, 21.0)	
Current (past-30-day) cigar smoking (3,447)	3.4 (2.6, 4.4)	21.9 (20.0, 24.0)	21.2 (19.3, 23.2)	29.3 (27.3, 31.5)	24.2 (22.2, 26.3)	

Note. CI = confidence interval. Data are visualized in Figure 2.8.
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Never and former alcohol use* are based on lifetime alcohol use and use in the past 30 days. Alcohol use within guidelines, binge, and risky weekly use are based on use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories within each sex group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 2D.15 Average Past-Year Alcohol Consumption (Overall and Beverage-Specific) by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex, Stratified by Age

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Alcohol type				*Alcohol type*				*Alcohol type*			
	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits
	Mean number of drinks consumed in past year (95% CI)											
Overall												
Never (8,918)	206.6 (174.6, 238.5)	83.0 (66.2, 99.7)	48.7 (39.9, 57.4)	76.1 (62.1, 90.1)	199.9 (178.6, 221.2)	80.4 (70.4, 90.3)	56.6 (49.5, 63.6)	63.6 (53.1, 74.1)	131.3 (108.9, 153.8)	45.5 (33.4, 57.6)	50.7 (41.0, 60.5)	35.6 (24.2, 46.9)
Former (2,534)	446.9+ (182.1, 711.7)	160.9 (57.2, 264.5)	63.5 (29.3, 97.6)	222.5 (45.7, 399.4)	261.9** (226.4, 297.4)	102.1* (81.7, 122.4)	69.3+ (55.9, 82.7)	90.9** (71.1, 110.7)	202.5** (175.8, 229.3)	57.3 (44.0, 70.6)	72.2** (59.6, 84.8)	74.6** (57.7, 91.4)
Current (past-year) (2,423)	503.7** (379.1, 628.4)	247.9** (177.6, 318.2)	64.2* (46.8, 81.6)	191.7** (119.3, 264.1)	432.3** (347.6, 516.9)	218.6** (155.9, 281.3)	52.7 (42.5, 62.9)	164.4** (118.5, 210.4)	286.7** (214.0, 359.4)	99.1** (64.3, 133.8)	73.7 (43.6, 103.9)	114.8** (71.1, 158.6)
Nondaily (601)	497.0** (363.7, 630.4)	191.4** (118.6, 264.3)	93.1* (58.5, 127.7)	212.4** (129.1, 295.8)	434.5** (329.0, 539.9)	167.8** (118.7, 217.0)	101.9** (71.0, 132.9)	166.9** (102.0, 231.8)	307.8** (190.9, 424.7)	94.6+ (39.2, 150.0)	79.1 (41.1, 117.1)	134.5** (62.4, 206.7)
Daily (1,822)	507.0** (333.7, 680.4)	275.2** (177.6, 372.8)	50.1 (31.0, 69.3)	181.7* (82.1, 281.3)	431.8** (330.9, 532.7)	230.0** (154.0, 306.0)	41.6* (31.6, 51.7)	163.9** (109.6, 218.2)	283.3** (201.1, 365.5)	99.8** (60.4, 139.1)	72.9 (38.4, 107.3)	111.7** (62.3, 161.1)
Men												
Never (3,326)	264.8 (211.9, 317.7)	125.3 (94.5, 156.0)	46.5 (34.4, 58.6)	95.8 (72.3, 119.3)	286.9 (247.1, 326.6)	131.9 (114.0, 149.7)	61.2 (49.2, 73.1)	94.3 (74.1, 114.6)	227.2 (174.5, 279.9)	97.8 (68.6, 127.0)	69.7 (48.9, 90.5)	60.4 (34.1, 86.8)
Former (1,149)	778.1* (274.6, 1,281.5)	235.4 (33.3, 437.5)	93.7 (35.2, 152.1)	449.0* (111.7, 786.3)	325.1 (267.2, 383.1)	149.6 (114.7, 184.5)	59.2 (41.6, 76.8)	116.4 (82.6, 150.2)	241.6 (201.8, 281.4)	93.4 (69.2, 117.6)	63.9 (49.0, 78.8)	86.1+ (63.0, 109.3)
Current (past-year) (1,133)	566.0** (377.7, 754.3)	316.9** (211.6, 422.2)	47.7 (29.8, 65.5)	201.4* (96.6, 306.3)	590.9** (444.0, 737.9)	313.5** (202.8, 424.3)	47.0 (33.8, 60.3)	233.7** (153.2, 314.2)	364.3** (256.3, 472.2)	141.7* (90.9, 192.4)	75.0 (27.5, 122.5)	149.1** (85.2, 212.9)
Nondaily (311)	570.4** (368.3, 772.6)	238.8* (128.2, 349.5)	78.6+ (40.9, 116.4)	252.9* (119.3, 386.6)	465.7* (329.9, 601.5)	223.8* (145.2, 302.4)	81.8 (47.4, 116.1)	164.1+ (91.5, 236.7)	410.7* (239.1, 582.3)	144.8 (54.0, 235.6)	82.9 (31.2, 134.7)	183.0** (81.7, 284.2)
Daily (822)	563.9** (302.7, 825.1)	354.3** (208.7, 499.9)	32.8 (13.6, 52.0)	176.8 (35.8, 317.7)	623.4** (442.1, 804.7)	336.6** (199.1, 474.2)	37.9* (24.1, 51.7)	251.7** (152.3, 351.1)	355.9* (232.1, 479.6)	141.1+ (83.4, 198.7)	73.5 (18.2, 128.9)	142.9* (69.7, 216.1)

Table 2D.15 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Alcohol type				*Alcohol type*				*Alcohol type*			
	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits
	Mean number of drinks consumed in past year (95% CI)											
Women												
Never (5,591)	153.6 (117.0, 190.1)	45.0 (30.7, 59.4)	50.6 (38.1, 63.1)	58.1 (42.3, 74.0)	123.1 (104.7, 141.4)	34.7 (25.5, 43.9)	52.5 (44.4, 60.6)	36.4 (28.3, 44.5)	72.1 (57.0, 87.1)	13.1 (6.1, 20.0)	39.0 (30.0, 48.0)	20.2 (11.8, 28.6)
Former (1,384)	155.9 (77.4, 234.3)	95.4 (28.1, 162.7)	37.0 (4.5, 69.4)	23.5** (11.8, 35.3)	195.6** (156.3, 235.0)	52.15+ (34.1, 70.2)	79.9 (59.8, 100.1)	64.1** (44.4, 83.7)	156.5** (121.4, 191.6)	16.53 (10.8, 22.3)	81.8** (61.2, 102.3)	58.7** (34.7, 82.8)
Current (past-year) (1,290)	409.7** (280.8, 538.7)	143.6** (69.1, 218.1)	89.1* (55.9, 122.3)	177.0** (87.9, 266.1)	251.4** (189.6, 313.3)	109.3** (69.3, 149.4)	59.1* (43.3, 74.9)	85.3** (52.8, 117.9)	200.9** (105.5, 296.3)	52.0 (5.6, 98.4)	72.34+ (36.7, 107.3)	77.1+ (17.7, 136.5)
Nondaily (290)	388.2** (248.5, 527.9)	121.3* (46.4, 196.2)	114.5* (51.9, 177.1)	152.4** (100.7, 204.1)	387.7** (219.3, 556.1)	84.8** (54.2, 115.3)	131.9** (76.3, 187.5)	171.0* (51.2, 290.8)	160.1 (23.3, 296.8)	21.7 (3.9, 39.6)	73.5 (18.1, 128.9)	65.0 (-32.9, 162.9)
Daily (1,000)	420.4** (240.6, 600.1)	154.6* (50.1, 259.2)	76.6 (38.7, 114.4)	189.1* (58.5, 319.8)	226.1** (160.5, 291.6)	114.0** (66.7, 161.2)	45.5 (30.9, 60.1)	69.2* (38.3, 100.0)	206.4* (99.9, 312.9)	56.0 (3.5, 108.5)	72.2 (32.4, 111.9)	78.7+ (12.6, 144.8)

Note. CI = confidence interval. Data are visualized in Figure 2.9.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime.
+$p < .1$ for association comparing alcohol consumption among tobacco use categories (referent = Never), within each sex and age group. *$p < .05$ for association comparing alcohol consumption among tobacco use categories (referent = Never), within each sex and age group. **$p < .01$ for association comparing alcohol consumption among tobacco use categories (referent = Never), within each sex and age group.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 2D.16 Past-Year Drinking Context by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), Overall and by Sex, Stratified by Age *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Frequency of past-year drinking by context[b]				*Frequency of past-year drinking by context*				*Frequency of past-year drinking by context*			
	Never	Monthly or less	Weekly or more	*p* value[c]	Never	Monthly or less	Weekly or more	*p* value[c]	Never	Monthly or less	Weekly or more	*p* value[c]
	Weighted % (95% CI)											
Overall												
Bar												
Never (8.926)	53.7 (50.4, 57.1)	40.1 (36.8, 43.4)	6.2 (4.7, 8.1)	<.001	53.2 (51.1, 55.4)	42.8 (40.7, 44.9)	4.0 (3.2, 5.0)	.07	76.0 (73.6, 78.2)	22.2 (20.0, 24.5)	1.8 (1.3, 2.5)	<.001
Former (2,537)	39.3 (27.7, 52.2)	55.2 (42.3, 67.5)	*		48.6 (44.2, 53.1)	47.3 (42.9, 51.7)	4.1 (2.7, 6.1)		67.9 (64.2, 71.3)	29.2 (25.9, 32.8)	2.9 (1.9, 4.6)	
Current (past-year) (2,427)	42.1 (35.3, 49.2)	45.5 (38.7, 52.5)	12.4 (8.2, 18.2)		53.9 (50.1, 57.7)	40.4 (36.7, 44.2)	5.7 (4.2, 7.8)		71.5 (65.9, 76.4)	22.7 (18.3, 27.7)	5.9 (3.4, 10.0)	
House parties												
Never (8,926)	49.3 (45.9, 52.6)	48.0 (44.7, 51.4)	2.7 (1.9, 3.9)	<.001	48.5 (46.4, 50.7)	50.2 (48.1, 52.4)	1.2 (0.8, 1.9)	<.001	63.0 (60.3, 65.7)	36.4 (33.7, 39.1)	0.6 (0.3, 1.1)	<.001
Former (2,537)	35.7 (24.9, 48.3)	61.6 (48.9, 72.9)	*		41.4 (37.1, 45.8)	58.2 (53.8, 62.4)	*		52.1 (48.5, 55.8)	47.0 (43.4, 50.7)	0.8 (0.4, 1.6)	
Current (past-year) (2,427)	34.6 (28.3, 41.4)	59.2 (52.2, 65.9)	6.2 (3.5, 10.6)		49.8 (46.0, 53.7)	47.3 (43.5, 51.1)	2.9 (1.8, 4.6)		63.2 (57.5, 68.6)	35.8 (30.5, 41.4)	*	
Home												
Never (8,926)	53.2 (49.8, 56.5)	32.8 (29.8, 36.1)	14.0 (11.7, 16.6)	<.001	46.9 (44.7, 49.0)	34.0 (32.0, 36.0)	19.1 (17.4, 21.0)	<.001	64.3 (61.5, 66.9)	22.1 (19.8, 24.5)	13.7 (11.9, 15.6)	<.001
Former (2,537)	36.4 (25.1, 49.3)	34.1 (23.1, 47.3)	29.5 (18.6, 43.4)		39.5 (35.3, 43.9)	32.7 (28.7, 37.0)	27.8 (24.0, 31.9)		52.7 (49.0, 56.3)	22.5 (19.6, 25.8)	24.8 (21.7, 28.2)	
Current (past-year) (2,427)	32.0 (25.9, 38.8)	38.5 (31.9, 45.5)	29.5 (23.5, 36.4)		44.1 (40.4, 48.0)	23.9 (20.8, 27.3)	32.0 (28.5, 35.7)		56.7 (50.8, 62.4)	19.4 (15.1, 24.6)	23.9 (19.2, 29.3)	
Men												
Bar												
Never (3,330)	54.6 (49.6, 59.5)	37.8 (33.0, 42.7)	7.6 (5.3, 10.9)	.008	47.6 (44.2, 51.0)	46.6 (43.2, 50.0)	5.9 (4.4, 7.8)	.62	68.7 (64.5, 72.6)	27.5 (23.7, 31.5)	3.8 (2.6, 5.6)	.13
Former (1,150)	23.7 (12.3, 40.6)	66.0 (47.5, 80.7)	*		48.9 (42.4, 55.5)	46.3 (39.9, 52.9)	4.8 (2.7, 8.1)		63.5 (58.4, 68.4)	31.8 (27.1, 36.9)	4.7 (2.8, 7.6)	
Current (past-year) (1,135)	44.0 (34.8, 53.7)	42.5 (33.5, 51.9)	13.5 (8.2, 21.4)		50.4 (45.0, 55.9)	42.5 (37.2, 48.0)	7.1 (4.8, 10.2)		65.4 (56.9, 73.1)	26.1 (19.5, 34.0)	8.5 (4.4, 15.8)	

Table 2D.16 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Frequency of past-year drinking by context[b]				*Frequency of past-year drinking by context*				*Frequency of past-year drinking by context*			
	Never	Monthly or less	Weekly or more	*p* value[c]	Never	Monthly or less	Weekly or more	*p* value[c]	Never	Monthly or less	Weekly or more	*p* value[c]
	Weighted % (95% CI)											
House parties												
Never (3,330)	50.6 (45.6, 55.6)	45.0 (40.1, 50.0)	4.4 (2.8, 6.8)	.004	43.9 (40.6, 47.3)	54.2 (50.8, 57.6)	1.9 (1.1, 3.1)	.13	58.5 (54.0, 62.8)	41.0 (36.7, 45.5)	*	.12
Former (1,150)	26.0 (13.5, 44.2)	69.3 (50.3, 83.5)	*		41.8 (35.4, 48.4)	57.7 (51.1, 64.0)	*		51.5 (46.4, 56.6)	47.7 (42.6, 52.8)	*	
Current (past-year) (1,135)	33.9 (25.6, 43.4)	61.4 (51.9, 70.1)	4.7 (2.4, 9.1)		45.6 (40.2, 51.1)	51.3 (45.8, 56.7)	3.1 (1.8, 5.4)		58.1 (49.6, 66.1)	39.9 (32.0, 48.3)	*	
Home												
Never (3,330)	54.4 (49.3, 59.3)	29.2 (24.9, 34.0)	16.4 (12.8, 20.8)	<.001	41.4 (38.1, 44.8)	34.4 (31.2, 37.7)	24.2 (21.3, 27.3)	.11	55.9 (51.3, 60.3)	26.0 (22.2, 30.1)	18.2 (15.1, 21.7)	.10
Former (1,150)	27.1 (14.1, 45.7)	29.5 (15.6, 48.8)	43.3 (24.8, 63.9)		39.5 (33.3, 46.2)	28.2 (22.7, 34.4)	32.3 (26.5, 38.7)		51.8 (46.7, 56.9)	22.3 (18.3, 26.8)	26.0 (21.8, 30.6)	
Current (past-year) (1,135)	35.7 (27.2, 45.1)	34.6 (26.1, 44.2)	29.7 (21.7, 39.2)		41.5 (36.2, 46.9)	30.1 (25.2, 35.5)	28.4 (23.8, 33.6)		51.3 (42.9, 59.8)	22.6 (16.1, 30.6)	26.1 (19.4, 34.1)	
Women												
Bar												
Never (5,595)	53.0 (48.4, 57.5)	42.1 (37.7, 46.7)	4.9 (3.3, 7.3)	.03	58.2 (55.5, 60.8)	2.3 (1.6, 3.3)	39.5 (36.9, 42.2)	.02	80.5 (77.6, 83.1)	18.9 (16.4, 21.8)	0.6 (0.3, 1.1)	<.001
Former (1,386)	52.6 (35.8, 68.9)	45.9 (29.8, 62.9)	*		48.3 (42.5, 54.2)	48.3 (42.4, 54.1)	3.4 (1.9, 6.1)		72.6 (67.5, 77.1)	26.4 (21.9, 31.5)	1.0 (0.4, 2.4)	
Current (past-year) (1,292)	39.2 (29.9, 49.4)	50.1 (40.1, 60.2)	10.6 (5.0, 21.1)		57.9 (52.6, 63.0)	37.9 (33.0, 43.2)	4.2 (2.4, 7.1)		78.1 (71.3, 83.6)	18.9 (13.8, 25.3)	*	
House parties												
Never (5,595)	48.1 (43.6, 52.6)	50.8 (46.2, 55.3)	1.2 (0.7, 2.1)	<.001	52.6 (49.9, 55.3)	46.7 (44.0, 49.4)	0.7 (0.4, 1.4)	<.001	65.9 (62.4, 69.2)	33.5 (30.2, 37.0)	*	<.001
Former (1,386)	44.1 (28.5, 61.0)	55.0 (38.2, 70.8)	*		41.0 (35.4, 46.7)	58.7 (52.9, 64.2)	*		53.1 (47.9, 58.3)	46.0 (40.8, 51.2)	*	
Current (past-year) (1,292)	35.6 (26.8, 45.5)	56.0 (45.7, 65.7)	8.4 (3.6, 18.5)		54.6 (49.3, 59.9)	42.8 (37.6, 48.0)	2.6 (1.1, 5.9)		68.8 (61.2, 75.5)	31.2 (24.5, 38.8)	*	

Table 2D.16 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Frequency of past-year drinking by context[b]				*Frequency of past-year drinking by context*				*Frequency of past-year drinking by context*			
	Never	Monthly or less	Weekly or more		Never	Monthly or less	Weekly or more		Never	Monthly or less	Weekly or more	
Home												
Never (5,595)	52.1 (47.5, 56.6)	36.1 (31.9, 40.6)	11.8 (9.2, 15.0)	<.001	51.7 (49.0, 54.4)	33.6 (31.2, 36.2)	14.7 (12.8, 16.8)	.001	69.4 (66.0, 72.7)	19.7 (16.9, 22.7)	10.9 (8.9, 13.2)	<.001
Former (1,386)	44.3 (28.4, 61.5)	38.1 (23.0, 55.9)	17.6 (8.7, 32.4)		39.5 (33.9, 45.3)	37.4 (31.9, 43.3)	23.1 (18.5, 28.4)		54.0 (48.7, 59.1)	23.0 (18.7, 27.8)	23.1 (18.6, 28.2)	
Current (past-year) (1,292)	26.4 (18.6, 36.0)	44.4 (34.6, 54.6)	29.2 (20.7, 39.6)		47.2 (41.9, 52.5)	34.1 (29.3,39.3)	18.7 (14.9, 23.2)		62.5 (54.5, 69.9)	16.0 (11.0, 22.8)	21.5 (15.5, 28.9)	

Note. CI = confidence interval. Data are visualized in Figure 2.10.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]Drinking context is measured within the past year. [c]*p* value for bivariate association, obtained through a chi-squared test, between tobacco and alcohol use categories within each sex and age group.
*Cells with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

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Tobacco and Alcohol: Understanding Co-Use to Reduce Cancer Incidence and Mortality

Chapter 3
Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Population Subgroups in the United States

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Chapter 3
Surveillance and Behavioral Epidemiology of Tobacco and Alcohol Use Among Population Subgroups in the United States

Introduction

Tobacco and alcohol are among the most widely used addictive and carcinogenic substances that, separately and together, have large impacts on the U.S. and global burden of disease.[1,2] Epidemiological surveillance of tobacco and alcohol co-use in specific population subgroups is critical for many public health goals. First, it can document disparities in health-related behaviors and associated morbidity and mortality, pointing to demographic groups in need of further attention. Second, it can help generate ideas about the factors influencing use and co-use of tobacco and alcohol. Third, it allows the detection of emerging trends over time in response to policy and societal changes.

This chapter, a companion to chapter 2, provides a summary of tobacco and alcohol co-use in specific population subgroups of adults based on a review of the literature and new analyses of National Alcohol Surveys (NAS) and National Survey on Drug Use and Health (NSDUH) data. The chapter reviews tobacco and alcohol co-use among groups by race and/or ethnicity, educational attainment, income, geographic location, sexual minority status, veteran status, and presence of comorbidities including cancer, chronic health conditions, HIV, mental health conditions, and pregnancy. Information about tobacco and alcohol co-use by adolescents in 8th 10th, and 12th grade is provided in chapter 2; however, co-use by other adolescent population subgroups is limited by small sample sizes in national surveillance surveys. This chapter focuses on differences between demographic groups. It is important to note that each demographic group includes a wide range of tobacco and alcohol use characteristics. Future studies could further examine within group variation in tobacco and alcohol use and co-use. Further, when this volume refers to smoking or tobacco use, it refers to the use of commercial tobacco and not the traditional use of tobacco by some American Indian and Alaksa Native communities.

Methods

To identify studies that estimated the prevalence of tobacco and alcohol co-use in the U.S. population overall, targeted keyword searches using combinations of tobacco and alcohol terms were conducted using Google Scholar, PubMed, Ovid, and Scopus. Literature searches were also conducted for prospective alcohol mortality and morbidity studies because these studies have often examined both tobacco and alcohol. Search terms are listed in Appendix 3A.

The chapter highlights findings from nationally representative studies; findings from non-nationally representative studies are also included to provide information about specific subgroups. In addition, the 2014–2015 NAS and the 2015–2019 NSDUH were analyzed to better understand tobacco and alcohol co-use patterns among adults. These surveys were chosen

because they are nationally representative, have large sample sizes, and include detailed tobacco and alcohol measures or subgroup data not included in other nationally representative surveys. A detailed description of the NAS and NSDUH measures and analytic approaches are described in chapter 2. The NAS analyses focus on past-year tobacco product (never, former, daily, and nondaily use) and past-year alcohol use, defined as no past-year use, use within guidelines (≤7/14 drinks per week for women/men or <4/5 drinks per day for women/men), and use exceeding guidelines (>7/14 drinks per week for women/men or ≥4/5 drinks per week for women/men). The NSDUH analyses focus on past-month use of cigarettes, smokeless tobacco, and cigars, and past-month alcohol use (never, former, use within guidelines, binge drinking [drinking ≥4/5 drinks on an occasion for women/men] without risky weekly use [>7/14 drinks per week for women/men] in past 30 days, and risky weekly use [>7/14 drinks per week for women/men] in past 30 days [with or without binge drinking]).

Within and Between Group Heterogeneity in Tobacco and Alcohol Use

Discussion and analysis of heterogenous populations require attention to several issues related to aggregated data. First, U.S. surveillance data often include aggregate data from multiple distinct demographic groups who may have different distributions of behaviors and health outcomes. For example, although Asian populations in the United States may have lower prevalence of tobacco and alcohol use overall, differences by country of origin, sex, or other demographic variables could conceal significant variation in prevalence within this group.[3,4] Second, within demographic groups with low levels of substance use or co-use, there are still people who use tobacco and/or alcohol and experience disproportionate harm. For example, lifetime abstinence from alcohol use is higher among American Indian and Alaska Native (AI/AN) populations than other demographic groups in the U.S. population.[5-8] However, mortality from alcohol-related outcomes such as cirrhosis is higher for AI/AN populations than for other demographic groups on average.[9-12] With respect to tobacco use, AI/AN populations report some of the highest prevalences of lifetime and current smoking among demographic groups, and they also experience greater mortality from lung cancer compared to other demographic groups.[4] Such observations further support the need to address within-group heterogeneity in tobacco and alcohol use even when overall prevalence is low. Much of the research in this chapter is based on federal health surveys, thus addressing heterogeneity in prevalence of co-use of tobacco and alcohol is limited by extant survey categories. Smaller studies or regional surveys designed to capture specific populations, such as the California Health Interview Survey,[13] can help address these challenges.

Differences in prevalence of tobacco and alcohol co-use in a specific demographic group may reflect differences in prevalence of use of one of the substances, resulting in higher prevalence of co-use. For example, a population might have 10% of people who use tobacco and 10% of people who consume alcohol. In this case 100% co-use would result in 10% of the population using both substances. On the other hand, if 25% of the population reports tobacco use and 75% report alcohol use, a weaker association between tobacco and alcohol use would still result in a greater prevalence of people reporting co-use of tobacco and alcohol. This monograph focuses on the prevalence of alcohol use among people who use tobacco, rather than the strength of the association between tobacco and alcohol use, but further exploration of such associations could provide insight into biological and environmental factors influencing co-use.

Co-Use by Race and/or Ethnicity

The 2024 Surgeon General's report, *Eliminating Tobacco-Related Disease and Death: Addressing Disparities*, concluded that "racial and ethnic disparities in cigarette smoking have persisted since 2000, with prevalence remaining highest among American Indian and Alaksa Native adults."[4,p.10] The report further noted that disparities in tobacco use "vary by ethnic group, nativity, and acculturation in the United States."[4] In the 2024 National Health Interview Survey (NHIS), cigarette smoking prevalence was highest among AI/AN (15.8%), Black (10.6%) and White (10.7%) adults, and lowest among Asian adults (3.8%).[14] In the 2024 NSDUH, past-30-day alcohol use prevalence was highest among White adults (54.5%) and those of 2 or more races (51.4%) and lowest among Asian adults (35.5%).[7] As described in this section, studies show differences in tobacco and alcohol co-use by race, ethnicity, and country of origin, with higher prevalence of co-use among White, Black, and AI/AN individuals compared to other groups and higher prevalence of co-use among U.S.-born compared to non-U.S.-born Hispanic individuals.[15-20]

Adults. In the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) 2001–2002 study,[17] prevalence of past-year tobacco and alcohol co-use varied by race and/or ethnicity. The group with the highest past-year co-use prevalence was AI/AN individuals (34.0% among men; 22.8% among women). The group with the lowest prevalence of co-use was Asian/Native Hawaiian/Pacific Islander individuals (18.1% among men; 6.6% among women). This study also included the prevalence of comorbidity of alcohol use disorder (AUD) and nicotine dependence. Again, the AI/AN sample had the highest prevalence of comorbidity (7.0% among men; 4.0% among women). The lowest prevalence for comorbidity was seen in the Hispanic population (2.2% among men; 0.9% among women).[17]

In a study analyzing NSDUH data from 2002 to 2016, cigarette smoking prevalence in 2016 among people with past-year AUDs was 37.8%, and ranged from 28.2% among Hispanic participants to 50.5% among non-Hispanic Black participants.[20] Among people without AUDs, cigarette smoking prevalence in 2016 was 16.1% and ranged from approximately 10.9% among Hispanic participants to 18.2% among White participants. Cigarette smoking declined significantly from 2002 to 2016 among participants with and without AUDs in all racial and/or ethnic groups except for non-Hispanic Black participants with AUDs. Smoking prevalence was consistently highest among people with severe AUDs. For example, in 2016, smoking prevalence was 50.5% among non-Hispanic White individuals with severe AUD, and 75.5% among non-Hispanic Black individuals with severe AUD.[20]

Another study used 2010–2019 NSDUH data to analyze tobacco use and nicotine dependence among people with no alcohol use, "low-risk" alcohol use (≤7/14 drinks per week for women/men), and "high-risk" alcohol use (>7/14 drinks per week for women/men).[18] Compared to non-Hispanic White individuals with high-risk alcohol use, Hispanic individuals with high-risk alcohol use had decreased odds of past-month tobacco use (adjusted odds ratio [aOR] = 0.58, 95% confidence interval [CI] = 0.51–0.67) and nicotine dependence (aOR = 0.33, 95% CI = 0.28–0.40), while Black individuals with high-risk alcohol use had increased odds of past-month tobacco and cannabis use (aOR = 1.19, 95% CI = 1.03–1.38).[18]

Adolescents. A study examining race and/or ethnic differences in substance use among 10th grade adolescents in Alabama, California, and Texas in 2009–2011 reported that the prevalence of cigarette and alcohol co-use (exclusively or with cannabis) was 8% for Black adolescents, 6% for Hispanic adolescents, and 12% for White adolescents.[19] The authors also found that White adolescents were more likely than Hispanic adolescents to report exclusive cigarette smoking and co-use of cigarette and alcohol. White adolescents were also more likely than Black adolescents to report cigarette and alcohol co-use and polysubstance use (concurrent use of cannabis, alcohol, and cigarettes).[19] Analyzing data from the 2011–2014 NSDUH, Banks and colleagues (2017)[16] found that among adolescents ages 12–18, alcohol and cigarette co-use was less prevalent among African American, Asian, and Hispanic adolescents compared to White adolescents, but found no statistically significant differences between White and Native American adolescents. Additionally, a study analyzed pooled 2011–2014 NSUDH data for African American adolescents and young adults (ages 12–25; $N = 11{,}124$) to understand the relationship between use of blunts (little cigars or cigarillos filled with cannabis) and alcohol.[21] Among those who consumed alcohol in the past year, more than one-third (36.8%) also reported blunt use. Additionally, the study found increased odds of alcohol dependence among adolescents who co-used blunts and alcohol, compared with those who exclusively used alcohol.[21]

Hispanic/Latino/a Adults. A study examined current cigarette smoking and binge drinking in a diverse sample of 16,415 Hispanic adults (ages 18–74 years) residing in 4 U.S. cities between 2008 and 2011.[15] Among men, co-use (i.e., current smoking and binge drinking) declined with age from 17.1% among 18–24-year-olds to 2.2% for those 65 and older. Similarly, among women, co-use declined with age from 7.0% for 18–24-year-olds to 0.3% for those 65 and older. U.S. born Hispanic women had a greater likelihood of co-use then foreign-born women (aOR = 1.8, 95% CI = 1.1–3.0). In contrast, among men, the likelihood of co-use did not differ significantly by birthplace. Significant differences in co-use were also found based on background country, education, marital status, and other variables.[15]

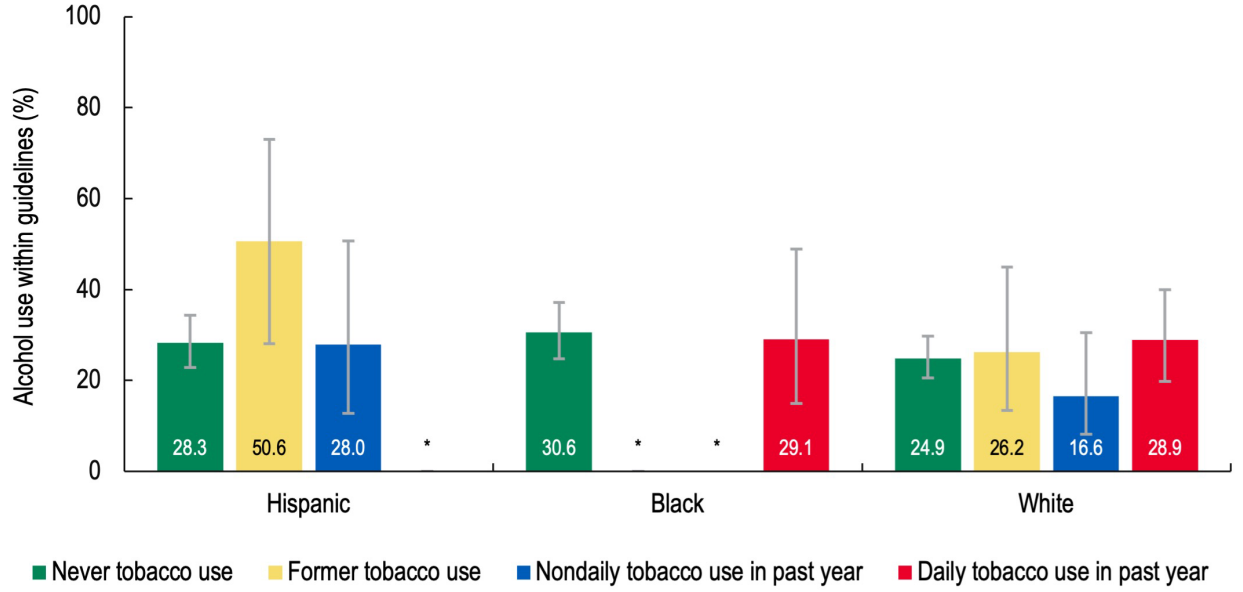
National Alcohol Surveys Analysis: Co-Use by Race and/or Ethnicity

Figure 3.1 (Appendix Table 3B.1) presents data from NAS on the proportions of adults who drank within and exceeded alcohol use guidelines for Black, White, and Hispanic populations by age group and tobacco use category. Across age groups and race and/or ethnicity, prevalence of alcohol use exceeding guidelines was generally highest among adults who used tobacco either daily or nondaily. The bivariate association between tobacco and alcohol use was statistically significant among all subgroups at the $p < .05$ level except among White participants ages 18–29 ($p = .08$). Among those who reported any past-year tobacco use, either daily or nondaily, more than one-half of those ages 18–29 and more than 35% of those ages 30–59 reported alcohol use exceeding guidelines across racial and/or ethnic categories. Prevalence of alcohol use exceeding guidelines among those ages 18–29 who reported daily tobacco use was 77.1% (95% CI, 54.1%–90.6%) among Hispanic participants, 56.0% (95% CI, 35.8%–74.4%) among Black participants, and 52.7% (95% CI, 38.1%–64.2%) among White participants. Prevalence of alcohol use exceeding guidelines among those ages 18–29 who reported nondaily tobacco use was 61.6% (95% CI, 40.1%–79.3%) among Hispanic participants, 60.8% (95% CI, 33.8%–82.5%) among

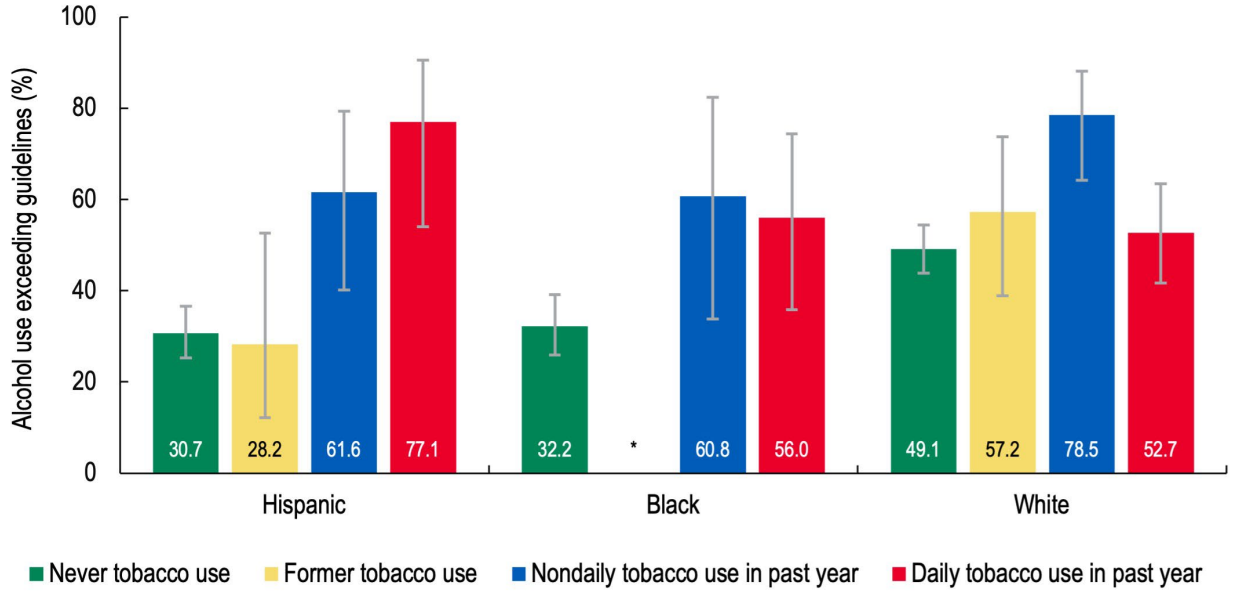
Black participants, and 78.5% (95% CI, 64.3%–88.1%) among White participants. Levels of no past-year alcohol use generally increased with age. Those reporting never tobacco use or former tobacco use were the most likely to report not using alcohol in the past year, compared to those who used tobacco (Appendix Table 3B.1). Among people ages 18–29, across race and/or ethnicity categories, most people who used tobacco (daily or nondaily) in the past year reported past-year alcohol use, and alcohol use exceeding guidelines was more common than alcohol use within guidelines. The prevalence of no past-year alcohol use among those ages 18–29 who used tobacco was only 6.8% (95% CI, 3.5%–13.1%) among Hispanic participants, 17.5% (95% CI, 8.5%–32.6%) among Black participants, and 14.6% (95% CI, 9.3%–22.1%) among White participants.

Figure 3.1 Past-Year Alcohol Use by Age, Past-Year Tobacco Use Status, and Race and/or Ethnicity (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

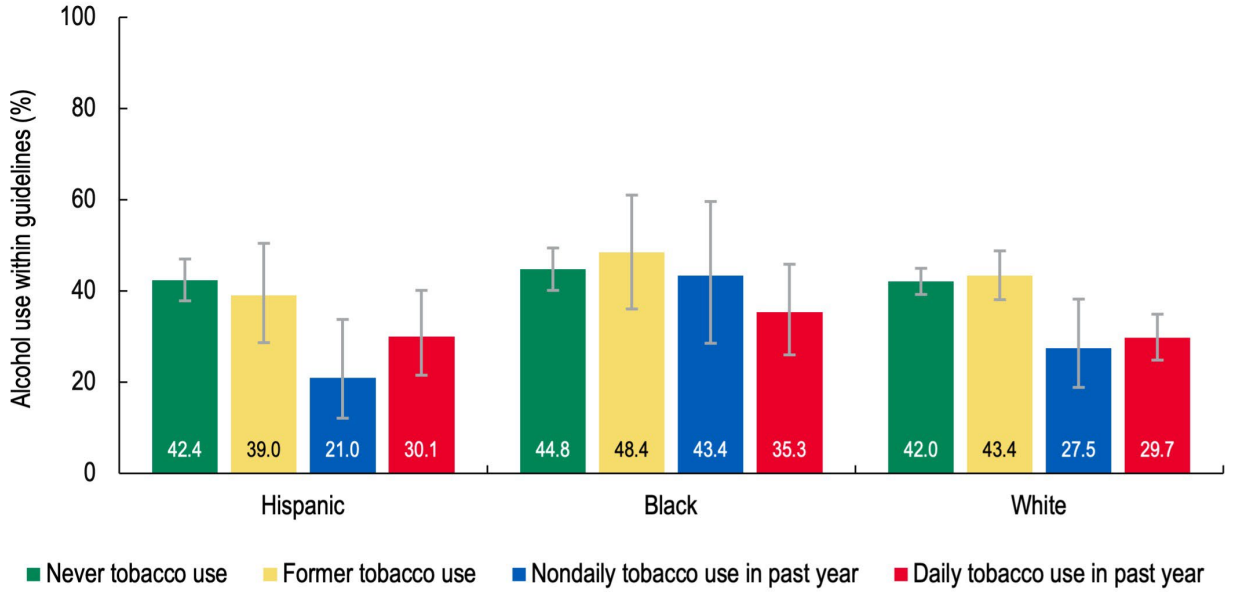
Panel A: Alcohol Use Within Guidelines, Ages 18–29



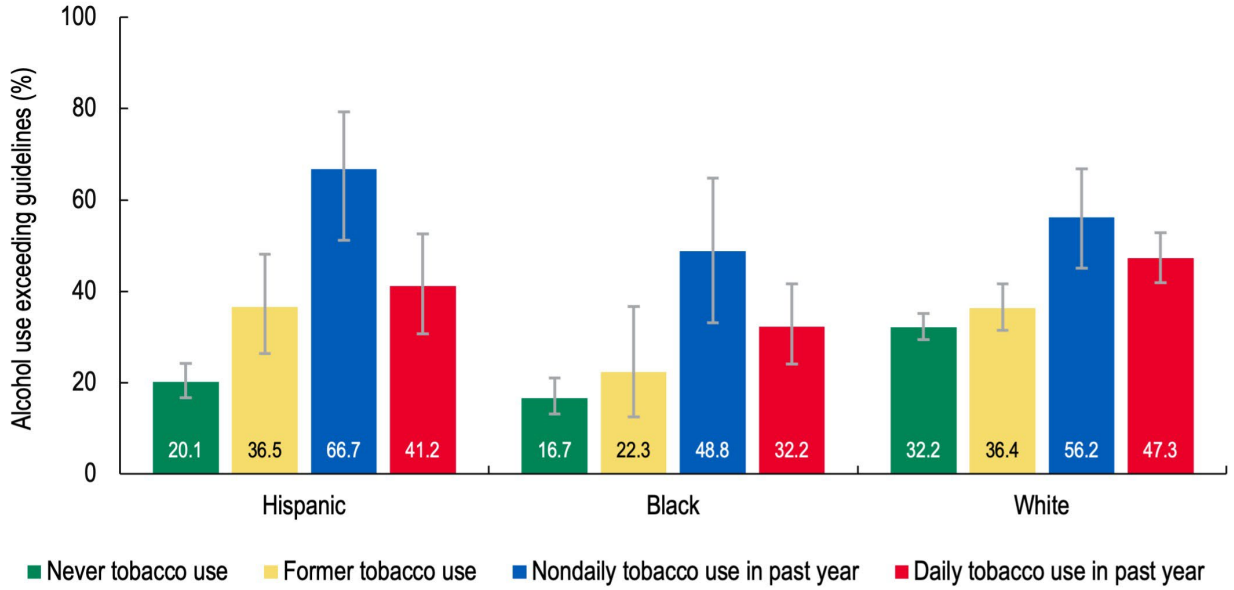
Panel B: Alcohol Use Exceeding Guidelines, Ages 18–29



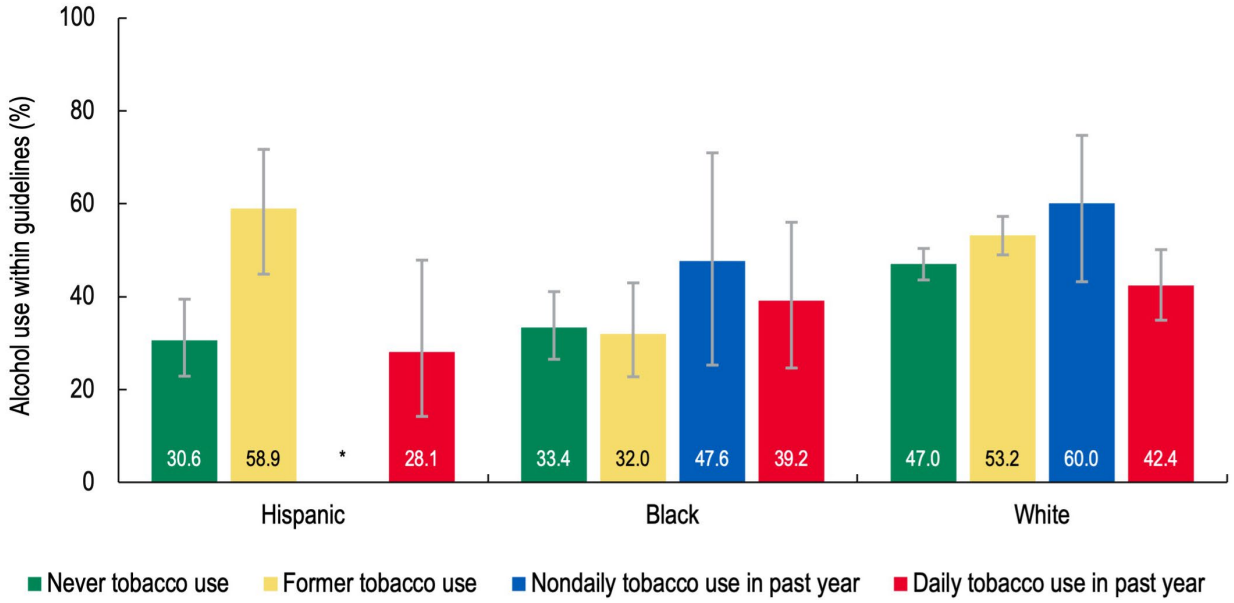
Panel C: Alcohol Use Within Guidelines, Ages 30–59



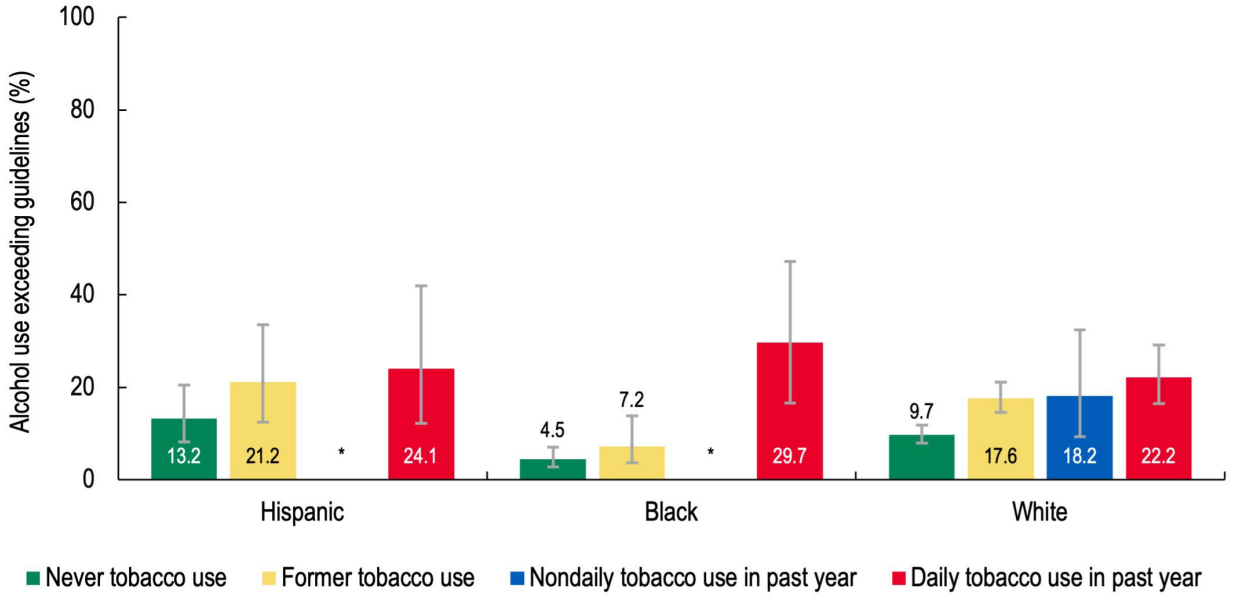
Panel D: Alcohol Use Exceeding Guidelines, Ages 30–59



Panel E: Alcohol Use Within Guidelines, Ages 60 and Older



Panel F: Alcohol Use Exceeding Guidelines, Ages 60 and Older



Note. For full data, see Appendix Table 3B.1. Black and White participants are non-Hispanic. *Tobacco use* is defined as use of non-e-cigarette tobacco products (due to survey question wording). *Nondaily* and *daily current tobacco* use are defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured within the past year. No past-year alcohol use is not shown in the charts. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking at least 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[22] and NIAAA's definition of heavy alcohol use.[23]
*Categories with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

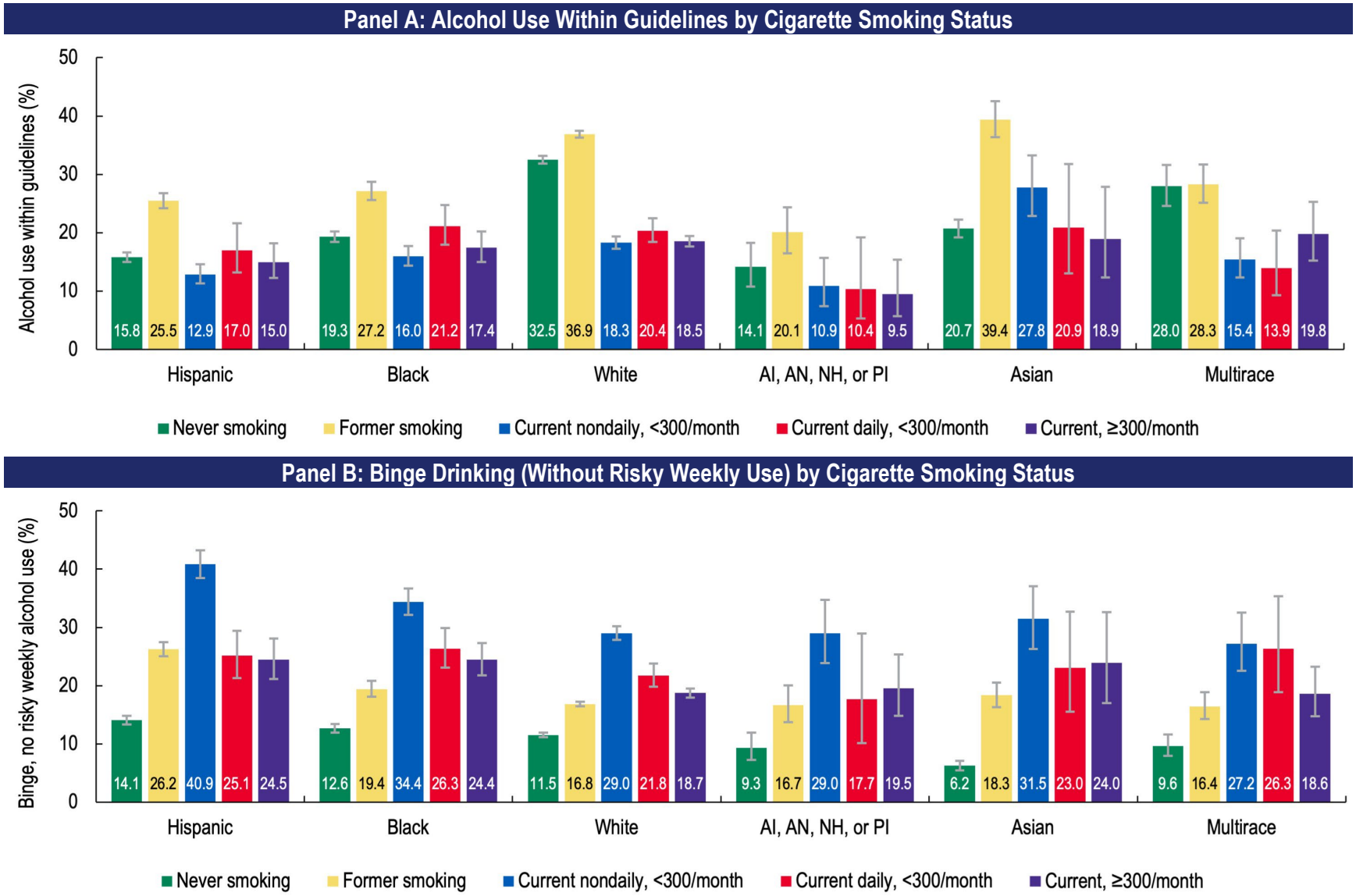
Tobacco use was also associated with higher prevalence of reporting having had 5+ drinks in a day in the past year, as well as reporting high intensity drinking (HID) (≥8/12 drinks in a day for women/men) in the past year. As shown in Appendix Table 3B.2, past-year nondaily and daily tobacco use are associated with a higher prevalence of 5+ drinking and HID compared to never tobacco use among those ages 18–59. Few people (<20%) ages 60 and older engaged in either 5+ drinking or HID. Prevalence of 5+ drinking among those ages 18–29 who used tobacco was 60.8% (95% CI, 45.4%–74.4%) among Hispanic participants, 46.6% (95% CI, 30.5–63.5%) among Black participants, and 54.1% (95% CI, 44.9%–63.0%) among White participants. There was a statistically significant association between tobacco use and HID or 5+ drinking among all groups at the $p < .05$ level except HID among Black participants ages 30–59 ($p = .27$) and ages 60 and older ($p = .68$).

National Survey on Drug Use and Health Analysis: Co-Use by Race and/or Ethnicity

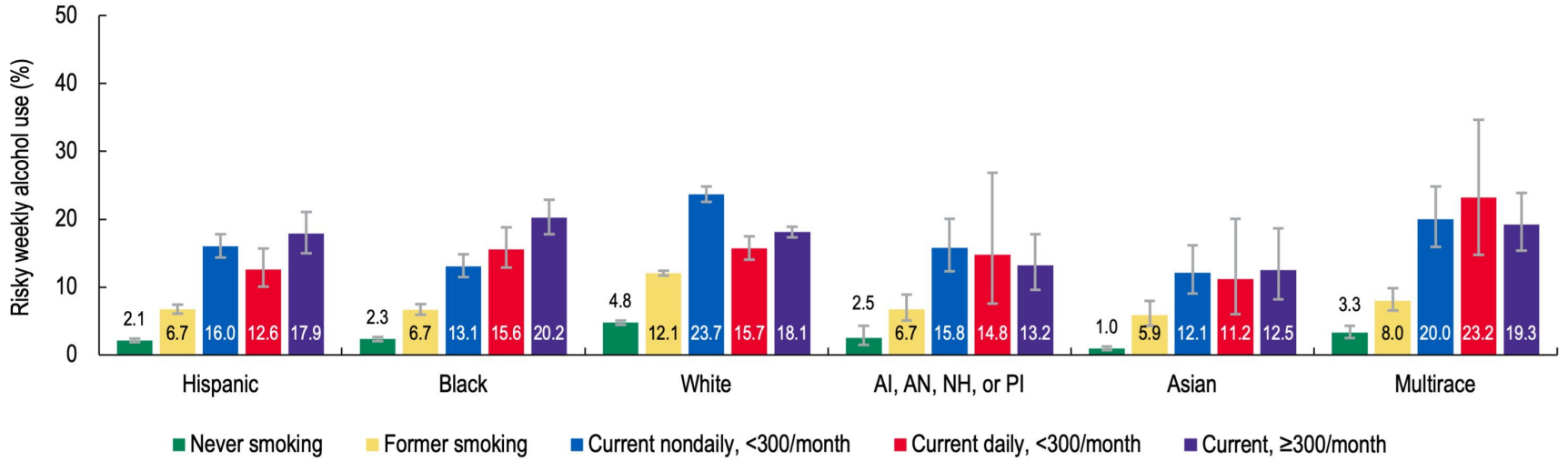
Estimates of alcohol use by cigarette smoking status from the 2015–2019 NSDUH are presented by race and/or ethnicity in Figure 3.2. Similar to the NAS results, there was a significant bivariate association between cigarette smoking and alcohol use across all racial and/or ethnic groups ($p < .001$). Those who reported never cigarette smoking were least likely to report alcohol use exceeding guidelines (binge drinking and risky weekly use). Further, those who formerly smoked had the highest prevalence of alcohol use within guidelines compared to other tobacco use categories. In general, those who smoked nondaily had the highest prevalence of alcohol use

exceeding guidelines compared to the other tobacco use categories. In particular, Hispanic, Black, and White participants who reported nondaily smoking were significantly more likely to report past month binge drinking than participants in any other tobacco use category, and White participants who reported nondaily smoking were also significantly more likely to report risky weekly alcohol use over the past month than White participants in any other tobacco use category. Prevalence of binge drinking (without risky weekly alcohol use) among those who smoked nondaily <300 cigarettes/month was 40.9% (95% CI, 38.5%–43.3%) among Hispanic participants; 34.4% (95% CI, 32.1%–36.7%) among Black participants; 29.0% (95% CI, 27.9%–30.2%) among White participants; 29.0% (95% CI, 23.9%–34.7%) among American Indian, Alaska Native, Native Hawaiian, or other Pacific Islander participants; 31.5% (95% CI, 26.3%–37.1%) among Asian participants; and 27.2% (95% CI, 22.5%–32.5%) among multiracial participants. Prevalence of risky weekly alcohol use among those who smoked nondaily was 16.0% (95% CI, 14.4%–17.8%) among Hispanic participants; 13.1% (95% CI, 11.5%–14.9%) among Black participants; 23.7% (95% CI, 22.6%–24.8%) among White participants; 15.8% (95% CI, 12.3%–20.1%) among American Indian, Alaska Native, Native Hawaiian, or other Pacific Islander participants; 12.1% (95% CI, 9.0%–16.1%) among Asian participants; and 20.0% (95% CI, 15.9%–24.8%) among multiracial participants. Overall, White and multiracial individuals were the least likely to report never use of alcohol (White: 9.1%, 95% CI = 8.9%–9.3%; multiracial: 11.2%, 95% CI = 9.8%–12.8%). Conversely, Asian individuals were the most likely to report never use of alcohol (33.7%, 95% CI = 32.2%–35.2%) (Appendix Table 3B.3). When considering smokeless tobacco use and cigar smoking, prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) was generally highest among individuals who reported past-30-day use of cigars or smokeless tobacco (daily or nondaily use), regardless of race and/or ethnicity (Appendix Tables 3B.4 and 3B.5).

Figure 3.2 Past-30-Day Alcohol Use by Race and/or Ethnicity and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



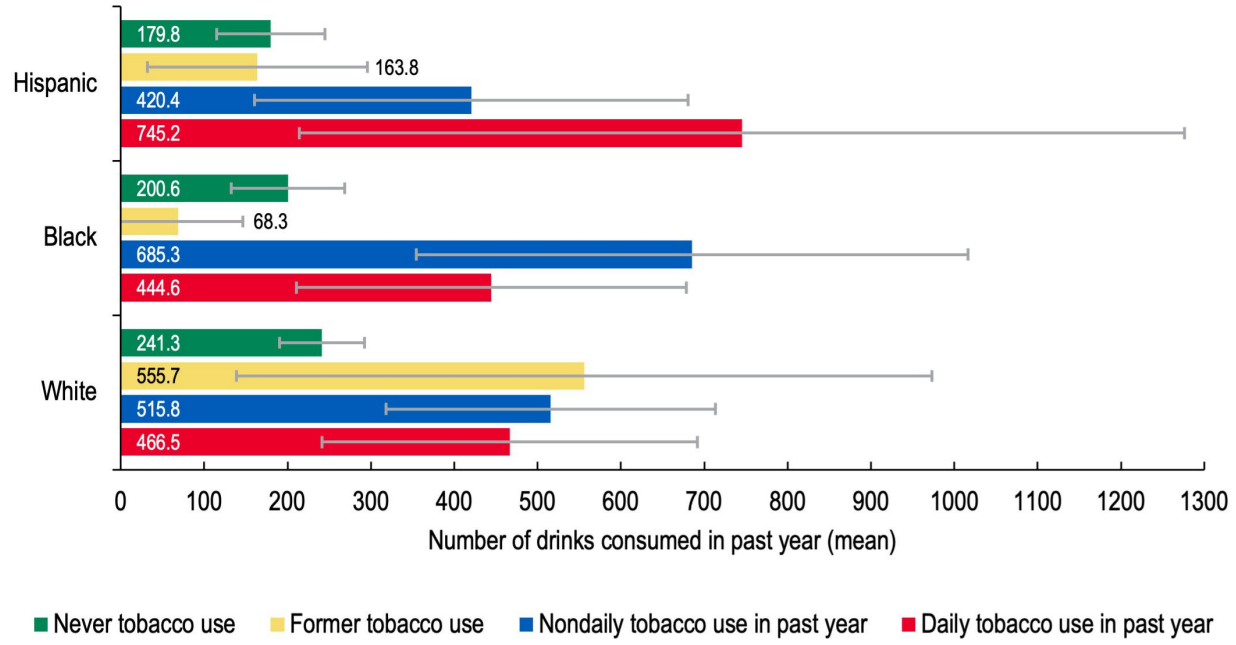
Note. AI = American Indian; AN = Alaska Native; NH = Native Hawaiian; PI = Pacific Islander. For full data, see Appendix Table 3B.3. Categories other than Hispanic are non-Hispanic. *Multirace* is defined as two or more races. AI, AN, NH, and PI participants were combined due to small sample sizes and because they are indigenous populations in the United States that have similar drinking patterns. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

National Alcohol Surveys Analysis: Past-Year Volume of Consumption by Race and/or Ethnicity

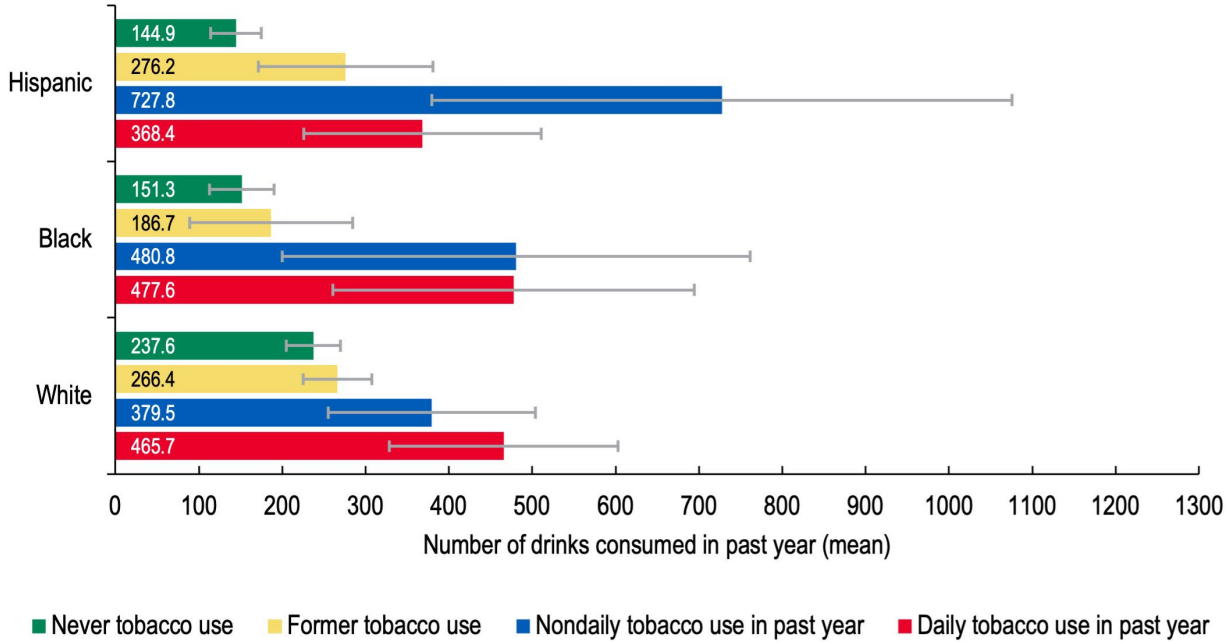
The association between volume of alcohol consumption, tobacco use, and race and/or ethnicity are shown in Figure 3.3. Across racial and/or ethnic groups, those with past-year tobacco use had higher overall alcohol consumption (mean number of drinks consumed in past year) than those who never used tobacco. For example, as shown in Appendix Table 3B.6, among those ages 18–29, the average number of drinks consumed in the past year among those who also used tobacco in the past year and who never used tobacco was 579.3 (95% CI, 291.3–867.3) versus 179.8 (95% CI, 114.7–244.8) among Hispanic individuals; 522.2 (95% CI, 328.4–716.0) versus 200.6 (95% CI, 132.7–268.5) among Black individuals; and 480.4 (95% CI, 309.9–651.0) versus 241.3 (95% CI, 190.1–292.4) among White individuals. Among Hispanic individuals, associations between past-year tobacco use and overall alcohol consumption (compared to never tobacco use) were statistically significant among those younger than 60 ($p < .01$) but not among those 60 and older. In contrast, among Black and White individuals, past-year tobacco use was significantly associated with overall alcohol consumption (compared to never tobacco use; $p < .01$) across all age groups. Figure 3.3 also includes data for daily and nondaily tobacco use. Nondaily tobacco use was associated with greater numbers of drinks consumed in the past year compared to never or former tobacco use, particularly among Hispanic adults ages 30–59, Black adults ages 18–59, and White adults of all ages. These results are consistent with the extensive evidence for strong associations between tobacco and alcohol use and that these associations are most apparent for people who use more tobacco and alcohol.

Figure 3.3 Average Number of Drinks Consumed in Past Year by Age, Past-Year Tobacco Use Status, and Race and/or Ethnicity (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

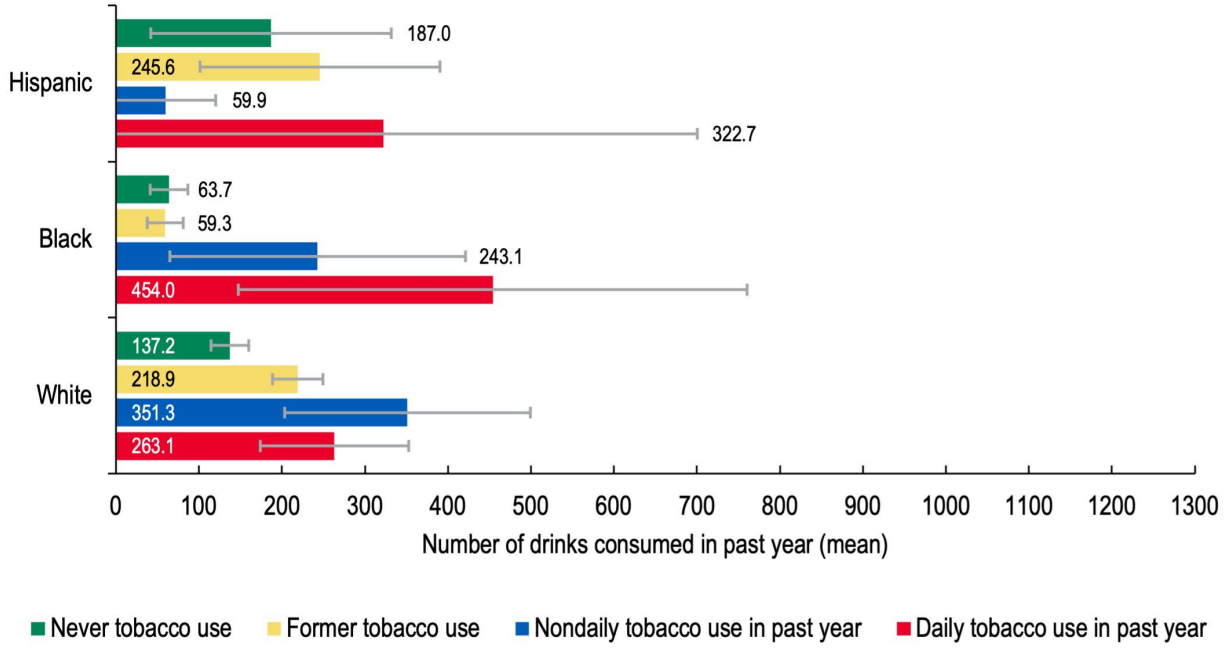
Panel A: Average Number of Drinks Consumed in Past Year, Ages 18–29



Panel B: Average Number of Drinks Consumed in Past Year, Ages 30–59



Panel C: Average Number of Drinks Consumed in Past Year, Ages 60 and older



Note. For full data, see Appendix Table 3B.6. Black and White participants are non-Hispanic. *Tobacco use* is defined as use of non-e-cigarette tobacco products (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured in the past year. Some confidence intervals contain negative values (not displayed).
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

National Alcohol Survey Analysis: Lifetime Cigarette Pack Years and Heavy Alcohol Use by Race and/or Ethnicity

Lifetime accumulated co-use of pack years of cigarettes and 5+ drinking days is presented in Appendix Table 3B.7. In general, across race and/or ethnicity, people who never smoked were the most likely to report zero lifetime 5+ drinking days. For example, among participants ages 30–59 with ≥40 pack years, 12.3% of Hispanic individuals (95% CI, 4.3%–30.5%), 39.8% of Black individuals (95% CI, 18.7%–65.5%) and 19.2% of White individuals (95% CI, 12.3%–28.8%) reported zero lifetime 5+ drinking days. In contrast, among those with zero pack years in this same age group, zero lifetime 5+ drinking days was reported by 58.0% of Hispanic participants (95% CI, 50.1%–65.5%), 70.6% of Black individuals (95% CI, 62.4%–77.6%), and 34.8% of White individuals (95% CI, 30.4%–39.5%).There was a statistically significant association between pack years and lifetime 5+ drinking days among all groups ($p < .001$) except Hispanic and Black participants ages 18–29. Small sample sizes precluded reporting some estimates, especially for people who reported high levels of smoking, although there were 41 people ages 18–29 who reported 40+ pack years of cigarettes. This is a very high level of tobacco use for this age group, and it is possible that respondents inaccurately reported their smoking history. Estimates of lifetime accumulated exposure are important because of the synergistic increase in risk associated with co-use of tobacco and alcohol and the high-risk ratios for head and neck cancers associated with higher levels of consumption of both substances (see chapter 4).

Summary: Co-Use by Race and/or Ethnicity

The literature shows an association between tobacco and alcohol use among racial and/or ethnic subgroups. In general, across racial and/or ethnic groups, those who reported never tobacco use (NAS) or never cigarette smoking (NSDUH) had the lowest levels of hazardous drinking (exceeding guidelines, binge drinking, or risky weekly alcohol use) and consumed fewer drinks per year. People who reported nondaily cigarette smoking in the NSDUH had the highest prevalence of binge drinking and risky weekly alcohol use compared to other cigarette smoking categories, particularly among Hispanic, Black, and White participants. In NAS, prevalence of no alcohol use increased with age across racial and/or ethnic groups.

Box 3.1. Tobacco and Alcohol Use Among American Indian and Alaska Native Individuals

One goal of this monograph is to examine whether addressing alcohol use can contribute to tobacco control for cancer prevention. It is imperative, then, to consider population groups that engage in greater use and co-use of both substances or are more susceptible to the effects of co-use and, therefore, may be especially important when preventing tobacco initiation and improving tobacco cessation.

In the United States, AI/AN adults and youth have the highest prevalence of cigarette smoking, with 15.8% of AI/AN adults reporting current cigarette smoking in 2024 (compared to the overall adult prevalence of 9.9%%).[14] Even while overall tobacco product use among middle and high school students in the U.S. declined from 2023 to 2024, Jamal and colleagues (2024)[24] found that tobacco use among AI/AN youth increased.

Whenever describing and working to address the disproportionately high prevalence of tobacco use in AI/AN populations, it is critical to differentiate between the use of traditional tobacco and commercial tobacco products to effectively and respectfully address the health effects of tobacco. Traditional tobacco refers to tobacco used by some AI/AN communities for ceremonial or medicinal purposes.[25,26] Commercial tobacco is tobacco sold for recreational use and includes cigarettes, e-cigarettes, smokeless tobacco, pipe tobacco, cigars, hookahs, and other products.[4]

With regard to alcohol use, according to the 2024 NSDUH, 404,000 AI/AN people ages 12 and older (29.7%) reported that they drank alcohol on at least 1 day in the past month.[7,8] AI/AN adults report some of the highest prevalence of lifetime alcohol abstinence among demographic groups in the United States,[27] but other studies in the literature have also described heavy episodic drinking reported by AI/AN adults and adolescents.[5,28] AI/AN adults also have higher rates of morbidity and mortality caused by alcohol compared with non-AI/AN people.[11,29]

Although there are few studies focusing on tobacco and alcohol co-use in AI/AN populations, 1 study found that co-use of tobacco and alcohol was highest among AI men (34.0%) compared with AI women (22.8%) and other racial and/or ethnic groups.[17]

However, a wide variation in tobacco and alcohol use patterns exists among AI/AN populations that arise from both Tribal membership and geography. For example, although the overall prevalence of tobacco use is high among people who identify as AI/AN,[4,30] prevalence varies widely from one Tribe to another. Redwood and colleagues (2010)[31] found that current cigarette smoking was much higher among AI/AN in Alaska (31.6%) than among those in the Southwest (8.3%). An earlier 2005 study found that 14% of Southwest Tribal members smoked compared to 50% of Northern Plains Tribal members.[32] Cunningham and colleagues (2019)[33] studied the roles of rurality and living on Tribal lands (living in Tribal lands vs. living in non-Tribal large metropolitan areas vs. living in non-Tribal small metropolitan areas vs. living in non-Tribal rural areas) on cigarette smoking among people identifying as AI/AN. This study found that although there was no statistical difference in smoking between AI/AN individuals living in urban non-Tribal lands and those living in non-Tribal suburban/rural areas, AI/AN individuals living in Tribal lands had a lower smoking rate compared to both non-Tribal urban and suburban/rural areas. In a study examining alcohol-attributable mortality among AI/AN by geographic region of the United States (and other factors), Landen and colleagues (2014)[9] found that AI/AN people living in the Northern Plains had the highest death rates due to alcohol while AI/AN people living in the East had the lowest. This variation in tobacco and alcohol use across Tribal nations and contexts highlights how other sociodemographic factors beyond racial and/or ethnic groupings are important for understanding co-use patterns and resulting health burden.

People identifying as AI/AN have disproportionally lower smoking cessation rates than any other racial and/or ethnic group, a long-spanning disparity that has been documented for more than 3 decades.[4,34] At the individual level, this may be due to a lack of treatment acceptability and cultural appropriateness in intervention approaches or delivery.[35,36] Researchers and practitioners who engage with AI/AN people for tobacco use treatment must distinguish the differences between traditional and commercial tobacco use, because tobacco is a sacred plant to many AI/AN. Another challenge to providing effective treatment is the mistrust that exists within many Tribal communities for government interventions based on historical government actions that adversely affected these communities.[37,38]

The policy environments unique to Tribal lands are important characteristics when considering AI/AN tobacco- and alcohol-associated behaviors and health outcomes. Tribal lands are generally exempt from state policies governing tobacco because federally recognized Tribes are considered sovereign nations;[39,40] this can result in less expensive products.[41,42] However, federally recognized Tribes are subject to federal and state alcohol policies but may also enact their own alcohol policies.[43] Lee and colleagues (2018)[44] concluded that standard measures used to assess alcohol policy may not adequately describe the effects experienced within reservations and the complex environments in which each Tribe operates.

Given the wide variation of both tobacco and alcohol use patterns and rates of cancer incidence and risk for AI/AN populations, disaggregated data are needed to reflect the heterogeneity within AI/AN subgroups. National estimates are often limited both by the number of participants in each race and/or ethnic group that was sampled for inclusion in the surveys and the measures used to assess race and/or ethnicity (e.g., collapsing people from many different Tribes, each with their own cultural practices and norms, into one AI/AN category).[45,46] This monograph highlights the need for disaggregated data reporting and oversampling among multiple Tribal populations to foster greater understanding of tobacco and alcohol co-use patterns and cancer burden among all racial and/or ethnic minority groups, including AI/AN populations.

Co-Use by Educational Attainment

Lower educational attainment is strongly associated with higher cigarette smoking prevalence[4,47]; as noted in the 2024 Surgeon General's report, "prevalence of smoking is substantially higher among people without college diplomas than it is among those with college diplomas."[4,p.10] A study of birth cohort effects among adults ages 25 and older in the 1966–2018 NHIS found that, although smoking prevalence decreased over time across all educational groups, the difference between educational groups widened.[48] In general, lower educational attainment is associated with less alcohol use overall but more frequent heavy episodic drinking,[49-52] although analysis of birth cohort effects in the NAS found that more education was associated with greater heavy drinking in younger birth cohorts (after 1976).[53] A growing literature suggests that although prevalence of alcohol use is lower in people with less education or lower incomes, the harms of drinking can be greater[52,54] (this topic is discussed further in chapter 4).

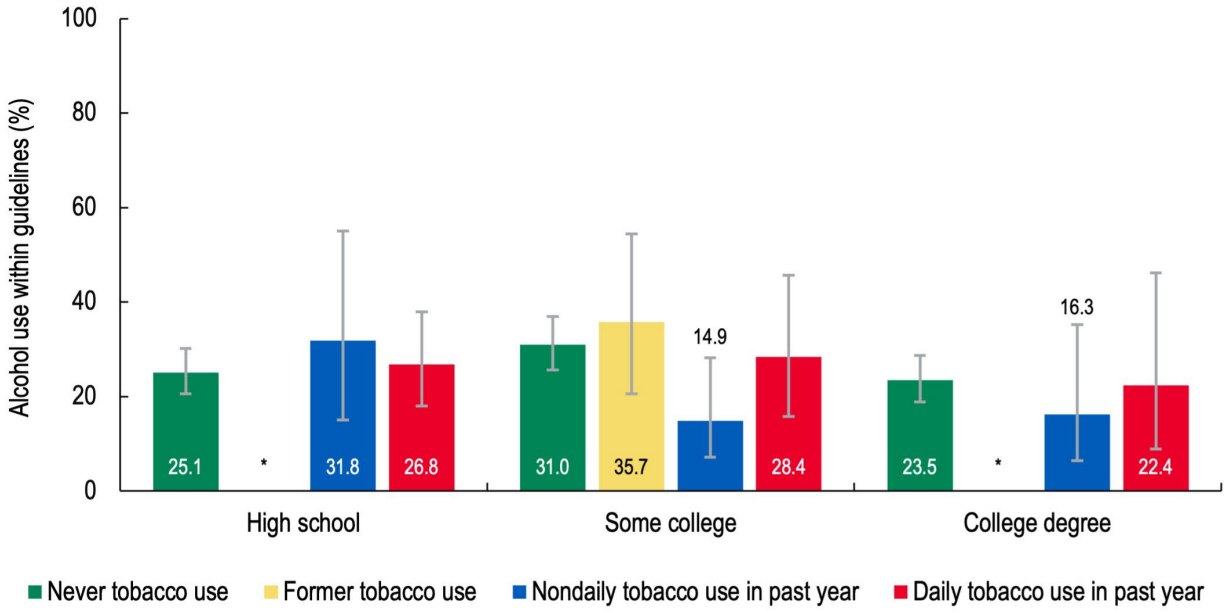
Few nationally representative studies of co-use by educational attainment were identified. In a sample of young adults ages 18–25 in the 2001–2002 NESARC, there was no difference in the relationship between smoking status and alcohol use among young adults by college student status (full- or part-time enrollment in college in the past year).[55] However, among adults overall in the 2012–2013 NESARC, lower educational attainment was associated with increased prevalence of past-year co-use of nicotine and alcohol compared with alcohol only use.[56] A study using 2010–2019 NSDUH data found that, among people who reported "high-risk" alcohol use (>7/14 drinks per week for women/men), educational attainment of high school or more was associated with decreased odds of past-month tobacco use (aOR = 0.6, 95% CI = 0.52–0.69) and nicotine dependence (aOR = 0.48, 95% CI = 0.42–0.54) compared to less than high school education.[18]

National Alcohol Surveys Analysis: Co-Use by Educational Attainment

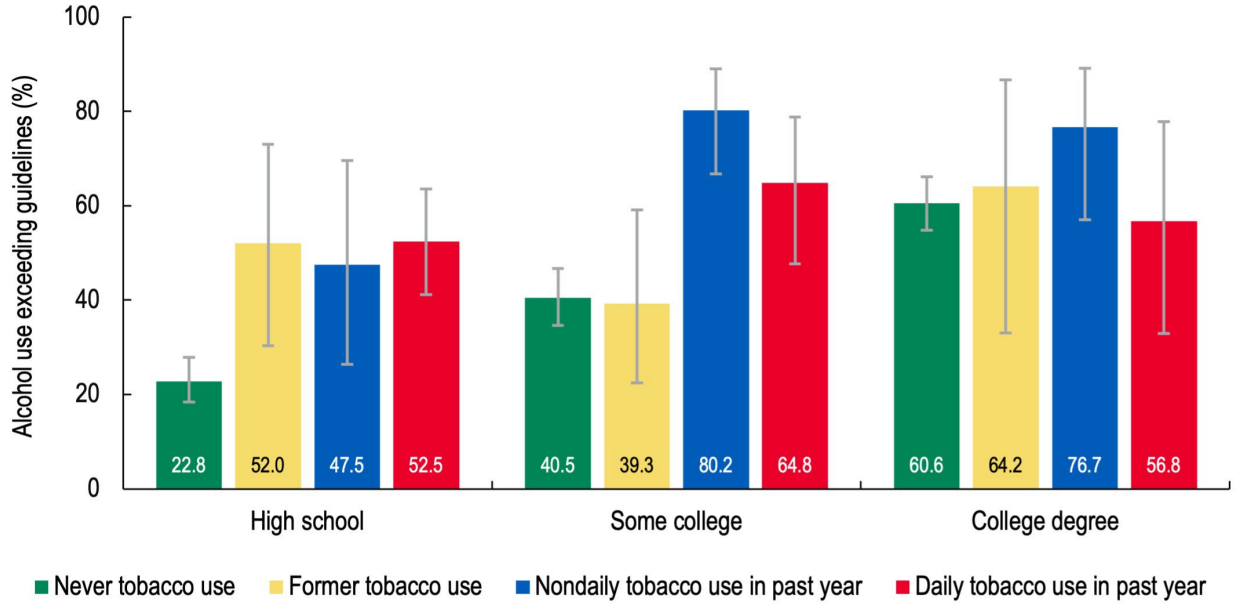
Figure 3.4 (Appendix Table 3B.8) shows alcohol use by tobacco use status and educational attainment among 2014–2015 and 2019–2020 NAS participants. There was a statistically significant association between tobacco use and alcohol use categories across all groups ($p < .001$) except those ages 18–29 with a college degree ($p = .38$). Across most age groups and education levels, those who reported past-year tobacco use had a higher prevalence of alcohol use exceeding guidelines compared to those who never used tobacco. In general, those with either some college education or a college degree had higher prevalence of using alcohol than those with a high school education or less, and this was reflected across age groups and tobacco use categories. Alcohol use exceeding guidelines was most common among those ages 18–29 with either some college education or a college degree who used tobacco and among those with a college education who formerly used tobacco. For example, among people ages 18–29 with a college degree, 76.7% (95% CI, 57.0%–89.1%) of those who used tobacco nondaily reported alcohol use exceeding guidelines, as did 56.8% (95% CI, 33.0%–77.8%) of those who used tobacco daily. Among those ages 30–59, around one-half of those with a college degree who used tobacco daily (54.4%, 95% CI, 43.1%–65.3%) or nondaily (56.4%, 95% CI, 42.6%–69.3%) reported alcohol use exceeding guidelines. Alcohol use exceeding guidelines among those ages 60 and older who reported any past-year tobacco use was around 20% across education categories.

Figure 3.4 Past-Year Alcohol Use by Age, Past-Year Tobacco Use Status, and Educational Attainment (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

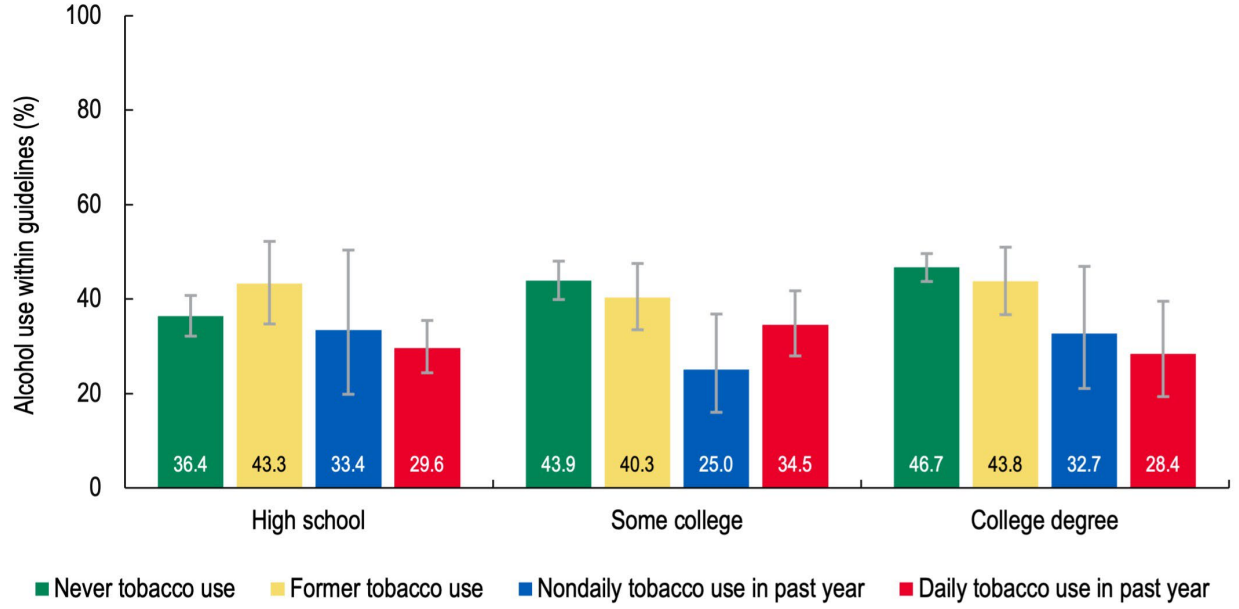
Panel A: Alcohol Use Within Guidelines, Ages 18–29



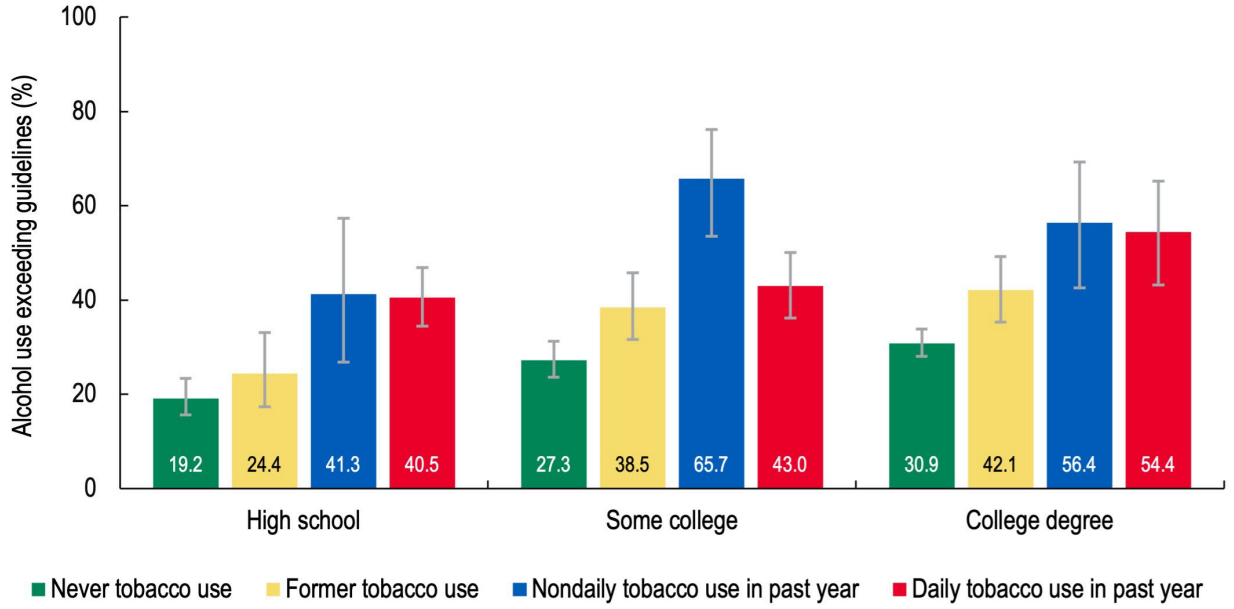
Panel B: Alcohol Use Exceeding Guidelines, Ages 18–29



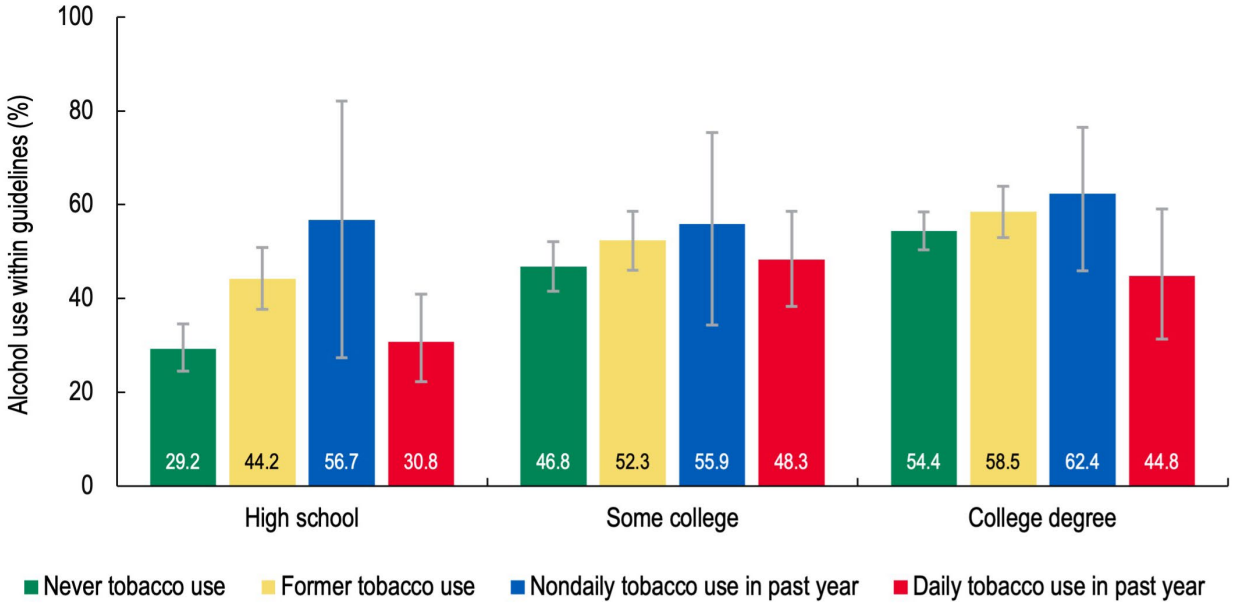
Panel C: Alcohol Use Within Guidelines, Ages 30–59



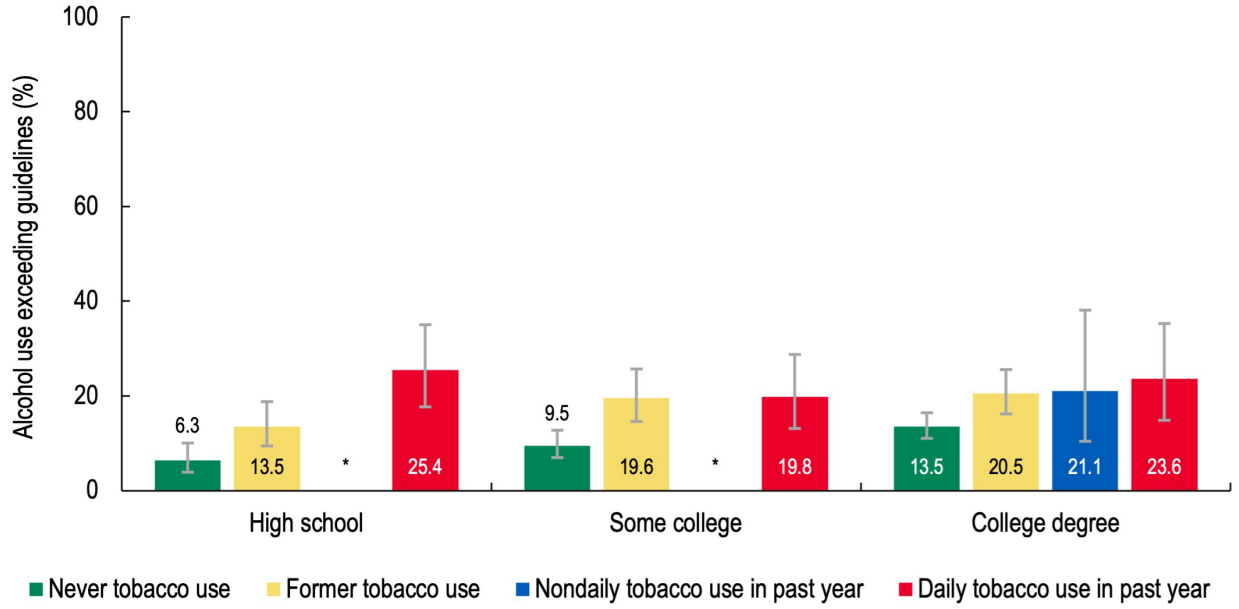
Panel D: Alcohol Use Exceeding Guidelines, Ages 30–59



Panel E: Alcohol Use Within Guidelines, Ages 60 and Older



Panel F: Alcohol Use Exceeding Guidelines, Ages 60 and Older



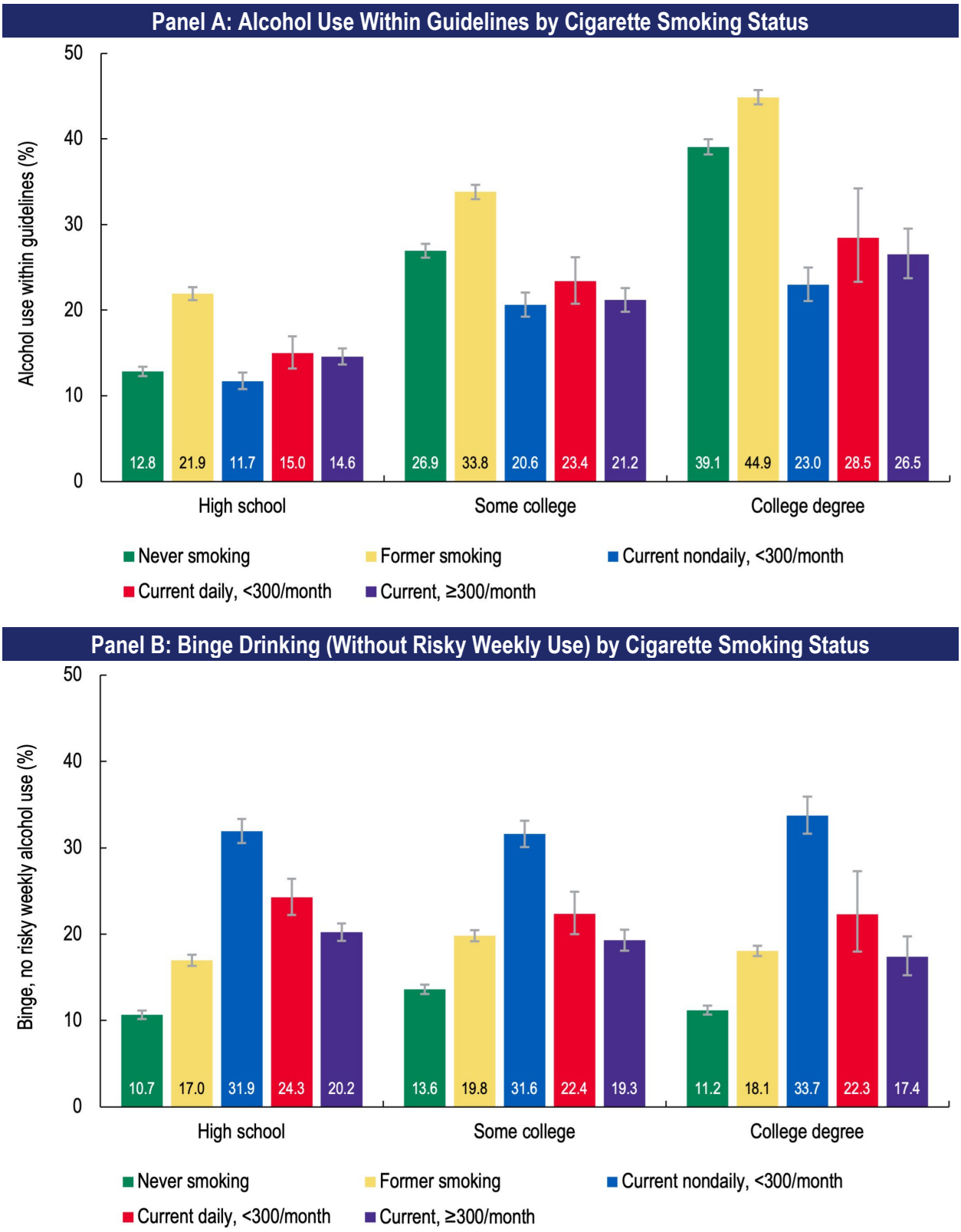
Note. For full data, see Appendix Table 3B.8. *Tobacco use* is defined as use of non-e-cigarette tobacco products (due to survey question wording). *Nondaily* and *daily current tobacco* use are defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured in the past year. No past-year alcohol use is not shown in the charts. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking at least 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[22] and NIAAA's definition of heavy alcohol use.[23]
*Categories with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

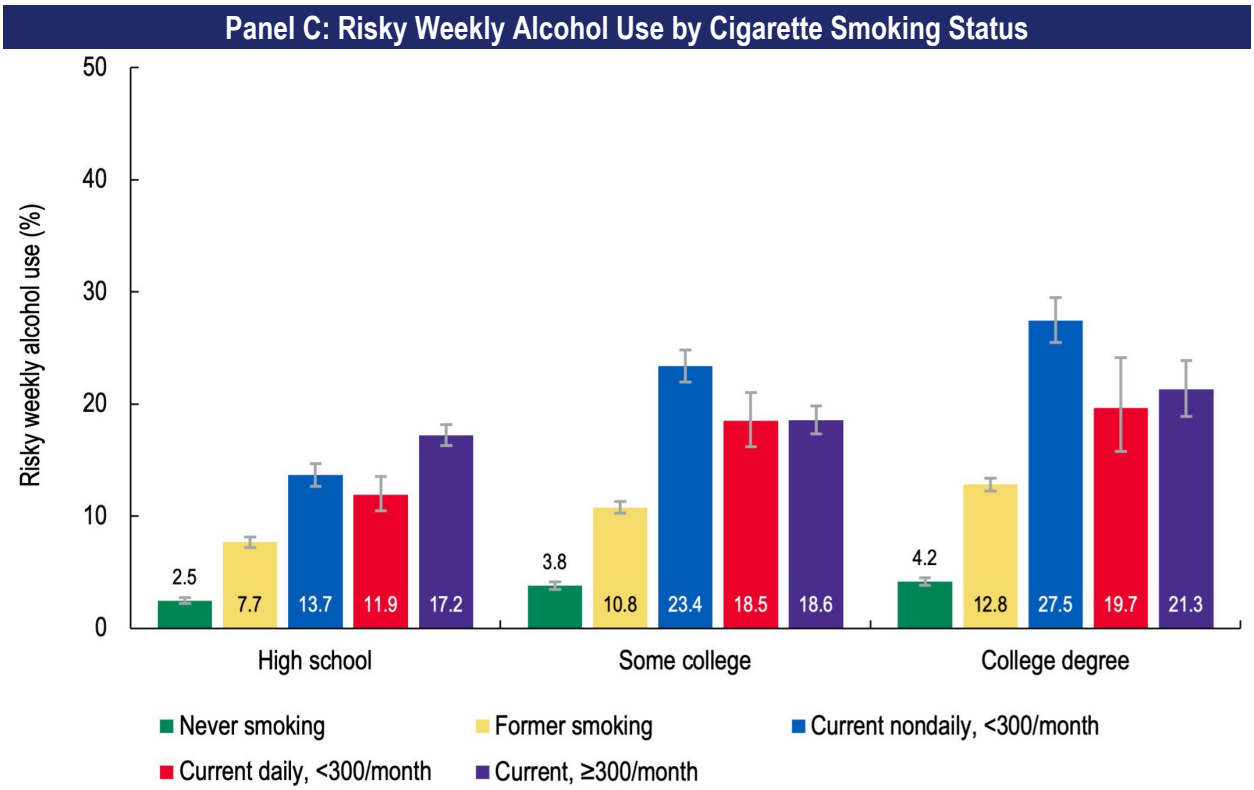
An association with level of education is also seen when looking at 5+ drinking and HID by tobacco use categories (Appendix Table 3B.9). There was no significant association between tobacco use and hazardous alcohol use (HID or 5+ drinking) among those ages 18–29 with a college degree or between tobacco use and HID among those ages 60 and older with some college education, but there was a significant association between tobacco use and hazardous alcohol use among other age and education categories at the $p < .05$ level. In general, individuals who had never used tobacco had a lower prevalence of 5+ drinking and HID than those who used tobacco (daily or nondaily) in the past year. For example, among those ages 30–59 who reported past-year tobacco use, prevalence of 5+ drinking was 31.6% (95% CI, 26.4%–37.4%) among those with a high school education, 35.9% (95% CI, 30.2%–42.1%) among those with some college education, and 48.7% (95% CI, 40.0%–57.4%) among those with a college degree. Among those who never used tobacco in the same age group, prevalence of 5+ drinking was 14.6% (95% CI, 11.4%–18.5%) among those with a high school education, 19.9% (95% CI, 16.8%–23.5%) among those with some college education, and 21.8% (95% CI, 19.2%–24.6%) among those with a college degree.

National Survey on Drug Use and Health Analysis: Co-Use by Educational Attainment

Findings from analysis of the 2015–2019 NSDUH complement the results of the NAS analysis. There was a statistically significant bivariate association between tobacco and alcohol use among all education attainment categories ($p < .001$). Similar to the NAS findings, people who never smoked cigarettes were the most likely to also report never use of alcohol. Among people who never smoked cigarettes, lifetime abstinence from alcohol was highest among those with a high school or less education (42.5%, 95% CI = 41.7%–43.3%); prevalence of lifetime abstinence from alcohol decreased as level of education increased. As seen in Figure 3.5 (Appendix Table 3B.10), past-30-day cigarette smoking was associated with higher prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly alcohol use). Results for smokeless tobacco and cigar smoking were similar to the results for cigarette smoking, with higher prevalence of any alcohol use and alcohol use exceeding guidelines among people with a college degree compared to other education groups (Appendix Tables 3B.11 and 3B.12). Binge drinking (without risky weekly use) was particularly prevalent among people who smoked cigarettes nondaily or used smokeless tobacco nondaily, but prevalence was very similar across levels of educational attainment (31.9% [95% CI, 30.5%–33.3%], 31.6% [95% CI, 30.1%–33.1%], and 33.7% [95% CI, 31.6%–35.9%] for high school education or less, some college education, and college degree, respectively, among those who smoked nondaily <300 cigarettes/month; 27.2% [95% CI, 24.8%–29.7%], 34.7% [95% CI, 31.8%–37.9%], and 39.7% [95% CI, 35.3%–44.3%] for high school education or less, some college education, and college degree, respectively, among those who used smokeless tobacco nondaily). In contrast, risky weekly alcohol use increased as level of education increased among all 3 groups of people who currently smoke cigarettes. This was especially true for people in the nondaily <300 cigarettes/month group; among those with a high school or less education, 13.7% (95% CI, 12.7%–14.7%) reported risky weekly alcohol use, compared to 27.5% (95% CI, 25.5%–29.5%) among those with a college degree.

Figure 3.5 Past-30-Day Alcohol Use by Educational Attainment and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)





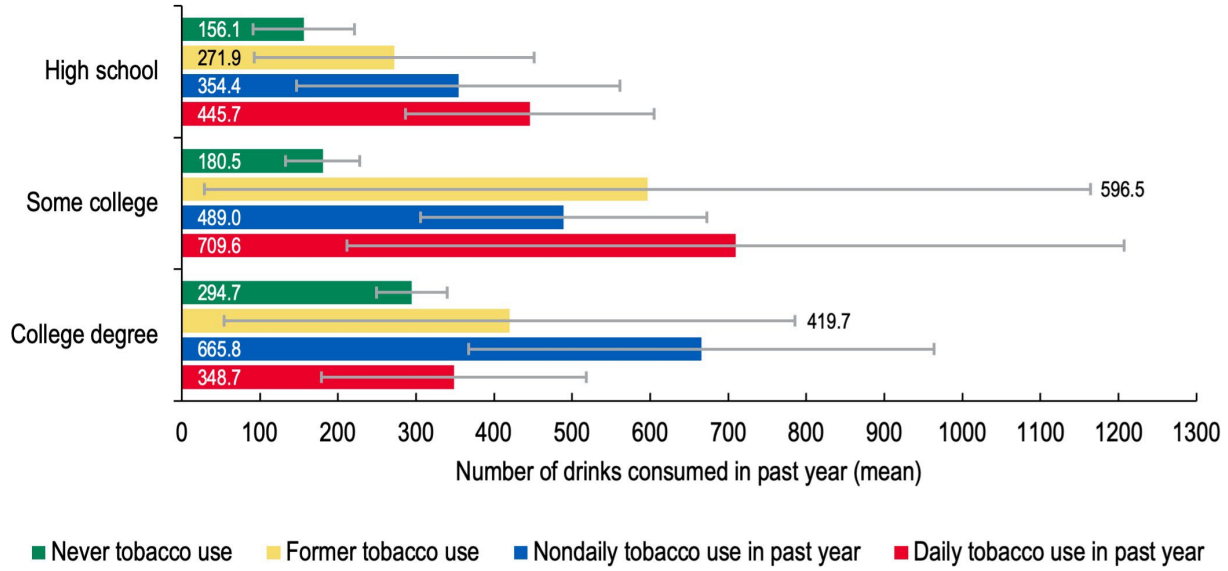
Note. For full data, see Appendix Table 3B.10. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

National Alcohol Surveys Analysis: Past-Year Volume of Consumption by Educational Attainment

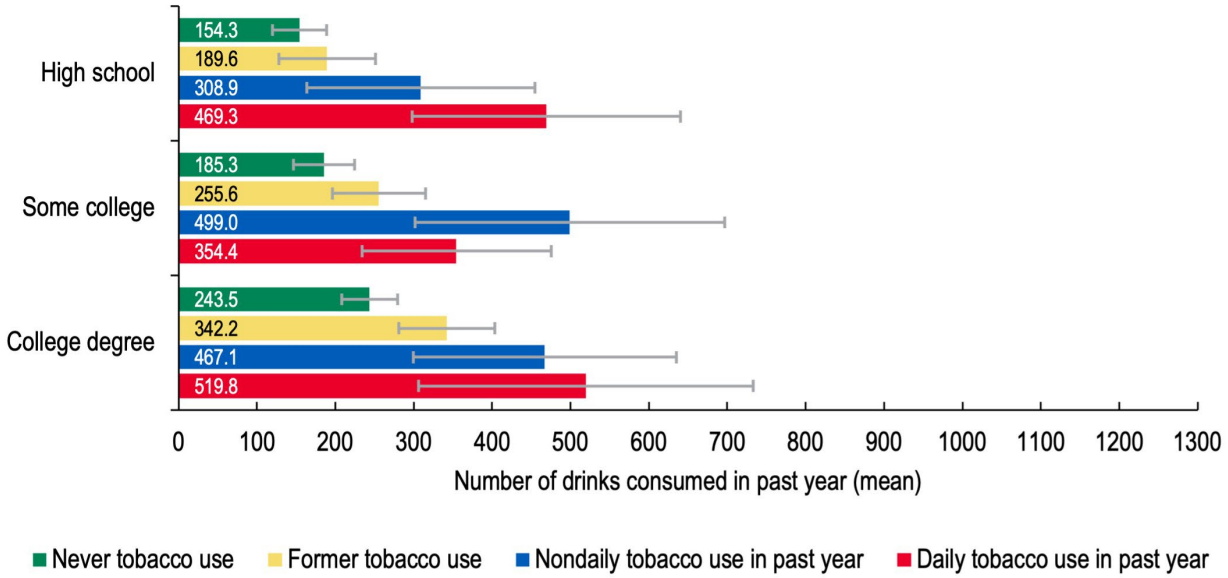
Overall alcohol consumption (mean number of drinks consumed in past year) among 2014–2015 and 2019–2020 NAS participants, stratified by educational attainment and tobacco use status, is shown in Figure 3.6 (Appendix Table 3B.13). Past-year tobacco use was significantly associated with higher overall alcohol consumption compared with never tobacco use at the $p < .05$ level across educational and age groups except those ages 60 and older with a college degree ($p < .1$). Among those ages 30–59 with some college education or a college degree, and among those ages 60 and older with any level of education, former tobacco use was associated with higher alcohol consumption compared with never tobacco use. In most age and education categories, daily and nondaily tobacco use were associated with the highest alcohol consumption. Adults ages 60 and older with a high school education who never used tobacco reported the fewest number of mean drinks consumed.

Figure 3.6 Average Number of Drinks Consumed in Past Year by Age, Past-Year Tobacco Use Status, and Educational Attainment (2014–2015 and 2019–2020 National Alcohol Surveys; *N* = 14,125)

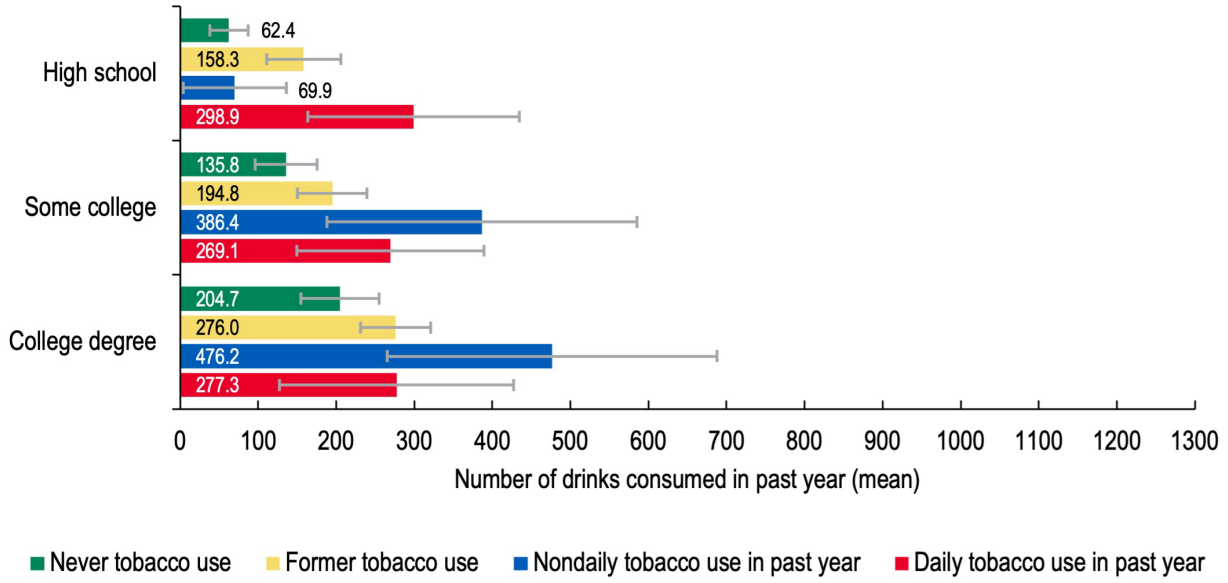
Panel A: Average Number of Drinks Consumed in Past Year, Ages 18–29



Panel B: Average Number of Drinks Consumed in Past Year, Ages 30–59



Panel C: Average Number of Drinks Consumed in Past Year, Ages 60 and older



Note. For full data, see Appendix Table 3B.13. *Tobacco use* is defined as use of non-e-cigarette tobacco products (due to survey question wording). *Nondaily* and *daily current tobacco use* are defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. Alcohol use is measured in the past year.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

National Alcohol Survey Analysis: Lifetime Cigarette Pack Years and Heavy Alcohol Use by Educational Attainment

Lifetime cigarette smoking and alcohol use associations for different education groups were assessed for 2014–2015 NAS participants (Appendix Table 3B.14). Across age and level of education, people who never smoked were least likely to reach the highest 5+ drinking day category for their age group (≥500 for those ages 18–29; ≥1,000 for those ages 30–59 and 60 and older). Reaching ≥1,000 5+ drinking days generally declined with age, regardless of level of education and cigarette pack year category. There was a statistically significant positive association between pack years and lifetime 5+ drinking days among all education and age groups ($p < .001$). As noted in chapter 2, the presence of a substantial number of adults younger than 30 reporting 40+ pack years of tobacco use warrants attention given the health effects of such high intensity use and the potential for measurement error associated with self-report.

Summary: Co-Use by Educational Attainment

The literature indicates that lower educational attainment is associated with higher cigarette smoking prevalence and less alcohol use compared to higher educational attainment. Across tobacco use categories, analysis of NAS and NSDUH data found that, in general, alcohol use was most common among those with some college education or a college degree. Analysis of the NAS demonstrated that alcohol use exceeding guidelines was most common among those 18–29 years with either some college education or a college degree who also used tobacco; the association of co-use with level of education was least apparent among NAS participants ages 60 and older. Analysis of NSDUH found that risky weekly alcohol use was most common among people who smoked nondaily and had some college education or a college degree. Trends among

NSDUH participants who reported use of smokeless tobacco or cigars were roughly similar to those among participants who reported cigarette smoking: alcohol use and alcohol use exceeding guidelines (binge drinking and risky weekly use) were most common among participants with some college education or a college degree.

Co-Use by Income

Compared to people with higher incomes, those with lower incomes have a higher prevalence of current smoking[4,47] and more abstinence from drinking.[57,58] However, people with lower incomes who do report drinking also report more heavy drinking and greater negative alcohol-related consequences than people with higher incomes who report drinking.[52,57] Higher incidence of alcohol- and tobacco-associated cancers, such as esophageal[59-62] and head and neck cancers,[63,64] has also been documented among people of lower socioeconomic status compared to those of higher socioeconomic status. Factors influencing income-based disparities may include lack of access to reliable preventive care and treatment, occupational exposures to carcinogens, and differential tobacco and alcohol use patterns.[65] Additional studies of tobacco and alcohol co-use patterns as a function of income have been conducted outside the United States. For example, studies in Australia[66] and Thailand[67] found greater odds of tobacco and alcohol co-use among people with lower income compared to those with higher income.

However, as of 2026, few studies have analyzed tobacco-alcohol co-use among nationally representative, U.S.-based population surveys. In the 2012–2013 NESARC, lower annual income was associated with higher odds of co-use of nicotine and alcohol than alcohol use alone.[56] A study using 2010–2019 NSDUH data found that, among people who reported "high-risk" alcohol use (>7/14 drinks per week for women/men), lower income was associated with increased odds of past-month tobacco use (<$50,000: aOR = 1.85, 95% CI = 1.69–2.02; $50,000–$74,999: aOR = 1.44, 95% CI = 1.29–1.62) and nicotine dependence (<$50,000: aOR = 2.12, 95% CI = 1.90–2.37; $50,000–$74,999: aOR = 1.71, 95% CI = 1.49–1.96) compared to income of $75,000 or higher.[18] There is literature that examines tobacco and alcohol co-use in specific subpopulations within the United States, although these studies are often conducted with non-representative samples. For example, a study of low-income Black women in the United States found that current alcohol use (having ≥1 beverage monthly or more) was highest among those reporting smoking 11–19 cigarettes per day.[68] This study also found that weekly heavy episodic drinking (5+ drinks on a single occasion) was significantly higher among those who smoked 1–10 cigarettes per day (23%), 11–19 cigarettes per day (57%), or ≥20 cigarettes per day (31%), compared with those who did not smoke (1%).[68] In addition, a study of 6-year trajectories of concurrent tobacco and alcohol use among college students found that those from higher income families had 1.3 times the odds of belonging to the highest risk concurrent trajectory class, defined as steady high alcohol/steady high tobacco use (versus a low alcohol/abstaining tobacco class).[69] Higher family income was also positively associated with trajectory classes involving decreasing tobacco use.

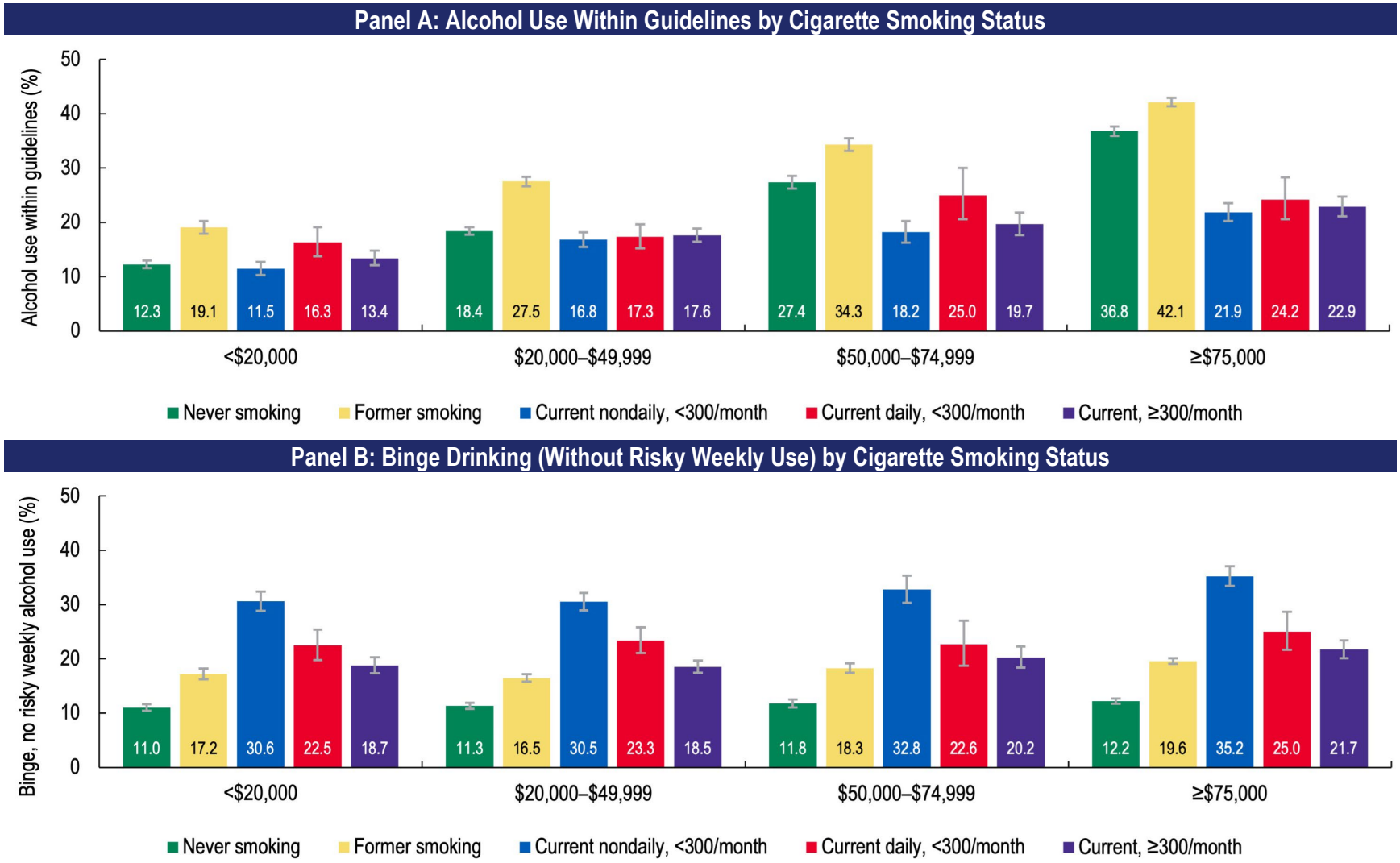
National Survey on Drug Use and Health Analysis: Co-Use by Income

Within income categories in the 2015–2019 NSDUH, levels of binge drinking (without risky weekly alcohol use) and risky weekly alcohol use were generally higher among people who used cigarettes, smokeless tobacco, or cigars in the past 30 days than among people who never or formerly used these products (Figure 3.7; Appendix Tables 3B.15, 3B.16, 3B.17). There was a statistically significant association between tobacco and alcohol use across income categories ($p < .001$). Across groups, people who smoked cigarettes nondaily had the highest prevalence of binge drinking (around 30% to 35%). Although binge drinking prevalence was similar across income categories among people who smoked, prevalence of risky weekly alcohol use among people who smoked increased as income increased. Around 20%–25% of people with an income of at least $75,000 who smoked reported risky weekly use, compared to around 12%–16% of people with an income of <$20,000 who smoked.

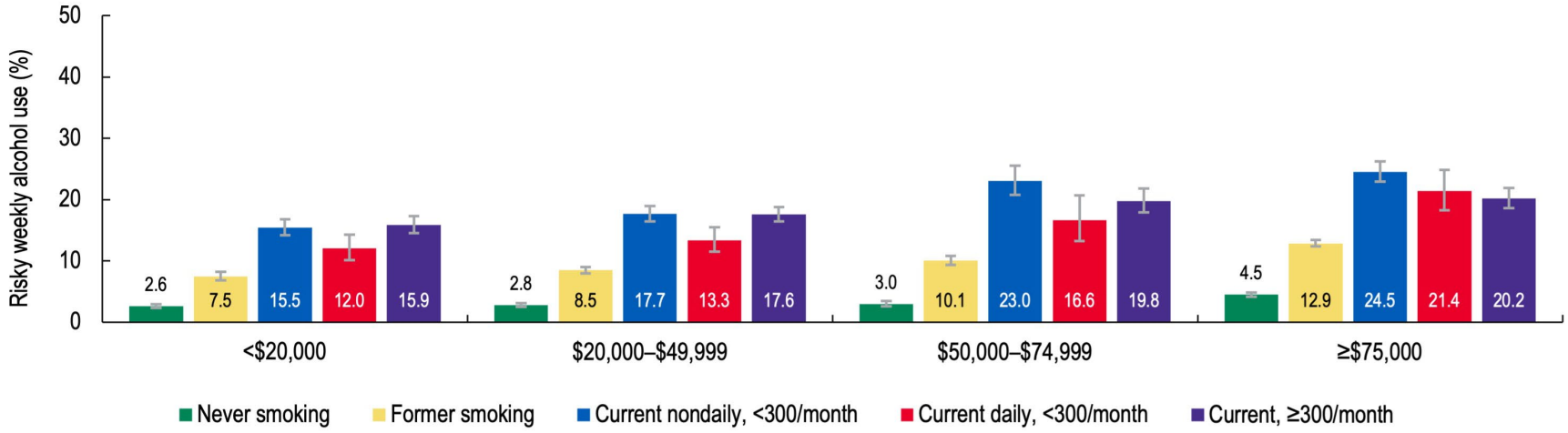
Further, the prevalence of overall alcohol use increased as income increased. Those with an annual income <$20,000 had almost 3 times the prevalence of lifetime abstinence from alcohol compared to those with an income of at least $75,000 (24.0% [95% CI, 23.3%–24.6%] versus 8.8% [95% CI, 8.5%–9.1%], respectively), and twice the prevalence of lifetime abstinence from alcohol compared to those with an income between $50,000 and $74,999 (12.2%, 95% CI = 11.7%–12.8%).

An association between income and alcohol use patterns was also seen among those who never smoked cigarettes. Those in the lowest income category had the highest prevalence of lifetime abstinence from alcohol (44.0%, 95% CI = 42.9%–45.2%). In contrast, among the highest income group, only 20.1% (95% CI, 19.4%–20.8%) of those who never smoked cigarettes reported lifetime abstinence from alcohol. Similar patterns were also observed among people who never used smokeless tobacco or cigars. In addition, lower income people who formerly smoked were more likely to report former alcohol use (46.9% [95% CI, 45.4%–48.3%] among those with income <$20,000) than higher income people who formerly smoked (23.6% [95% CI, 22.9%–24.3%] among those with income of at least $75,000).

Figure 3.7 Past-30-Day Alcohol Use by Income and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* =210,684)



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



Note. For full data, see Appendix Table 3B.15. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Summary: Co-Use by Income

The literature indicates that lower income is associated with higher cigarette smoking prevalence and less alcohol use compared to higher income. Across tobacco use categories, analysis of NSDUH data found that, in general, alcohol use was most common among those with income of $50,000 or higher. Risky weekly alcohol use among people who smoked also increased as income increased. Trends among NSDUH participants who reported use of smokeless tobacco or cigars were roughly similar to those among participants who reported cigarette smoking; alcohol use and alcohol use exceeding guidelines (binge drinking and risky weekly use) were most common among participants in the highest income category.

Co-Use by Geographic Location: Region, Urban/Rural, and Other Location Differences

Determining place-based differences in tobacco and alcohol co-use can help guide prevention efforts and effective policies in reducing disparities. U.S. regions that might warrant focused attention include the Midwest and South, where prevalence of tobacco or alcohol use is higher for some demographic groups than in other regions.[4,70] A study that used 2018 Behavioral Risk Factor Surveillance System (BRFSS) data to estimate regional prevalence of past-30-day binge drinking (≥4/5 drinks on an occasion for women/men) found that the Midwest region had the highest prevalence of binge drinking (20.0%, 95% CI = 19.6%–20.5%).[49] Similarly, a study that used 1965–2018 NHIS data and 1992–2019 Tobacco Use Supplement to the Current Population Survey data, found that smoking prevalence was generally higher in states in the Midwest and South than the Northeast and West.[71] Twelve states in the Midwest and South (Alabama, Arkansas, Indiana, Kentucky, Louisiana, Michigan, Mississippi, Missouri, Ohio, Oklahoma, Tennessee, and West Virginia) have nearly 50% higher smoking prevalence than other states, according to 2020 BRFSS data.[72] Differences in prevalence of tobacco and alcohol use may be attributed, in part, to differences in the sociodemographic makeup of states and regions and differences in tobacco and alcohol control policies. No studies have examined prevalence of tobacco and alcohol co-use by state and/or region.

The proportion of urban and rural residents within a geographic area can have a substantial influence on the prevalence of co-use. People living in urban, suburban, and rural areas vary in their use of tobacco and alcohol as well as their use patterns, such as types of products used. In rural areas, studies have found earlier initiation and higher prevalence of tobacco use[4,73] compared to urban areas. With regard to alcohol use, adults living in rural areas are more likely to abstain from alcohol compared to those in urban areas,[70,74,75] but those who do consume alcohol are more likely to report hazardous alcohol use (e.g., binge drinking, diagnosis of AUD, daily drinking, or Alcohol Use Disorder Identification Test [AUDIT] scores above a defined threshold) and alcohol-related harms.[76] A study using 2010–2019 NSDUH data found that "high-risk" alcohol use (>7/14 drinks per week for women/men), was more prevalent in metropolitan areas than rural areas, and, among adults who reported high-risk alcohol use, residing in a metropolitan area was associated with decreased odds of past-month tobacco use (small metropolitan: aOR = 0.87, 95% CI = 0.78–0.97; large metropolitan: aOR = 0.74, 95% CI = 0.67–0.83) and nicotine dependence (small metropolitan: aOR = 0.87, 95% CI = 0.78–0.97; large metropolitan: aOR = 0.75, 95% CI = 0.67–0.84) compared with residing in an rural area.[18] Further, in a study looking at tobacco and alcohol use in adolescents (ages 12–17

years) and adults (18 years or older) in the 2002–2004 NSDUH,[77] both adolescents and adults in rural counties had the highest proportion of any past-month tobacco use (adolescents: 20.5% in rural counties vs. 13.7% in urban counties; adults: 35.7% in rural counties vs. 30.7% in urban counties). Use of smokeless tobacco products was also higher among both adolescents and adults in rural counties than in urban counties (adolescents: 5.7% in rural counties vs. 1.6% in urban counties; adults: 7.5% in rural counties vs. 2.7% in urban counties). However, age differences between adolescents and adults in rural counties emerged when looking at alcohol use. Among adolescents, participants in rural counties had significantly higher past month alcohol use, binge alcohol use (defined as ≥5 drinks on the same occasion at least 1 day in the past 30 days), and heavy alcohol use (defined as ≥5 drinks on the same occasion on 5 or more days in the past 30 days) than those in urban counties (past month: 20.4% vs. 17.2%; binge: 13.8% vs. 10.3%; heavy: 3.3% vs. 2.5%). In contrast, adults in rural counties consistently had a lower prevalence of alcohol use (all measures) compared to those in urban counties (past month: 42.6% vs. 56.0%; binge: 21.2% vs. 24.5%; heavy: 6.6% vs. 7.3%).[77]

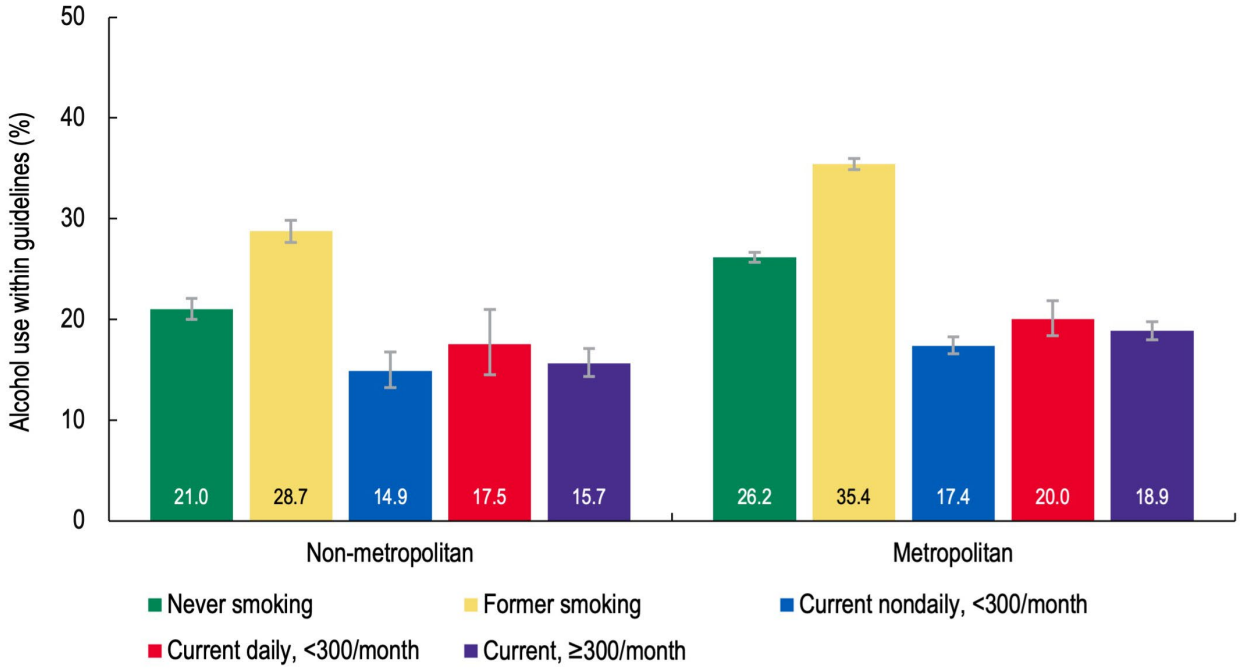
Few studies examine the prevalence of co-use patterns in rural areas within the United States. One cross-sectional study, using data collected in 1993 from 630 high school students from a rural, tobacco-growing county, found that about 40% of the students had smoked cigarettes and almost 65% had consumed alcohol in the past 30 days.[78] Students who reported approval of drinking were more likely to drink (beta = 0.57) and smoke (beta = 0.37) than those who did not approve of drinking. Additionally, students who reported smoking tobacco in the past 30 days (compared to those who did not) were about three times more likely to report drinking alcohol, and students who reported drinking alcohol in the past 30 days (compared to those who did not) were about three times more likely to report smoking tobacco. Further, those who had friends who drank alcohol also had an increased likelihood of smoking (beta = 0.23) than those who did not report having friends who drink alcohol. Absolute levels of tobacco and alcohol use in adolescents have declined dramatically since 1993,[79] but tobacco and alcohol use persist in both rural and urban areas.[70,80] There is a critical need for more research on tobacco and alcohol co-use by geographic location to inform prevention and policy efforts, particularly to address harms from alcohol.[76] As discussed in relation to race and/or ethnicity, aggregated estimates across geographic areas that merge rural and urban spaces may conceal important variation in substance use and its consequences.

National Survey on Drug Use and Health Analysis: Co-Use by Metropolitan Status

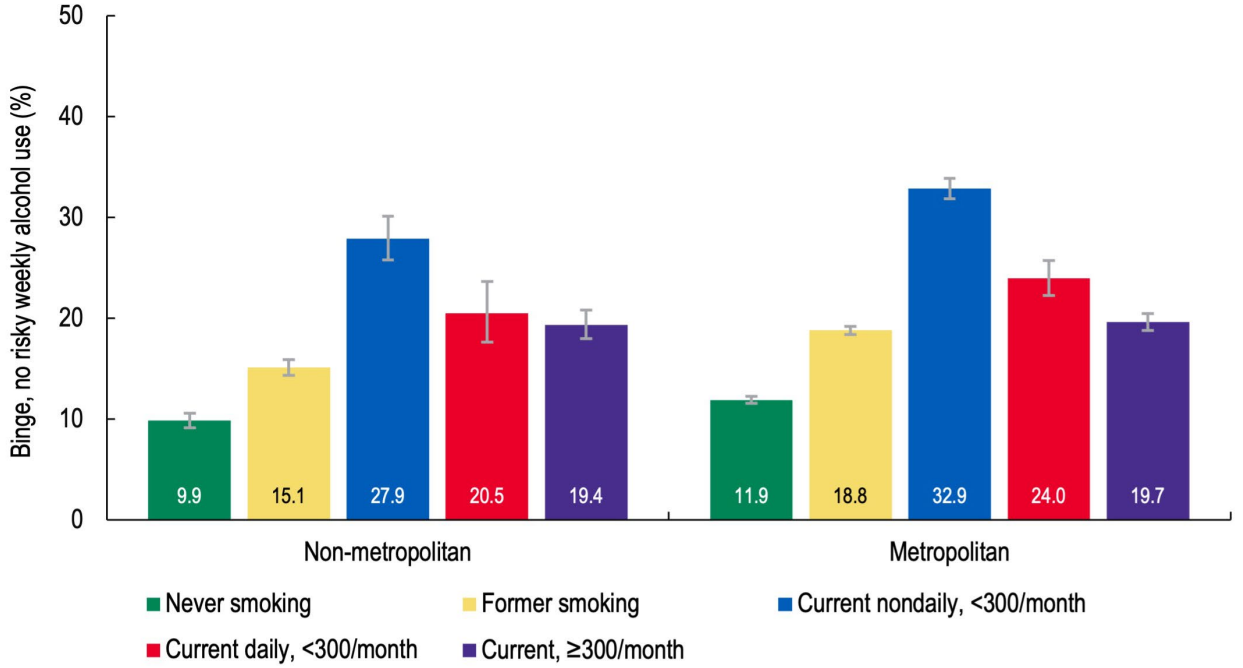
NSDUH data were analyzed to examine alcohol use by tobacco use and county metropolitan status (Figure 3.8; Appendix Tables 3B.18, 3B.19, 3B.20). There was a statistically significant bivariate association between tobacco and alcohol use among participants in both metropolitan and nonmetropolitan counties ($p < .001$). Overall, prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) was highest among adults in metropolitan counties who reported use of cigarettes or smokeless tobacco in the past 30 days. For example, prevalence of binge drinking (without risky weekly use) among people in nonmetropolitan counties who smoked nondaily <300 cigarettes/month was 27.9% (95% CI, 25.8%–30.2%) and 32.9% (95% CI, 31.9%–33.9%) in metropolitan counties. Among people who reported daily use of smokeless tobacco, prevalence of risky weekly use was 15.3% (95% CI, 13.1%–17.8%) among people in nonmetropolitan counties and 23.9% (95% CI, 21.4%–26.5%) among people in metropolitan counties.

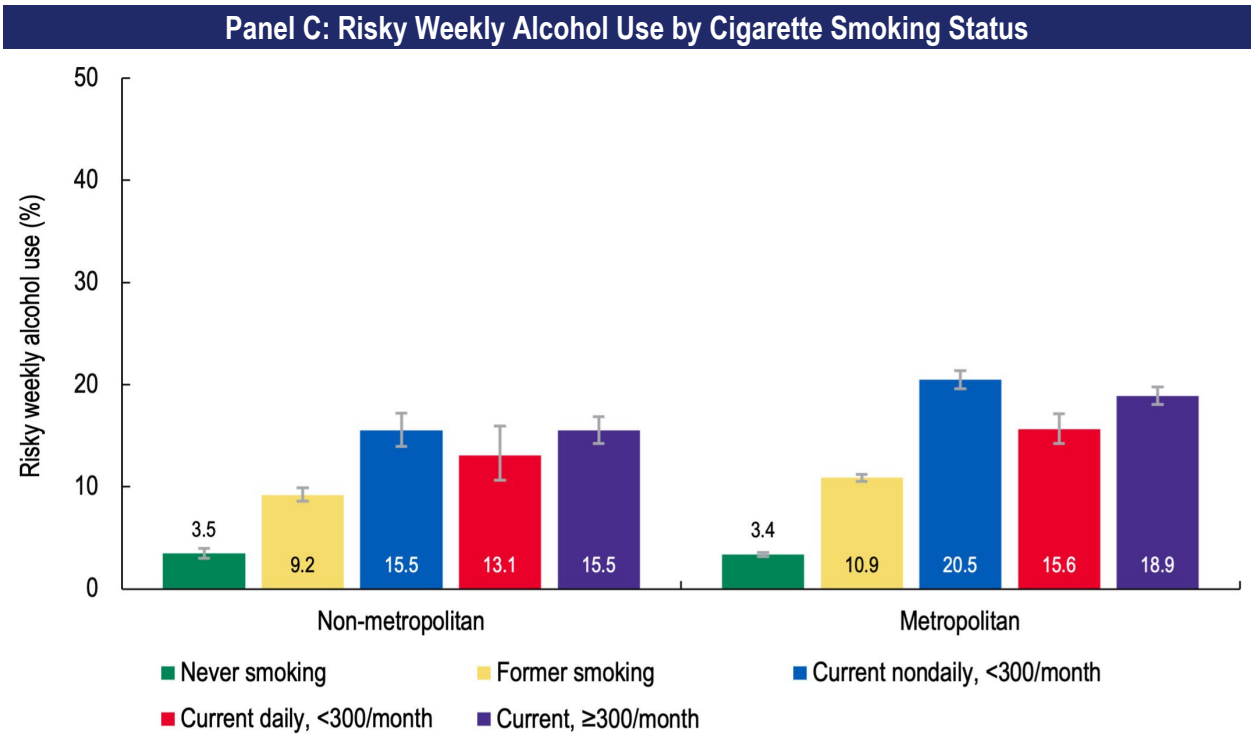
Figure 3.8 Past-30-Day Alcohol Use by Metropolitan Status and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)

Panel A: Alcohol Use Within Guidelines by Cigarette Smoking Status



Panel B: Binge Drinking (Without Risky Weekly Use) by Cigarette Smoking Status





Note. For full data, see Appendix Table 3B.18. The 2015–2019 National Survey on Drug Use and Health categorizes participants' counties of residence as large metropolitan, small metropolitan, and nonmetropolitan based on the 2013 Rural/Urban Continuum Codes[81]; large and small metropolitan counties are defined as *metropolitan* in this analysis. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as no binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Summary: Co-Use by Geographic Location

Compared to urban areas, studies have found that people in rural areas had higher prevalence of tobacco use and lifetime abstinence from alcohol, but also higher likelihood of hazardous alcohol use among people who drink. In the 2015–2019 NSDUH, prevalence of past-30-day risky weekly alcohol use was highest among adults in metropolitan counties who reported use of cigarettes or smokeless tobacco. Urban and rural areas seem likely to differ in different regions of the United States.

Co-Use Among People Who Identify as Part of a Sexual Minority Group

People who identify as a sexual minority, or those who self-report lesbian, gay, bisexual, or another nonheterosexual orientation, can face unique challenges and stressors as a result of their sexual orientation that puts them at increased risk for substance use.[82] These individuals have consistently shown greater tobacco use, alcohol use, and their co-use compared to people who identify as heterosexual.[4,83-88] In a 2022 scoping review of 63 studies on sexuality differences in polysubstance use in adolescents and adults,[86] one of the few consistent findings was that sexual minority individuals report more frequent polysubstance use than heterosexual individuals. A nationally representative study using 2013–2014 Population Assessment of Tobacco and

Health (PATH) study data found in a latent class analysis that, compared to heterosexual participants, sexual minority adolescents ages 15–17 had greater odds of being in 2 polysubstance use classes that included use of tobacco products and alcohol than in the class characterized by abstention from tobacco, alcohol, and cannabis.[88] Further, in a study of same-day cigarette, cannabis, and alcohol use among people who smoke cigarettes (N = 147), almost 71% of sexual minority individuals reported using all 3 substances on the same day compared to 56% of heterosexual individuals.[89]

In a study using data from the 2015 Youth Risk Behavior Surveillance System (YRBS), Dermody and colleagues (2018)[84] used a latent class analysis to characterize use patterns of tobacco products, alcohol, and cannabis among adolescents in 9th through 12th grade. Compared to heterosexual adolescents, bisexual adolescents had 2.64 (95% CI, 1.62–4.29) times the odds of belonging to a class characterized by polysubstance use of alcohol, binge drinking, cannabis, e-cigarettes, and other tobacco products, and adolescents who indicated they were "not sure" of their sexual orientation had 2.61 (95% CI, 1.48–4.63) times the odds of belonging to this polysubstance use class. However, lesbian/gay adolescents did not have significantly increased odds of belonging to this polysubstance use class compared with heterosexual adolescents (aOR = 2.14, 95% CI = 0.92–4.97).[84] A separate study that conducted a latent class analysis using 2015 YRBS data found that both lesbian/gay and bisexual adolescents had increased odds over heterosexual adolescents of belonging to a class characterized by tobacco and alcohol co-use (lesbian/gay: aOR = 3.10 [95% CI = 2.30–4.18]; bisexual: aOR = 2.95 [95% CI 1.87–4.63]) than a class of nonusers.[83] Adolescents who were unsure of their identity also had increased odds of using tobacco and alcohol (aOR = 1.67, 95% CI = 1.02–2.73) compared to heterosexual adolescents.

Another study used daily surveys to capture information about concurrent daily cigarette and alcohol use among 246 women and found that, compared to heterosexual women, sexual minority women drank more per drinking day, had a higher proportion of days smoking across the study (18% vs. 4%), smoked more per smoking day, and had a higher proportion of days drinking and smoking (7% vs. 1%).[87] The prevalence of days drinking, smoking, and drinking and smoking were similar for lesbian and bisexual women and elevated compared to heterosexual women.

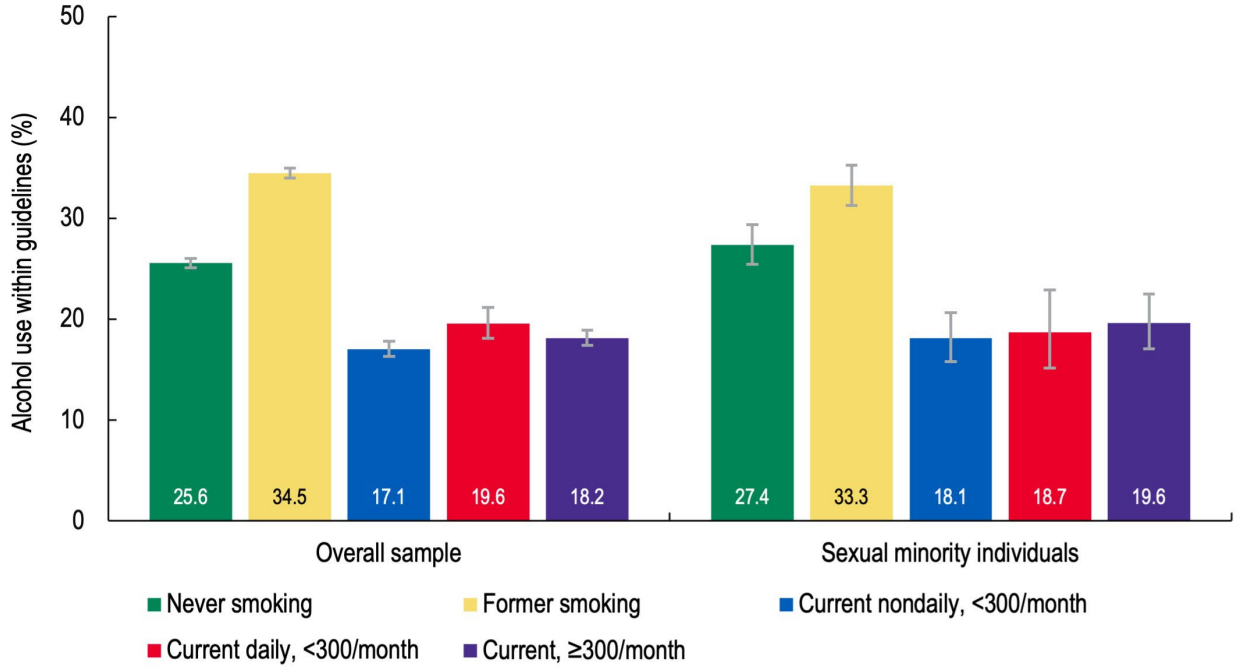
National Survey on Drug Use and Health Analysis: Co-Use by Sexual Minority Status

Analysis of NSDUH data found higher alcohol use among sexual minority adults who smoke cigarettes, compared to the general population (Figure 3.9; Appendix Table 3B.21). There was a statistically significant bivariate association between tobacco and alcohol use among sexual minority participants and the overall NSDUH sample ($p < .001$; Appendix Tables 3B.21, 3B.22, 3B.23). Overall, among those who reported sexual minority status (lesbian, gay, or bisexual), around one-third reported any alcohol use exceeding guidelines (binge drinking without risky weekly use: 21.2%, 95% CI = 20.3%–22.2%; risky weekly alcohol use: 14.5%, 95% CI = 13.6%–15.4%), compared to around one-quarter among the overall NSDUH sample (binge drinking without risky weekly use: 17.0%, 95% CI = 16.8%–17.2%; risky weekly use: 9.3%, 95% CI = 9.2%–9.5%). Additionally, for the 3 past-30-day cigarette smoking categories, a higher proportion of sexual minority individuals reported risky weekly alcohol use compared to their counterparts in the overall sample; similar results were also observed for people who reported

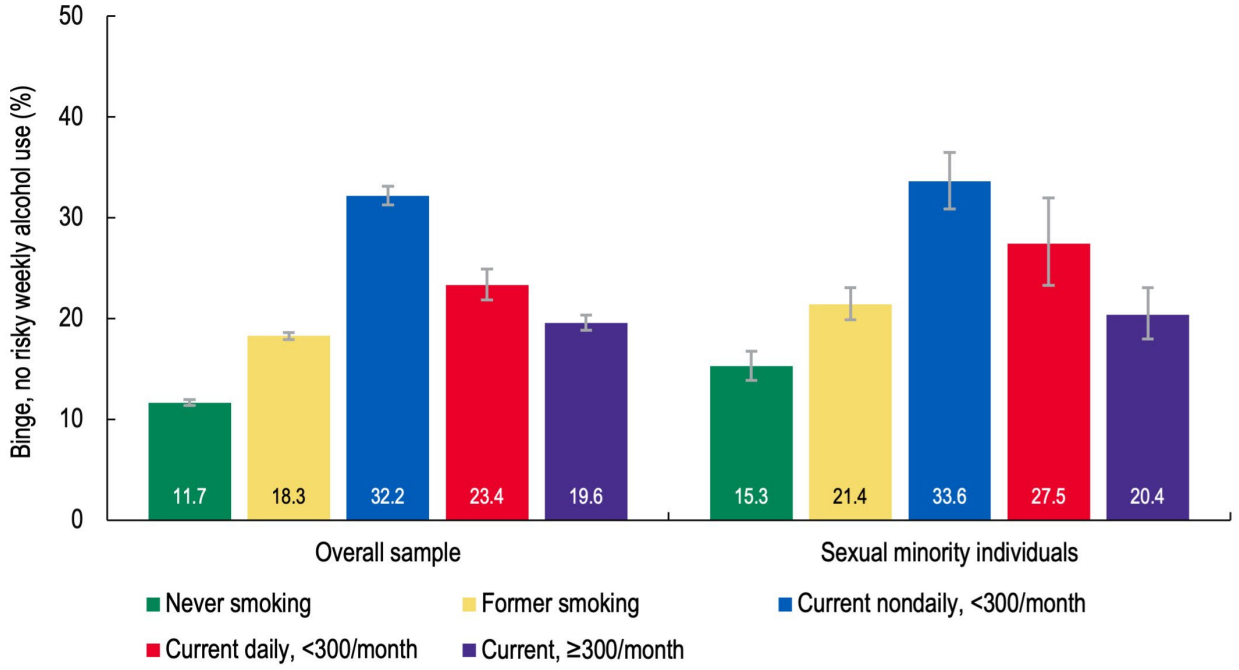
past-30-day cigar smoking. For example, among those who smoked <300 cigarettes/month on a nondaily basis, risky weekly alcohol use was reported by 25.5% (95% CI, 22.9%–28.3%) of sexual minority individuals, compared to 19.8% (95% CI, 19.0%–20.6%) of individuals among the overall NSDUH sample. Among sexual minority individuals, alcohol use exceeding guidelines was highest among those who smoked <300 cigarettes/month; for example, 33.6% (95% CI, 30.9%–36.5%) of those who smoked nondaily <300 cigarettes/month in the past 30 days and 27.5% (95% CI, 23.3%–32.0%) of those who smoked daily <300 cigarettes/month also reported binge drinking (without risky weekly use). Among those who reported past-30-day cigar smoking, 24.7% (95% CI, 21.3%–28.4%) of sexual minority individuals reported risky weekly use, compared with 20.2% (95% CI, 19.2%–21.2%) of the overall NSDUH sample. These findings are consistent with the literature, although smaller differences between sexual minority individuals and the general population in tobacco and alcohol use patterns were observed in this analysis. Greater differences might be observed if stratification by age, sex, and sexual minority status were possible; however, the NSDUH sample had a limited number of sexual minority individuals, preventing a more refined analysis.

Figure 3.9 Past-30-Day Alcohol Use by Sexual Minority Status and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)

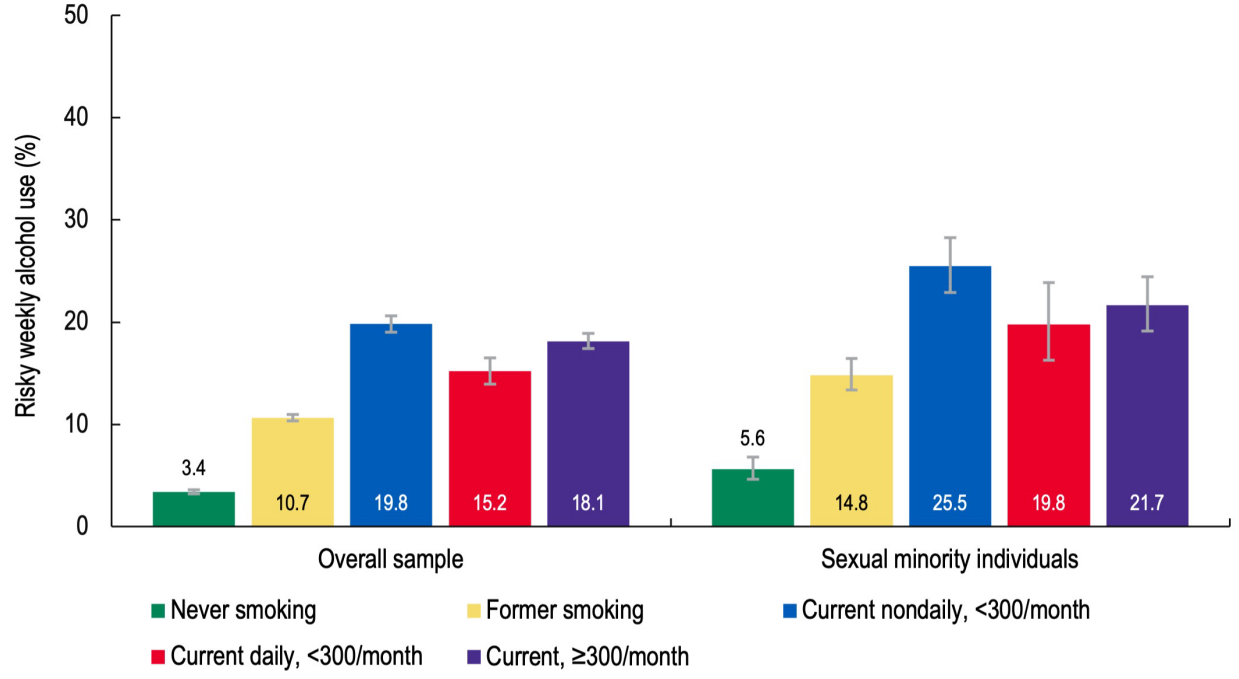
Panel A: Alcohol Use Within Guidelines by Cigarette Smoking Status



Panel B: Binge Drinking (Without Risky Weekly Use) by Cigarette Smoking Status



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



Note. For full data, see Appendix Table 3B.21. *Overall* refers to the total 2015–2019 National Survey on Drug Use and Health sample. *Sexual minority individuals* are defined as participants who identified as "lesbian or gay" or "bisexual", *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as no binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Summary: Co-Use Among People Who Identify as Part of a Sexual Minority Group

The literature indicates that sexual minority individuals have higher prevalence of tobacco and alcohol use compared to heterosexual individuals. Data from the 2015–2019 NSDUH showed that around one-third of sexual minority adults reported past-30-day alcohol use exceeding guidelines (binge drinking and risky weekly alcohol use), compared to one-quarter among the overall NSDUH sample. Binge drinking and risky weekly alcohol use were highest among sexual minority adults who smoked <300 cigarettes per month.

Co-Use Among Military Service Members and Veterans

Veterans have higher prevalence of tobacco use and AUDs compared to the civilian population.[90-92] However, few studies have assessed tobacco and alcohol co-use among military service members and veterans. A national study of more than 5 million U.S. veterans found that, of those who received Veterans Health Administration services in fiscal year 2012, 11.1% received a diagnosis of tobacco use disorder (TUD) only, 3.3% received a diagnosis of AUD only, and 2.7% received diagnoses of both AUD and TUD.[93] Veterans diagnosed with AUD and TUD were more likely to receive diagnoses of other psychiatric disorders compared to those with a TUD only. A study that used data from the 2015 U.S. Department of Defense Health-Related Behaviors Survey found that active duty military service members with probable AUDs (based on Alcohol Use Disorders Identification Test-Concise [AUDIT-C] scores) had significantly greater odds of exclusive cigarette use (aOR = 2.61, 95% CI = 1.98–3.45), exclusive e-cigarette use (aOR = 2.06, 95% CI = 1.50–2.82), and dual use of cigarettes and e-cigarettes (aOR = 3.36, 95% CI = 2.17–5.20) compared to service members without AUDs.[94]

Results from several studies indicate an association between tobacco use and hazardous alcohol use among veterans. An analysis of the Millennium Cohort Study looked at 6,909 military service members and veterans who reported problem drinking (assessed using the lifetime CAGE questionnaire and the PRIME-MD Patient Health Questionnaire [PHQ]) in a 2000 baseline assessment but did not report problem drinking (based on the PHQ only) in the Wave 2 assessment (2004–2006).[95] At the Wave 3 assessment in 2007–2008, 16% of study participants reported a relapse to problem drinking (based on the PHQ) and current smoking was associated with increased relapse to problem drinking (aOR = 1.26, 95% CI = 1.05–1.51).[95] An analysis of tobacco use trajectories among veterans, using the PATH Study Wave 1 (2013–2014) and Wave 2 (2014–2015) data, showed that participants who reported binge drinking had increased odds of being in the continued tobacco product use group (aOR = 4.23, CI = 2.38–7.52) and the tobacco product initiation group (aOR = 8.63, CI = 3.79–19.63) than the non-tobacco use group.[90]

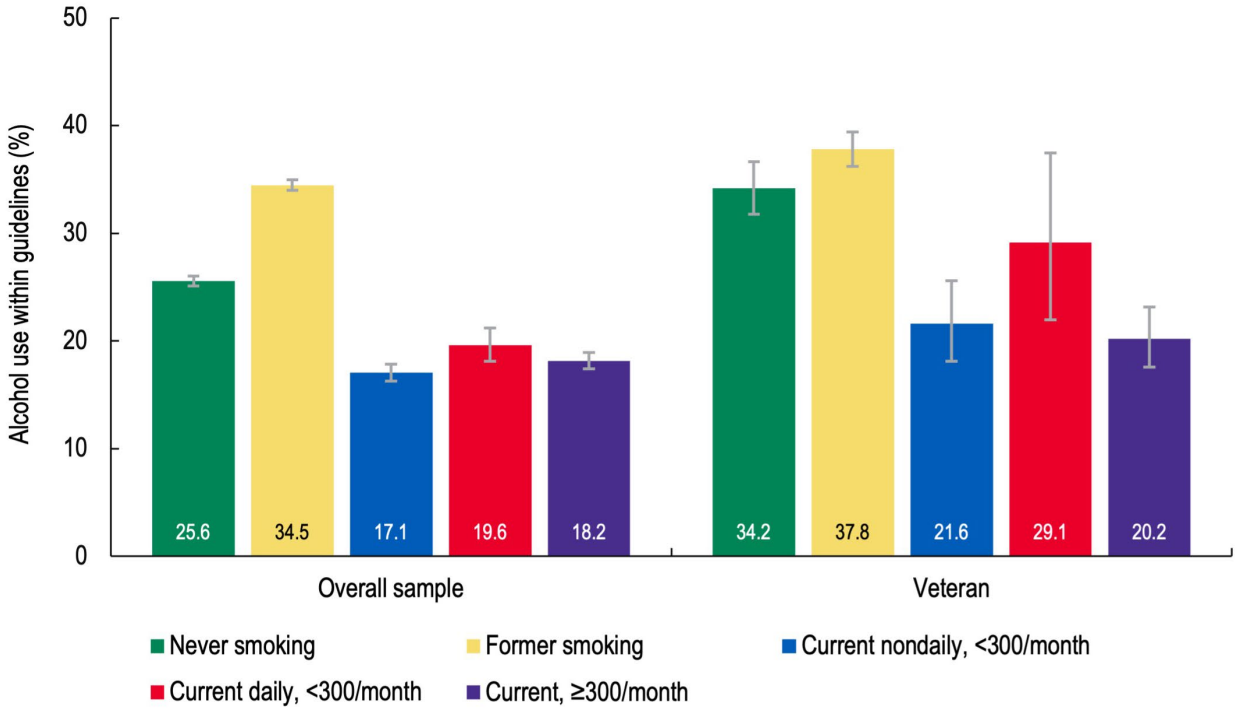
Patterns of tobacco and alcohol co-use among veterans from different racial and/or ethnic groups are not presented in-depth here. However a 2023 study presents data on alcohol consumption and AUD from the Million Veteran Program cohort.[96] This study reports significantly higher levels of diagnosed AUD in Black and Hispanic veterans compared to White veterans at similar levels of self-reported alcohol consumption using AUDIT-C. The authors interpret this result as evidence for underdiagnosis of AUD in White veterans and state, "The absence of other factors to explain this discrepancy strongly suggests the presence of racial and ethnic biases in the diagnosis of AUD."[96,p.434] This misclassification bias has important implications for epidemiological and evaluation studies as well as for potential consequences for careers and employment.

National Survey on Drug Use and Health Analysis: Co-Use by Veteran Status

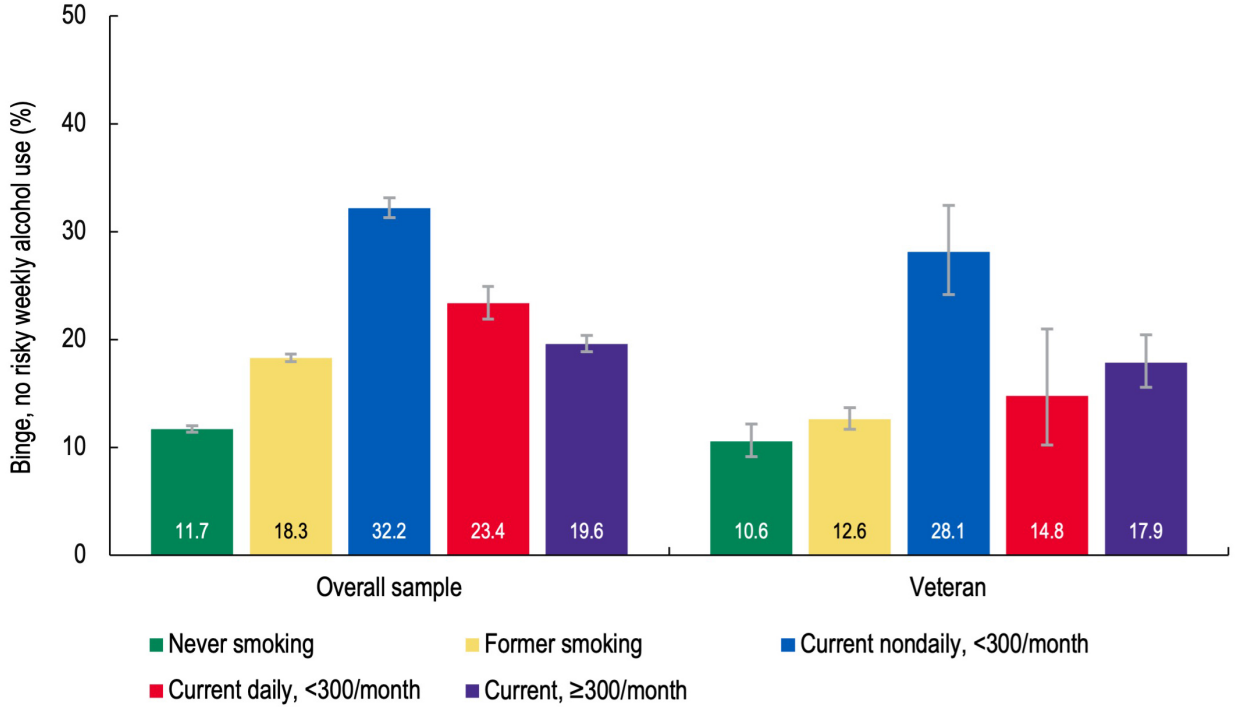
Alcohol use prevalence by cigarette smoking status among U.S. military veterans in the 2015–2019 NSDUH are shown in Figure 3.10 (Appendix Table 3B.24). (The NSDUH sampling frame includes veterans but does not allow examination of tobacco and alcohol use patterns among active-duty military members.) There was a statistically significant bivariate association between tobacco and alcohol use among veteran participants and the overall NSDUH sample ($p < .001$). Overall, the prevalence of risky weekly alcohol use was similar among veterans (9.1%, 95% CI = 8.4%–9.8%) and the overall NSDUH sample (9.3%, 95% CI = 9.2%–9.5%). However, a smaller proportion of veterans overall reported never using alcohol than the overall NSDUH sample (6.6% [95 % CI, 6.0%–7.2%] vs. 14.2% [95% CI, 14.0%–14.5%). As in the NSDUH sample overall, cigarette smoking among veterans was associated with a higher prevalence of binge drinking (without risky weekly alcohol use) and risky weekly alcohol use, particularly among those who smoked nondaily. For example, a higher percentage of veterans reporting nondaily smoking <300 cigarettes/month reported binge drinking (without risky weekly use), compared with veterans who reported never smoking (28.1% [95% CI, 24.2%–32.4%] versus 10.6% [95% CI, 9.1%–12.2%]). Additionally, veterans reporting nondaily smoking <300 cigarettes/month, daily <300 cigarettes/month, and ≥300 cigarettes/month had higher prevalence of risky weekly alcohol use than those who reported never smoking (18.7% [95% CI, 15.3%–22.8%], 11.2% [95% CI, 7.4%–16.8%], and 18.5% [95% CI, 16.1%–21.1%], respectively versus 4.8% [95% CI, 3.8%–6.0%] among veterans who never smoked).

Figure 3.10 Past-30-Day Alcohol Use by Veteran Status and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)

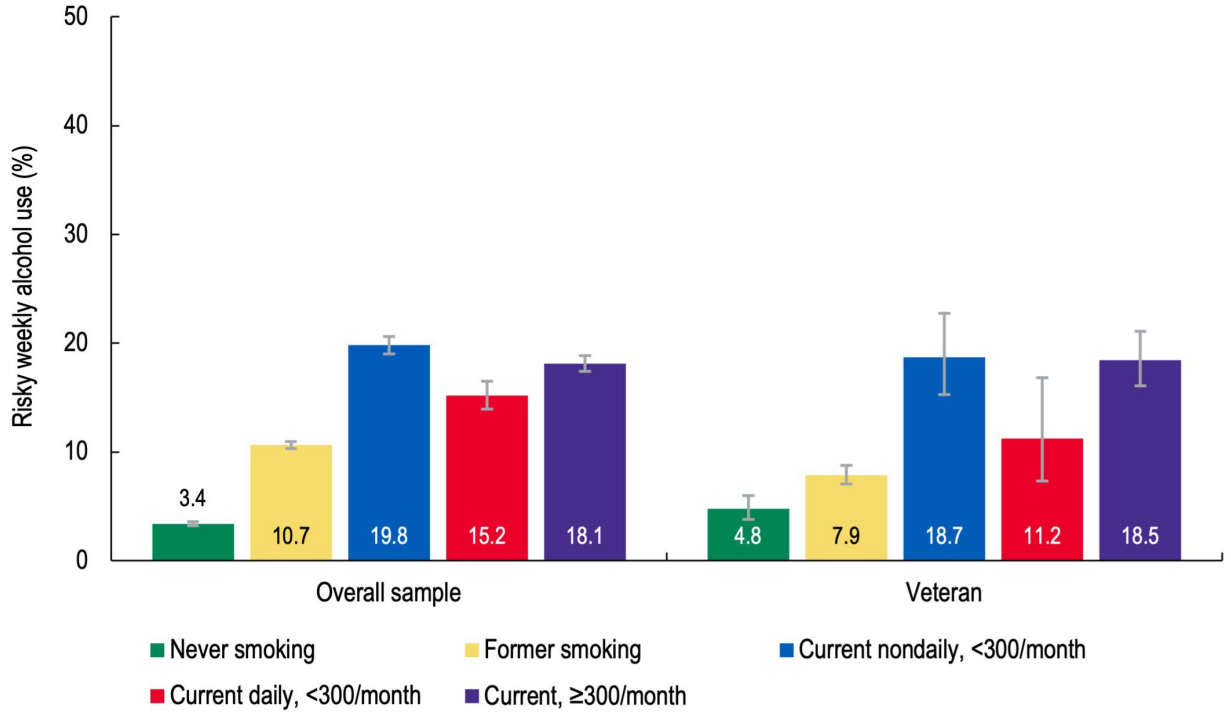
Panel A: Alcohol Use Within Guidelines by Cigarette Smoking Status



Panel B: Binge Drinking (Without Risky Weekly Use) by Cigarette Smoking Status



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



Note. For full data, see Appendix Table 3B.24. *Overall* refers to the total 2015–2019 National Survey on Drug Use and Health sample. *Veterans* are participants who indicated they are "now separated or retired from the military." *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as no binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Summary: Co-Use Among Military Service Members and Veterans

The literature indicates that veterans have higher prevalence of tobacco use and AUD compared to the civilian population. In the 2015–2019 NSDUH, cigarette smoking was associated with a higher prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) among veterans, particularly among those who smoked nondaily, similar to the general population. A smaller proportion of veterans reported never using alcohol than the overall NSDUH sample.

Co-Use Among People with Comorbidities

This section discusses tobacco and alcohol co-use among the following groups with comorbidities: cancer survivors, people with mental health conditions, people with chronic health conditions (heart disease, hypertension, diabetes), people with HIV, and during pregnancy.

Co-Use Among Cancer Survivors

People previously diagnosed with cancer are an important group to consider when examining tobacco and alcohol co-use. This is because continued use of tobacco after a cancer diagnosis increases the risk of cancer-related mortality (and mortality from other diseases), as well as the risk of cancer recurrence, second primary cancers, and adverse treatment related outcomes, such as impaired response to chemotherapy and radiation therapy.[47,97] Additionally, some evidence suggests that alcohol use may worsen response to cancer treatment.[98] Overall, among cancer survivors, co-use is not uncommon and is associated with symptoms of depression and anxiety.[99,100] Polysubstance use in this population further merits attention because cannabis use is associated with tobacco and alcohol use among cancer survivors,[101-104] and cannabis use among cancer survivors may increase if or when more states legalize cannabis for medical and/or recreational purposes.[105,106]

Adolescents and Young Adult Cancer Survivors. In a hospital-based study of 100 participants diagnosed with cancer between the ages 15–39, 15% reported past-year tobacco and alcohol co-use and most participants (65%) were exclusively using alcohol.[99] Compared to the other subgroups, those reporting co-use reported more symptoms of depression and anxiety. A study of 129 childhood cancer survivors 15 to 25 years old examined past-30-day substance use approximately 8 years after an initial cancer diagnosis.[100] Overall, 12.5% reported past-30-day tobacco use, 13.2% reported past-30-day binge drinking (≥5 drinks on 1 occasion), and 12.4% reported polysubstance use (cannabis, tobacco, and binge drinking); co-use of only tobacco and alcohol was not assessed.

Another study of 128 adolescent survivors (ages 14–20) of pediatric cancer explored the beliefs and behaviors associated with substance use.[107] Every participant who reported ever trying cigarette smoking ($n = 31$) also reported ever using alcohol. Conversely, 31 of the 71 participants who reported ever using alcohol had ever smoked a cigarette. Lower knowledge of the risks of smoking or binge drinking was associated with increased likelihood of cigarette and alcohol use, respectively.[107]

National Studies of Adult Cancer Survivors. Several national studies have examined tobacco and alcohol co-use among adult cancer survivors using data from the general population.[108-111] One such large study used data collected between 2018–2022 from the National Institutes of Health *All of Us* Research Program and included 15,199 participants who reported ever being diagnosed with cancer.[111] Overall, 77.7% of participants reported past-year alcohol use, and among those who did report past year alcohol use, 13% exceeded moderate drinking (>2 drinks on a typical day when drinking), 23.8% reported binge drinking (≥6 drinks on 1 occasion), and 38.3% reported hazardous drinking (AUDIT-C scores of 3 or higher for women and 4 or higher for men). Regarding smoking status, 35.1% of participants reported former smoking and 6.6% reported current smoking. In adjusted analyses, former smoking and current smoking (compared to never smoking) were associated with increased likelihood of current drinking, exceeding moderate drinking levels, binge drinking, and engaging in hazardous drinking (aORs ranging from 1.27 to 2.46 for former smoking and 1.44 to 4.14 for current smoking). Further, those

who were younger than 65 years old (versus older), men (versus women), or Hispanic (versus non-Hispanic White) were more likely to report exceeding moderate drinking and binge drinking. A cancer diagnosis before the age of 18 (versus a diagnosis at age 65 or older) predicted elevated risks of binge drinking and hazardous drinking.[111]

Sanford and colleagues (2020)[110] used NHIS data from 2000 to 2017 to examine alcohol use patterns among 34,080 adults reporting a diagnosis of cancer. A total of 56.5% of participants reported current drinking (≥1 drink in the past year). 34.9% of those reporting current drinking also reported exceeding moderate drinking (>1 drink per day for women and >2 drinks per day for men), and 21.0% reported a history of binge drinking (≥5 drinks on ≥1 day during the past year, for both men and women). Binge drinking was more prevalent among those who currently smoked than among those who never smoked (23.6% versus 8.0%, respectively). In adjusted logistic regression models, people who currently or formerly smoked were more likely than those who never smoked to report current drinking (versus never or former drinking), drinking in excess of moderate drinking guidelines (versus not exceeding moderate drinking), and binge drinking (versus not binge drinking).

Another study used data from the 2017–2020 Health Information National Trends Survey to examine risky health behaviors among 2,579 cancer survivors.[109] Former, but not current, cigarette smoking (compared to never smoking) was associated with higher odds of past-30-day moderate/heavy drinking (4–6 or ≥7 drinks per week; aOR = 1.58, 95% CI = 1.04–2.41). In addition, past-30-day moderate/heavy drinking (compared to light drinking, 0–3 drinks per week) was associated with higher odds of current cigarette smoking (aOR = 1.75, 95% CI = 1.22–2.52).

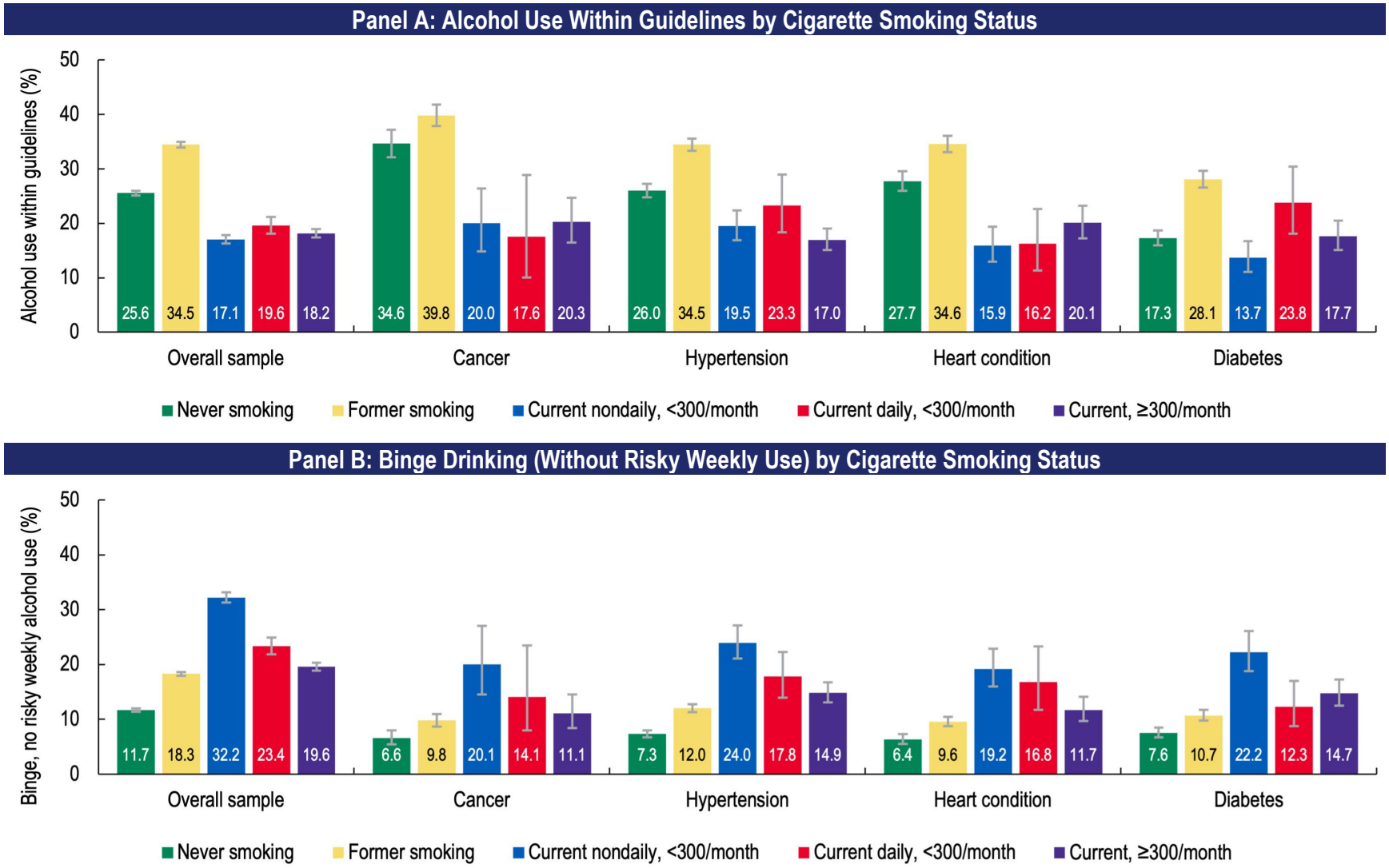
In a study of 1,438 adult survivors of childhood cancer, Asfar and colleagues (2016)[108] pooled data from the 1997–2010 NHIS to identify trends in smoking prevalence and correlates of cigarette smoking. Survivors who reported current drinking (≥12 drinks in the past year) were significantly more likely to report current smoking (defined as smoking every day or some days; aOR = 2.07, 95% CI = 1.42–3.01) than those who reported never or former drinking.

Studies of Adult Cancer Survivors—Data From Health Care Systems, Hospitals, and Cancer Registries. Several U.S. studies have examined tobacco and alcohol co-use by collecting data from health care systems, hospitals, or cancer registries among people with a variety of cancer types. Among 1,926 Black or African American female breast cancer survivors, those who currently smoked at the time of diagnosis reported drinking more drinks per week in the past year than people who formerly or never smoked.[112] Among 517 patients with cancers of the hepatobiliary-pancreatic system, 15.3% reported pre-diagnosis use of both tobacco and alcohol, whereas only 2.3% reported use of both substances after diagnosis.[113] Among 503 people with a lymphoma diagnosis and 1,254 with a metastatic cancer diagnosis admitted to a mid-Atlantic acute health care system, around 4% reported current smoking and an AUD diagnosis.[114]

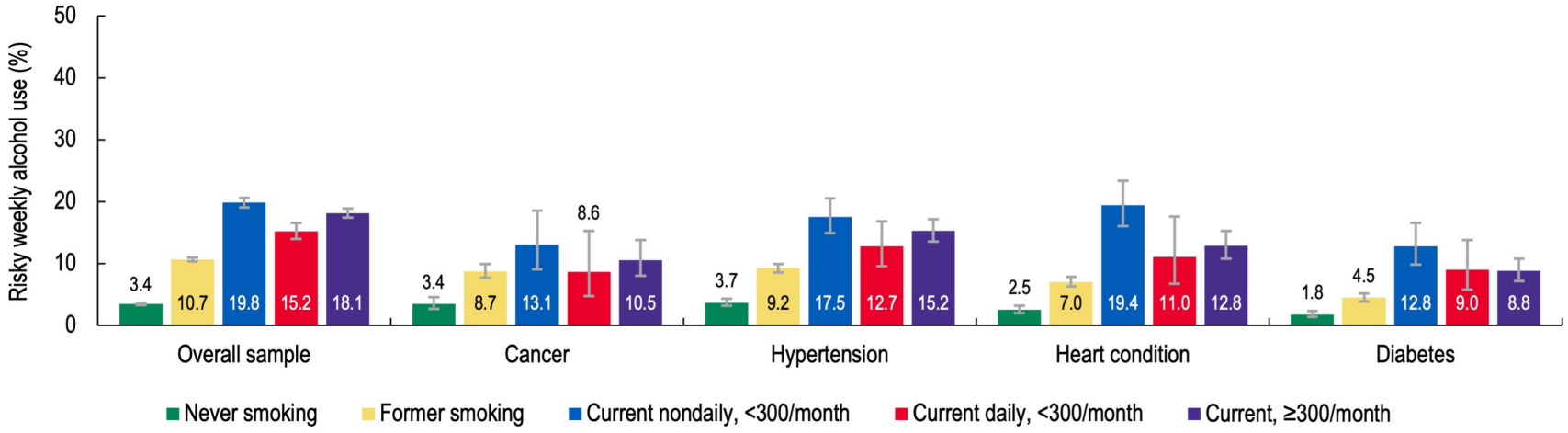
National Survey on Drug Use and Health Analysis: Co-Use by Cancer Diagnosis

Data from the 2015–2019 NSDUH were analyzed to understand alcohol use by cigarette smoking status among people with a cancer diagnosis, defined as reporting that a doctor or other health care professional had ever told participants that they had cancer. There was a statistically significant bivariate association between tobacco and alcohol use among people with a cancer diagnosis and the overall NSDUH sample ($p < .001$). As shown in Figure 3.11 (Appendix Table 3B.25), compared with the overall NSDUH sample, people with a cancer diagnosis had a lower prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly alcohol use) and a higher prevalence of former alcohol use overall and across tobacco use categories. Nonetheless, cigarette smoking was associated with alcohol use exceeding guidelines among cancer survivors. For example, among people with a cancer diagnosis, those reporting nondaily <300 cigarettes/month smoking had the highest prevalence of binge drinking without risky weekly use (20.1%, 95% CI = 14.6%–27.0%) and the highest prevalence of risky weekly alcohol use (13.1%, 95% CI = 9.0%–18.5%), compared with those who reported never smoking (6.6% [95% CI, 5.5%–8.0%] binge drinking without risky weekly use and 3.4% [95% CI = 2.6%–4.5%] risky weekly use) and former smoking (9.8% [95% CI, 8.7%–11.0%] binge drinking without risky weekly use and 8.7% [95% CI, 7.6%–9.9%] risky weekly use). An important caveat is that this analysis could not account for loss to follow-up from cancer-related mortality, which may have resulted in underestimates of substance use prevalence for people with a cancer diagnosis. Further, in the literature, prevalence of any use of alcohol is known to be similar or only slightly lower in cancer survivors than in the U.S. population.[115-117]

Figure 3.11 Past-30-Day Alcohol Use by Health Condition Status and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



Note. For full data, see Appendix Table 3B.25. *Overall* refers to the total 2015–2019 National Survey on Drug Use and Health sample. *Cancer* is defined as a doctor or other health care professional having ever told participants that they had cancer. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as no binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

National Survey on Drug Use and Health Analysis: People with Other Chronic Health Conditions

NSDUH data also were examined to determine prevalence of alcohol use by cigarette smoking status among people who reported hypertension, a heart condition, or diabetes (Figure 3.11; Appendix Table 3B.25). There was a statistically significant bivariate association between tobacco and alcohol use among people with other health conditions and the overall NSDUH sample ($p < .001$). As was true for cancer, people with one of these three chronic health conditions had a lower prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly alcohol use) and a higher prevalence of former alcohol use than people in the overall NSDUH sample. For example, although 9.3% (95% CI, 9.2%–9.5%) of the overall NSDUH sample reported risky weekly alcohol use, only 8.1% (95% CI, 7.7%–8.6%), 6.8% (95% CI, 6.3%–7.4%), and 4.3% (95% CI, 3.9%–4.7%) of those with a history of hypertension, heart disease, or diabetes reported alcohol use exceeding guidelines, respectively. However, similar to the overall NSDUH sample, those who smoked generally had the highest frequency of binge drinking (without risky weekly use) and risky weekly alcohol use across these health conditions. Prevalence of binge drinking in the nondaily <300 cigarettes/month group was 24.0% (95% CI, 21.1%–27.2%), 19.2% (95% CI, 16.0%–22.9%), and 22.2% (95% CI, 18.8%–26.2%) among those with a history of hypertension, heart disease, and diabetes, respectively.

Co-Use Among People with Mental Health Conditions

Substantial literature demonstrates that the prevalence of tobacco use and AUDs are higher among people with mental health conditions than people without.[4,118-121] The 2024 Surgeon General's report concluded:

> People living with any mental health condition or substance use disorder are at increased risk of tobacco use. The intersection of mental health, substance use, and sociodemographic characteristics—such as age, sex, race and/or ethnicity, sexual orientation, and socioeconomic status—adds to the risk of tobacco use.[4,pp.10-11]

This section defines "mental health condition" to include negative affect, internalizing disorders (e.g., depression and anxiety), externalizing disorders (e.g., attention deficit disorder and antisocial personality disorder), or symptoms of mental health disorders (including severe psychiatric illness), but excludes substance use disorders. The extant literature often examines self-reported diagnoses or symptoms from screeners for mental health disorders rather than having met diagnostic criteria.

NSDUH data from 2024 show that prevalence of past-year tobacco and AUD was 30.9% and 18.1%, respectively, among adults with any mental illness, and 20.3% and 8.0%, respectively, among those without a mental illness.[122,123] Although the prevalence and patterns of tobacco and alcohol co-use among people with mental health conditions are less well studied, the limited literature suggests that co-use in this population is common. A study using 2010–2019 NSDUH data found that "high-risk" alcohol use (>7/14 drinks per week for women/men), was more prevalent among adults with a past-year major depressive episode, and among those who reported high-risk alcohol use, a past-year major depressive episode was associated with increased odds of past-month use of both tobacco and cannabis (aOR = 1.38, 95% CI = 1.22–1.56) compared with no past-year major depressive episode.[18] Several studies used 2012–2013 NESARC data to examine the relationship between co-occurring DSM-5 substance

use and psychiatric disorders and cigarette smoking. These analyses found that past-year cigarette smoking was more than twice as prevalent among respondents with co-occurring affective disorders and substance use disorders than those without a co-occurring disorder.[120,124,125] However, AUD was not examined as the specific co-occurring disorder to an affective disorder in these analyses. In a study of 631 college students, a greater proportion of participants with mental health conditions had ever tried e-cigarettes (33.6%) compared to those without mental health conditions (19.2%), and the proportion of past-30-day use of e-cigarettes was highest among those reporting binge drinking (13.5%) compared to moderate drinking (8.4%, respectively) and abstaining (4.6%, respectively).[126] The study further examined alcohol use as a moderator of the association between mental health and e-cigarette use and observed that mental health and alcohol use were each significant predictors of ever using e-cigarettes, but there was no significant interaction between alcohol use and mental health.

A small number of studies have analyzed datasets that measure the use of tobacco, alcohol, and cannabis to investigate the association of different co-use patterns with mental health conditions.[127,128] In one of the largest such studies, data from the 2013–2014 Wave 1 of the PATH study were analyzed to examine various patterns of current alcohol, cannabis, and tobacco product co-use among 9,112 U.S. young adults (ages 18–24) and 23,208 adults (ages 25 and older) and their association with mental health problems as measured by the Global Appraisal of Individual Needs–Short Screener, which included symptoms of internalizing and externalizing disorders.[127] In adjusted logistic regression models, mental health problems among young adults were associated with higher odds of hookah and alcohol co-use (aOR = 1.48, 95% CI = 1.03–2.13) and cigarette, alcohol, and cannabis co-use (aOR = 1.72, 95% CI = 1.19–2.48), but not with cigarette and alcohol co-use (aOR = 1.15, 95% CI = 0.87–1.54), compared to alcohol-only use. In adjusted models among older adults, mental health problems were associated with all tobacco and alcohol product co-use patterns (aORs ranging from 1.19–1.69).[127]

A longitudinal study investigated mental health symptoms in the transition from smoking only tobacco to dual- and polysubstance use of alcohol, cigarettes, and cannabis among 1,263 9th and 10th graders followed over a 7-year period.[128] Mental health symptoms were measured using the Center for Epidemiological Studies-Depression scale and the Mood and Affect Symptoms Questionnaire. Cigarette and alcohol co-use was the most common pattern of dual substance use observed at baseline and most follow-up assessments, accounting for around one-half of those who reported co-using any two substances at most time points. Depression and anxiety symptoms significantly predicted increased cigarette smoking frequency, alcohol use frequency, and polysubstance use over time.

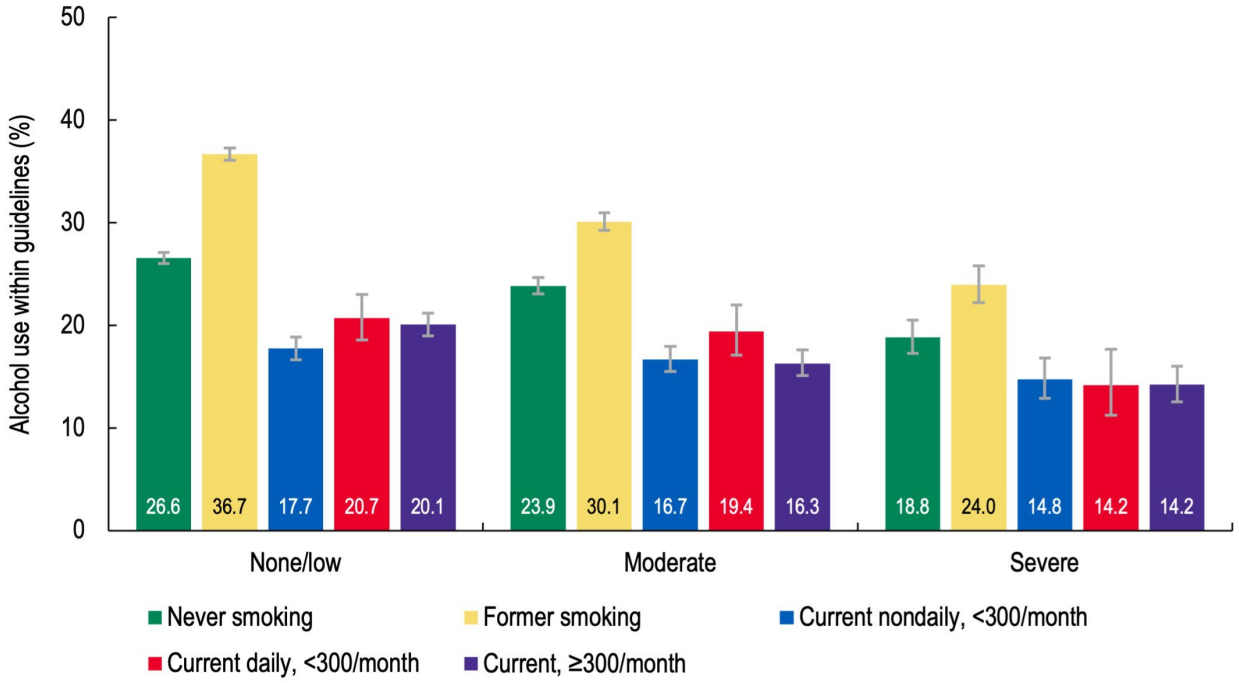
National Survey on Drug Use and Health Analysis: Co-Use by Mental Distress

NSDUH data were analyzed to examine past-30-day alcohol use by cigarette smoking status and level of mental distress (none/low, moderate, or severe) based on scores on the Kessler Psychological Distress Scale–6 items (K6)[129] (Figure 3.12; Appendix Table 3B.26). There was a statistically significant bivariate association between tobacco and alcohol use among all categories of mental distress ($p < .001$). Overall, prevalence of past-30-day alcohol use exceeding guidelines (binge drinking and risky weekly use) was lowest among people who never smoked and highest among people who smoked in the past 30 days, particularly among people who smoked nondaily. Among people who reported past-30-day smoking, the levels of alcohol

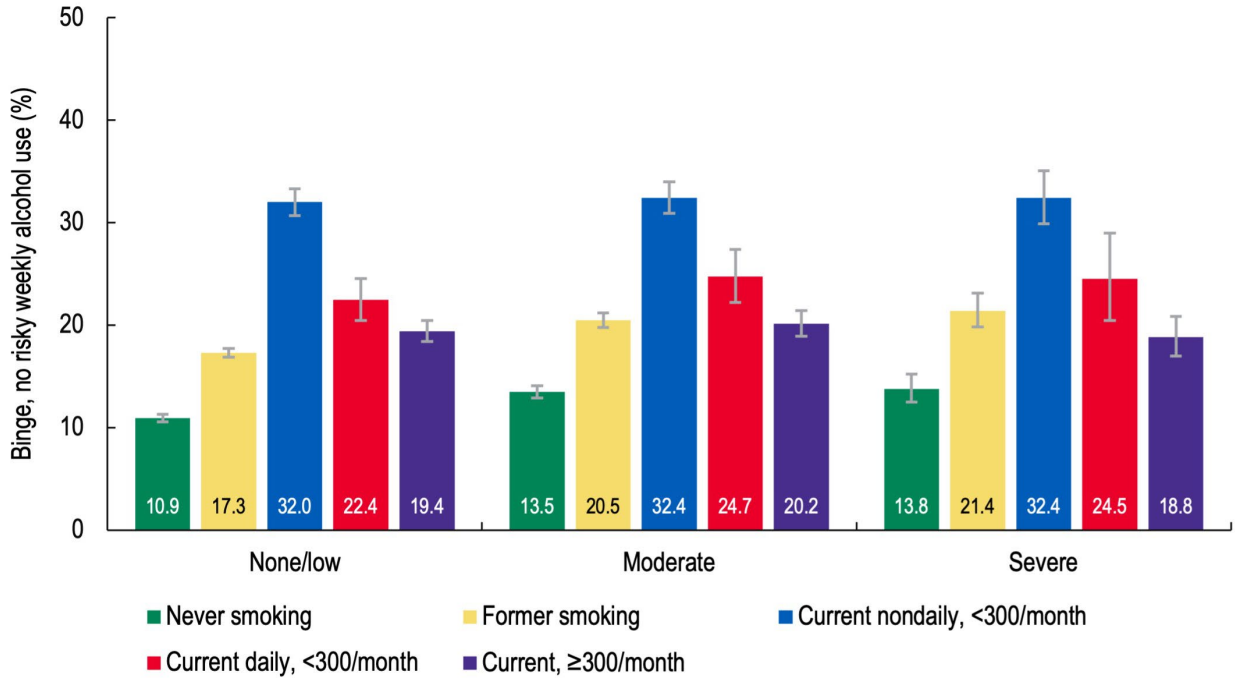
use exceeding guidelines were generally similar across mental distress categories. For example, among those who smoked nondaily, binge drinking was 32.0% (95% CI, 30.7%–33.3%) among those with no/low mental distress, 32.4% (95% CI, 30.9%–34.0%) among those with moderate mental distress, and 32.4% (95% CI, 29.9%–35.1%) among those with severe mental distress. Among people who never or formerly smoked, alcohol use within guidelines was more prevalent among people with no/low mental distress; for example, prevalence of alcohol use within guidelines was 36.7% (95% CI, 36.1%–37.2%), among people with low/no mental distress who formerly smoked, and 24.0% (95% CI, 22.2%–25.8%) among people with severe mental distress who formerly smoked. Around 37% to 56% of people with mental distress who smoke also reported alcohol use exceeding guidelines (binge drinking and risky weekly alcohol use), similar to alcohol use exceeding guidelines among people who smoke in the overall NSDUH sample.

Figure 3.12 Past-30-Day Alcohol Use by Mental Distress and Past-30-Day Cigarette Smoking Status (2015–2019 National Survey on Drug Use and Health; *N* = 210,684)

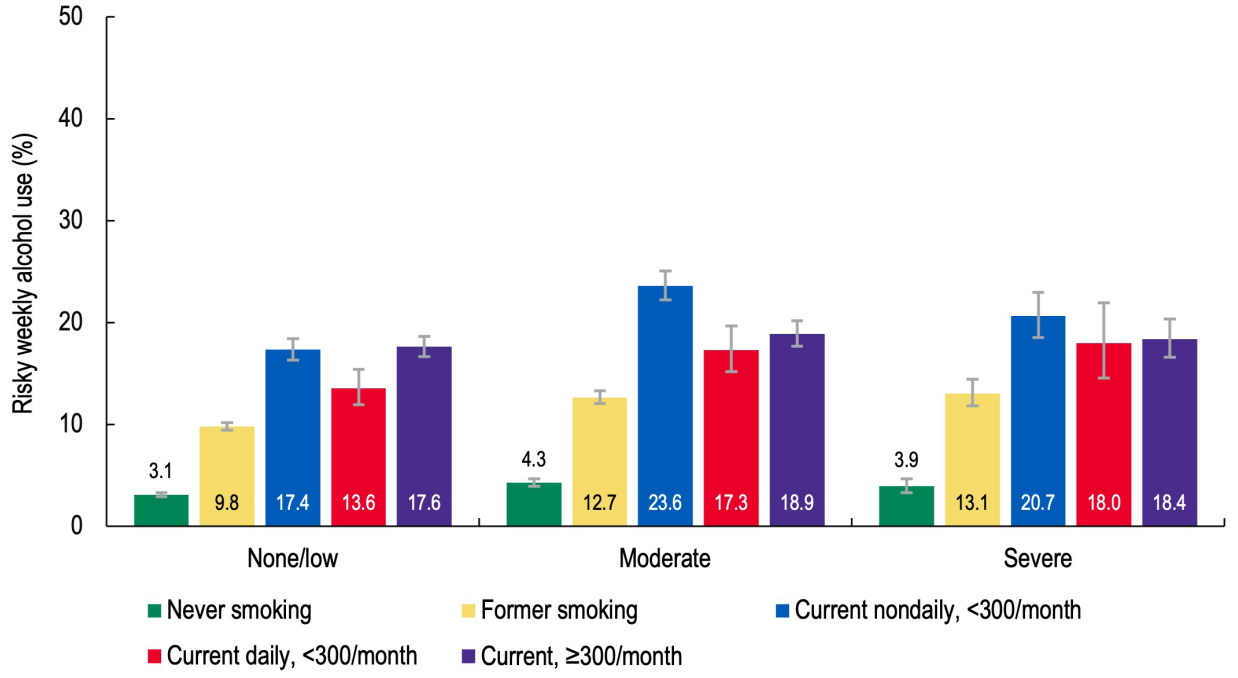
Panel A: Alcohol Use Within Guidelines by Cigarette Smoking Status



Panel B: Binge Drinking (Without Risky Weekly Use) by Cigarette Smoking Status



Panel C: Risky Weekly Alcohol Use by Cigarette Smoking Status



Note. For full data, see Appendix Table 3B.26. *Mental distress* levels are based on scores on the Kessler K6 non-specific distress scale, where a score of 0–4 = none/low, 5–12 = moderate, and 13–24 = severe. *Current cigarette smoking* is defined as use in the past 30 days. Never and former alcohol use (not shown in the charts) are based on lifetime alcohol use and use in the past 30 days. *Alcohol use within guidelines* is defined as no binge drinking or risky weekly use in the past 30 days. *Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. *Risky weekly use* is defined as drinking >7/14 drinks per week for women/men in the past 30 days.
Source: 2015–2019 National Survey on Drug Use and Health.

Co-Use Among People Living with HIV

As treatment for human immunodeficiency virus (HIV) improves, people living with HIV (PLWH) are aging and, therefore, have increased likelihood of developing other health conditions, including cancer. Indeed, non-AIDS-associated cancers are a leading cause of mortality among PLWH,[130] and PLWH may more heavily engage in behaviors that contribute to these cancers than the general population.[131,132] Although PLWH have demonstrated greater tobacco use,[131,132] and higher prevalence of AUD compared to people without HIV,[133] few studies have explored the link between HIV status and co-use of tobacco and alcohol. In a nationally representative sample using data on 31,810 participants from the 2009 Medical Monitoring Project and NHIS,[131] PLWH had more than double the prevalence of current smoking compared to the general population (42.4% vs. 20.6%), and the prevalence of current smoking was higher among those who engaged in past-30-day binge drinking (≥4 drinks per day for women/5 drinks per day for men) compared to those who did not (59.2% vs. 38.9%).

Similar findings were seen in region-specific studies that measured smoking and alcohol use among PLWH. A study examined lifetime alcohol use, measured by lifetime drinking history (measured in kilograms [kg] of alcohol consumed), among 365 PLWH currently receiving care in a Louisiana HIV clinic. Among those with a lifetime drinking history of >600 kg, or the equivalent of drinking ≥4 standard drinks per day for 28 years, 71.4% were currently smoking and 16.1% formerly smoked.[134] In comparison, among those with a lifetime drinking history of <100 kg, 52.3% and 14.2% were currently and formerly smoking, respectively. Similarly, in a study of 313 HIV/hepatitis C coinfected patients under care at a university-affiliated clinic in Alabama, the prevalence of substance use disorders was found to be higher among those who currently smoked cigarettes (44% versus 29% among those who did not currently smoke cigarettes), and those who currently smoked cigarettes were also more likely to report use of both alcohol and other substances (40% versus 21% among those who did not currently smoke cigarettes).[135] Further, those who used both alcohol and other substances had more than 3 times the odds of currently smoking cigarettes (OR = 3.06, p = .011) compared with not using any substances. As both smoking and alcohol use are associated with adverse health consequences among people with HIV,[136-138] these levels of co-use are of particular concern.

Co-Use During Pregnancy

Abstinence from tobacco and alcohol during pregnancy is important for the health of the individual and developing fetus.[47,139] A study of co-use in a 2015–2018 NSDUH sample of pregnant girls and women ages 12–44 found that 9.8% reported drinking in the past 30 days, and 2.7% of participants reported past-30 day drinking and tobacco use.[140] More than one-third of participants (38.2%) who reported past-30-day drinking also reported use of another substance, with tobacco being the most commonly reported substance (28.1%). A study of 1,814 South Dakota American Indian and White mothers in 2014 did not report co-use prevalence but provided details of disparities in tobacco and alcohol use.[141] The study found that smoking during pregnancy was reported by 10.4% of White mothers and 27.1% of American Indian mothers, and alcohol use during pregnancy was 9.5% for White mothers and 4.6% for American Indian mothers, though no differences by racial group in the odds of smoking during pregnancy were observed after adjusting for other demographic factors. In a study of 4,828 pregnant women in 3 largely rural states (Montana, South Dakota, North Dakota), current and past use of tobacco was significantly associated with alcohol use during pregnancy.[142] A study of adolescent girls

and women (ages 12–44 years) in the 2005–2014 NSDUH estimated that 11.5% of pregnant adolescents and 8.7% of pregnant adults reported past-30-day alcohol use, and 23.0% of pregnant adolescents and 14.9% of pregnant adults reported past-30-day tobacco use.[143] Among those who used alcohol, more than one-half (55.8%) of pregnant adolescents and more than one-quarter (27.5%) of pregnant adults reported also using tobacco. Among those who used tobacco, 27.9% of pregnant adolescents and 16.0% of pregnant adults reported also using alcohol. It is worth noting that self-report measures may underestimate the prevalence of use and co-use of tobacco and alcohol during pregnancy; participants might fear negative consequences for reporting use because of social stigma associated with substance use during pregnancy.[144]

Summary: Co-Use Among People with Comorbidities

Smoking is associated with alcohol use and hazardous drinking among people with comorbidities, including cancer survivors, chronic health conditions, mental health conditions, HIV, and pregnancy. In the 2015–2019 NSDUH, nondaily smoking was particularly associated with alcohol use exceeding guidelines among people with comorbidities, compared to never smoking. Past-30-day binge drinking (without risky weekly use) was reported by around 1 in 5 people who smoked nondaily and had a diagnosis of cancer, hypertension, heart disease, and diabetes. Past-30-day risky weekly alcohol use was reported by around 13% of people with diabetes or a cancer diagnosis and almost 20% of people with hypertension or a heart condition. When considering mental distress, prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) among people who reported past-30-day cigarette smoking was similar across mental distress categories.

Co-Use Among Other Populations: Unhoused or Criminal Justice-Involved Individuals and People with Disabilities

Unhoused individuals have very high prevalence of tobacco and alcohol use as well as co-use of both substances.[145-149] In a 2004–2009 study of 754 chronically unhoused people from 11 sites, 80% reported smoking; 56% of those who smoked regularly also reported alcohol abuse or dependence (DSM-IV criteria), significantly more than the 40% of those who did not smoke.[148] Quitting or limiting smoking (self-reported as trying to reduce or use fewer than a certain number of cigarettes per day or week) at 12-month follow-up was associated with reduced alcohol use. Cross-sectional surveys of people experiencing homelessness in Minnesota in 2015 and 2018 found that prevalence of e-cigarette use was 11.6% in 2015 and 14.6% in 2018, and 46.9% of those who used e-cigarettes also used alcohol in 2018.[146] In a 2015 study of 780 female patients at Health Care Clinics for the Homeless in 9 states, current smoking was reported by 54.5% of the sample while 21% reported past year risky drinking.[149] Further, 17.4% reported problem-drinking, 5.5% reported DSM-IV alcohol abuse, and 11.8% reported DSM-IV alcohol dependence. In logistic regression models, cigarette smoking significantly predicted having an AUD (aOR = 1.72, 95% CI = 1.10–2.70).

Only one study was identified for co-use of tobacco and alcohol among individuals with criminal justice system involvement in the past year.[150] Using NSDUH data from 2008 to 2016, this study found prevalence of past-month tobacco use between 60% and 65% among individuals with a history of criminal justice involvement for each year of the study. Among those with past-month cigarette smoking, 23.7% had an AUD.

Similarly, only one study was identified for people with intellectual disabilities.[151] This study included 122 individuals with mild to moderate intellectual disabilities and showed that people with intellectual disabilities who met criteria for misusing alcohol were more likely to smoke cigarettes than people who drank but did not misuse alcohol or who drank no alcohol use at all. No studies were identified of co-use among individuals with other types of disabilities. Additional research about co-use among unhoused individuals, individuals with criminal justice system involvement, and people with disabilities will help to better inform public health efforts to address tobacco- and alcohol-associated health disparities.

Chapter Summary

As summarized in chapter 2, survey data on tobacco and alcohol use in the United States show that individuals commonly engage in both behaviors, with co-use of both tobacco and alcohol reported by 15.6% of adults in the past year, and co-use of both cigarettes and alcohol reported by 11.8% of adults in the past 30 days. Smoking and drinking are strongly related behaviors and co-occur frequently, particularly among those drinking the heaviest and those with alcohol use disorders. This association between use of tobacco and alcohol is present in all or most demographic subgroups, reflecting genetic factors (see chapters 5 and 6), environmental factors (see chapter 9), and cultural factors. Although the prevalence of alcohol use by people who report tobacco use varies, approximately 50% or more of people in various subpopulations who report tobacco use also report risky patterns of alcohol use, and hazardous alcohol use is particularly common among people who smoke cigarettes nondaily.

This chapter presents tobacco and alcohol co-use prevalence estimates for different demographic groups, focusing on race and/or ethnicity; educational attainment; income; geographic location; sexual minority status; veteran status; and presence of comorbidities, including cancer, chronic health conditions, and mental health conditions. These estimates are obtained from bivariate analyses between a demographic variable and tobacco or alcohol use and co-use. Thus, some of the observed patterns might reflect covariation between multiple population characteristics. For example, urban-rural differences could be related to differences in age distribution or race and/or ethnic composition in urban and rural areas. Surveillance of tobacco and alcohol use among specific subgroups is important for considering population-level public health priorities, but it is also important to remain aware that there is substantial within-group heterogeneity in tobacco and alcohol use and co-use. Regardless of this heterogeneity, tobacco and alcohol co-use are a significant public health problem and greater efforts to address them concurrently are needed.

Much of the literature investigating tobacco and alcohol co-use among the different racial and/or ethnic groups that comprise the U.S. population has focused on non-Hispanic Black, Hispanic, and non-Hispanic White people. New analyses presented here of the National Alcohol Surveys and National Survey on Drug Use and Health show that hazardous alcohol use among those who use tobacco is common across racial and/or ethnic groups. Among National Alcohol Survey participants who used tobacco in the past year, more than 55% of Hispanic, Black, and White

individuals ages 18–29 and more than 35% of those ages 30–59 reported past-year alcohol use exceeding guidelines. Among National Survey on Drug Use and Health participants, around one-half of Hispanic, Black, and White individuals who reported past-30-day nondaily cigarette smoking also reported past-30-day alcohol use exceeding guidelines (binge drinking and risky weekly use).

Lower income and lower educational attainment are strongly associated with a higher prevalence of cigarette smoking compared to higher income and education. The relationship between socioeconomic status and alcohol use patterns is more complex. Higher alcohol use is observed among those with more education or higher income, but detrimental patterns of use, such as more heavy episodic drinking, are observed among those with lower income or levels of education. In the National Alcohol Survey analysis, the prevalence of past-year alcohol use exceeding guidelines is most common among those who used tobacco in the past year and had a college degree or who have some college education. This is particularly true for those younger than age 30; for example, 72.5% (95% CI, 61.5%–81.4%) of those ages 18–29 who had some college education and used tobacco in the past year reported alcohol use exceeding guidelines. In the NSDUH analysis, more than one-half of people who smoked nondaily and had some college education or a college degree reported alcohol use exceeding guidelines (binge drinking and risky weekly use).

The existing literature indicates that prevalence of tobacco and alcohol use and types of products used can vary based on geographic areas. Compared to urban areas, studies have found that people in rural areas have higher prevalence of tobacco use and lifetime abstinence from alcohol, but higher likelihood of alcohol use exceeding guidelines among people who drink. Prevalence of past-30-day alcohol use exceeding guidelines in the National Survey on Drug Use and Health was slightly higher among those in metropolitan counties (compared to nonmetropolitan counties) who smoked cigarettes or used smokeless tobacco in the past month. For example, among people who smoked cigarettes nondaily in the past 30 days, 32.9% (95% CI, 31.9%–33.9%) of those in metropolitan counties and 27.9% (95% CI, 25.8%–30.2%) of those in nonmetropolitan counties reported binge drinking (without risky weekly use).

Sexual minority populations report higher prevalence of tobacco use and alcohol use compared to heterosexual individuals. In the National Survey on Drug Use and Health, around one-third of sexual minority individuals reported past-30-day alcohol use exceeding guidelines (binge drinking and risky weekly use) compared to around one-quarter among the overall National Survey on Drug Use and Health sample. Hazardous alcohol use was highest among sexual minority individuals who smoked <300 cigarettes per month, where 33.6% (95% CI, 30.9%–36.5%) of those in this group who smoked nondaily in the past 30 days and 27.5% (95% CI, 23.3%–32.0%) of those who smoked daily also reported binge drinking (without risky weekly use).

Few studies have examined tobacco and alcohol co-use among veterans. The results of the National Survey on Drug Use and Health analysis indicate that, similar to the overall population, cigarette smoking is associated with higher prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use). For example, veterans with nondaily <300 cigarettes/

month smoking, daily <300 cigarettes/month smoking, and ≥300 cigarettes/month smoking had higher prevalence of risky weekly alcohol use (18.7% [95% CI, 15.3%–22.8%], 11.2% [95% CI, 7.4%–16.8%], and 18.5% [95% CI, 16.1%–21.1%], respectively) than those who reported never smoking (4.8%, 95% CI = 3.8%–6.0%). Further, a smaller proportion of veterans reported never using alcohol than the overall National Survey on Drug Use and Health sample.

As with the general population, studies show associations between the use of tobacco and alcohol among cancer survivors. Results of the National Survey on Drug Use and Health analysis indicate that people with a cancer diagnosis have a lower prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) and a higher prevalence of former alcohol use than the overall National Survey on Drug Use and Health sample. However, in the literature, prevalence of any use of alcohol is known to be similar or only slightly lower in cancer survivors than the U.S. population. Cigarette smoking is associated with alcohol use exceeding guidelines among cancer survivors; among cancer survivors who smoked nondaily in the past month, for example, 20.1% (95% CI, 14.6%–27.0%) reported binge drinking (without risky weekly use) compared to 6.6% (95% CI, 5.5%–8.0%) of those who never smoked. National Survey on Drug Use and Health participants with a history of hypertension, heart disease, or diabetes similarly had a higher prevalence of binge drinking among those who smoked nondaily.

People with mental health conditions have also been found to co-use tobacco and alcohol (particularly alcohol use exceeding guidelines) at a higher prevalence than the general population. Among National Survey on Drug Use and Health participants, around 37% to 56% of people with mental distress who smoke also reported alcohol use exceeding guidelines (binge drinking and risky weekly use), similar to people who smoke in the overall National Survey on Drug Use and Health sample. Prevalence of alcohol use exceeding guidelines was generally similar across mental distress categories among people who reported past-30-day cigarette smoking.

Research Needs

More research is needed to characterize co-use of tobacco and alcohol among specific subgroups. Groups where very few studies were found on co-use include immigrant populations, people involved in the criminal justice system, and people with intellectual and physical disabilities. In 2023, people with disabilities were formally designated by the National Institute on Minority Health and Health Disparities as a population experiencing health disparities, underscoring the need for more research.[152] People with disabilities experience discrimination, stigma, and physical and mental health co-morbidities at rates higher than the general population[153,154] and may therefore be especially vulnerable to the negative effects of tobacco and alcohol co-use.

Disaggregated data are further needed to provide a more nuanced understanding of heterogeneity in co-use patterns within demographic groups, including Asian American and other heterogeneous demographic groups. As noted in the 2024 Surgeon General's report:

> Aggregation of data on tobacco product use, disease incidence, and mortality, may mask disparities within population groups, such as within Asian American and Native Hawaiian and Other Pacific Islander groups. Disaggregation of data reporting and oversampling among disparate populations will foster greater understanding of tobacco-related health disparities.[4,p.13]

A further challenge for research on co-use involves temporal and survey specific differences in measures of sociodemographic and other characteristics, limiting comparability across studies. Definitions and use of racial and/or ethnic groups change over time, complicating comparisons to previous research. For example, in 2024, the Office of Management and Budget revised its standards for collecting race and/or ethnicity data, adding "Middle Eastern and North African" as a category[155]; this category was not a response option in the National Survey on Drug Use and Health surveys used for analyses in this chapter. Such changes are naturally necessary as the population characteristics of the United States change and as attention turns to health needs of understudied populations.

A more detailed understanding of co-use patterns among subpopulations is needed. There is a crucial need for basic epidemiological studies of the co-use of tobacco and alcohol in the subgroups discussed in this chapter. Measures should capture current and lifetime use patterns, simultaneous co-use patterns, and product and context details; this will provide a broad and in-depth understanding of co-use patterns in these important populations and changes over time. It is of particular importance to understanding co-use trends over time among specific subgroups that account for an increasing share of the U.S. population, such as Hispanic individuals, individuals with disabilities or mental health conditions, and sexual minority populations. Further stratified or model-based estimates of co-use of tobacco and alcohol would be useful for several demographic groups. For example, cross-tabulation of data by income or education and rurality could help further understand the distribution of risk. Also, studies are needed to analyze tobacco and alcohol use among individuals that belong to multiple subgroups that experience tobacco- and/or alcohol-related health disparities, as risks of co-use and risks of cancer and other negative health outcomes may be compounded by membership in more than one of these defined groups. There is also a critical need for more research on tobacco and alcohol co-use by geographic location to inform prevention and policy efforts, particularly to address harms from alcohol.

Model-based analysis of co-use accounting for multiple covariates is needed to better understand drivers of patterns of co-use. This chapter (and chapter 2) presents bivariate associations between multiple demographic characteristics and patterns of tobacco and alcohol co-use. Demographic and socioeconomic characteristics are correlated, and model-based analysis is needed to better understand how such are jointly associated with patterns of behavior. Such estimates could inform design of interventions, public health needs, and evaluation efforts.

Further research among cancer survivors is warranted. Among cancer survivors, the prevalence of tobacco and alcohol co-use is generally lower than the use of each substance individually. However, co-use is not uncommon and deserves more attention, considering the potential for synergistic effects on cancer outcomes.[156-158] Further, the co-use of tobacco and alcohol appears to vary by cancer type and to decline post-diagnosis. This is likely due in part to whether tobacco and alcohol are known or suspected risk factors for a particular cancer, which has implications for potential differential effects of co-use by cancer type and for recommendations to limit or abstain from both substances after a cancer diagnosis.[98] Although the prevalence of tobacco and alcohol co-use per se is not always reported and differences in use measures make comparisons across studies difficult, there is clearly a positive association between using tobacco and alcohol in this population. Explicitly measuring, analyzing, and reporting co-use will allow for a better understanding of the demographics of who is engaging in co-use and refine investigations of the effect of tobacco and alcohol use on cancer-related outcomes.

Conclusions

1. **Tobacco use is positively associated with alcohol use.** Analyses of various population subgroups in the National Survey on Drug Use and Health found a statistically significant bivariate association between tobacco use and alcohol use across all subgroups, including all racial and/or ethnic groups examined.
2. **Hazardous alcohol use patterns (alcohol use exceeding guidelines, binge drinking, or risky weekly alcohol use) were most prevalent among people who used tobacco nondaily.** This association was consistent across multiple demographic groups. For example, among National Survey on Drug Use and Health participants, around one-half of Hispanic, Black, and White individuals who reported past-30-day nondaily cigarette smoking also reported past-30-day alcohol use exceeding guidelines (binge drinking and risky weekly use). Furthermore, number of drinks consumed in the past year is generally higher or much higher for people who use tobacco nondaily or daily than those who never or formerly used tobacco, particularly among Hispanic adults ages 30–59, Black adults ages 18–59, and White adults of all ages.
3. **Prevalence of tobacco use is higher in people with lower income and educational attainment, but co-use of tobacco and alcohol is more likely among people with higher incomes and educational attainment than people with lower incomes and educational attainment.** For example, alcohol use is more common among people who use tobacco and have some college education, a college degree, or income of $50,000 or higher compared to people with lower educational attainment or lower income. Further, among people who use tobacco and have some college education or a college degree, alcohol use exceeding 2020–2025 U.S. Dietary Guidelines is reported by more than two-thirds of people ages 18–29. These countervailing trends can influence estimated prevalence of co-use depending on the distribution of income or education in a demographic or geographic group.

4. **There is geographic variation in the United States in prevalence of tobacco and alcohol use and co-use**. Compared to people in urban areas, the literature shows that people in rural areas have higher tobacco use prevalence, higher lifetime abstinence from alcohol, and higher likelihood of alcohol use exceeding guidelines among people who drink. Analysis of 2015–2019 National Survey on Drug Use and Health data finds that among people who reported current use of cigarettes or used smokeless tobacco, alcohol use exceeding guidelines (binge drinking and risky weekly use) is slightly higher among people in metropolitan counties compared to people in nonmetropolitan counties.
5. **Tobacco and alcohol co-use prevalence is higher in some sexual minority subgroups compared with the general population**. However, survey data do not always sample these populations in sufficient detail to distinguish among subsets of these demographic groups.
6. **Veterans have a higher prevalence of both tobacco use and alcohol use disorder compared to the civilian population**. Analysis of 2015–2019 National Survey on Drug Use and Health data finds that, among veterans, cigarette smoking is associated with a higher prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) compared with not smoking. Similar to the general population, the prevalence of alcohol use is particularly high among veterans who smoked nondaily.
7. **Cancer survivors who reported past-month smoking were more likely to use alcohol compared with those who did not report past-month smoking.** Overall, the prevalence of any alcohol use among cancer survivors is similar to or only a little lower than that of the general population; current alcohol use and alcohol use exceeding guidelines are common among cancer survivors. Additionally, cigarette smoking is associated with alcohol use exceeding guidelines (binge drinking and risky weekly use) among cancer survivors. Younger cancer survivors and men are more likely to engage in heavy drinking and binge drinking than other cancer survivors.
8. **People with mental health conditions have a higher prevalence of tobacco and alcohol use than the general population**. Mental distress, tobacco use, and alcohol use are interconnected. At all levels of mental distress, people who smoked cigarettes were more likely to engage in hazardous drinking behaviors than people who did not smoke. Among National Survey on Drug Use and Health participants who reported past-30-day cigarette smoking, the prevalence of alcohol use exceeding guidelines (binge drinking and risky weekly use) was similar across mental distress categories (around 37%–56%).

Appendix 3A Literature Search Strategy

The following search terms were used in PubMed, Google Scholar, Ovid, and Scopus between June 2023 and September 2024 to identify literature regarding co-use of tobacco and alcohol among specific subpopulations.

Co-use search terms:

- Alcohol, drinking, beer, wine, spirits, drinker, drinking patterns
- Tobacco, cigarette, smoking, smoker, co-use, simultaneous use, co-use, co-user, tobacco product, e-cigarette, vaping, polysubstance use

Subpopulation search terms:

- Race, ethnicity, Black, African American, Hispanic, Latino, Latina, Latinx, Asian, Indigenous, American Indian, Native American, Alaska Native, Pacific Islander
- Age, young, adolescent, youth, young adult, adult, middle age, older
- Gender, sex, men, women, male, female
- Sexual minority, LGBTQ+, gay, lesbian, bisexual, queer
- Income, poverty, socioeconomic status
- Education, high school, some college, college graduate, college
- Unhoused, homeless
- Regional, urban, state, area, rural, spatial, geospatial, location
- Pregnant, pregnancy, birthing
- Cohort, trends, trends over time, prospective, mortality, morbidity
- Cancer, cancer survivors, malignancies, cancer patients, hypertension, diabetes, heart disease, heart problems
- Mental health symptoms, mental health problems, mental health disorders, depression, depressive symptoms, anxiety, anxious symptoms, negative affect, negative mood
- HIV, people with HIV
- Disability, disabilities

Additional studies published after 2024 were incorporated into the chapter based on their relevance.

Appendix 3B National Alcohol Surveys and National Survey on Drug Use and Health Tables

Table 3B.1 Past-Year Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Race and/or Ethnicity, Stratified by Age *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
Hispanic, Latino/a, or of Spanish origin												
Overall (2,656)	33.7 (28.6, 39.2)	28.2 (28.2, 33.6)	38.1 (32.7, 43.7)		33.1 (29.5, 37.0)	39.9 (36.2, 43.8)	27.0 (23.6, 30.6)		45.4 (38.3, 52.8)	38.0 (31.2, 45.2)	16.6 (12.2, 22.1)	
Never (1,961)	41.0 (34.9, 47.4)	28.3 (22.9, 34.4)	30.7 (25.3, 36.7)	<.001	37.5 (33.0, 42.1)	42.4 (37.9, 47.0)	20.1 (16.6, 24.2)	<.001	56.2 (46.9, 65.1)	30.6 (23.0, 39.4)	13.2 (8.2, 20.6)	<.001
Former (318)	*	50.6 (28.1, 73.0)	28.2 (12.2, 52.6)		24.4 (15.7, 36.0)	39.0 (28.7, 50.5)	36.5 (26.3, 48.1)		19.9 (11.4, 32.3)	58.9 (44.8, 71.7)	21.2 (12.5, 33.6)	
Current (past-year) (377)	6.8 (3.5, 13.1)	24.0 (13.1, 39.7)	69.2 (53.9, 81.2)		24.4 (16.7, 34.2)	27.7 (20.9, 35.7)	48.0 (38.9, 57.2)		48.3 (30.7, 66.4)	28.5 (15.6, 46.3)	23.2 (12.3, 39.4)	
Nondaily (132)	*	28.0 (12.8, 50.6)	61.6 (40.1, 79.3)		*	21.0 (12.2, 33.7)	66.7 (51.2, 79.3)		*	*	*	
Daily (245)	*	*	77.1 (54.1, 90.6)		28.7 (19.1, 40.8)	30.1 (21.6, 40.1)	41.2 (30.7, 52.6)		47.8 (28.8, 67.4)	28.1 (14.3, 47.9)	24.1 (12.2, 42.0)	
Black or African American												
Overall (2,937)	32.9 (27.0, 39.4)	31.2 (25.8, 37.2)	35.9 (29.9, 42.4)		35.0 (31.3, 38.9)	43.0 (39.1, 47.0)	22.0 (18.8, 25.6)		55.9 (49.9, 61.6)	34.2 (28.8, 39.9)	10.0 (7.0, 14.1)	
Never (1,946)	37.2 (30.4, 44.5)	30.6 (24.8, 37.2)	32.2 (26.0, 39.1)	.001	38.6 (34.1, 43.3)	44.8 (40.1, 49.5)	16.7 (13.1, 20.9)	<.001	62.1 (54.4, 69.3)	33.4 (26.5, 41.1)	4.5 (2.8, 7.0)	<.001
Former (415)	*	*	*		29.3 (19.7, 41.2)	48.4 (36.1, 61.0)	22.3 (12.5, 36.6)		60.8 (49.8, 70.8)	32.0 (22.7, 42.9)	7.2 (3.6, 13.8)	
Current (past-year) (576)	17.5 (8.5, 32.6)	25.0 (14.2, 40.0)	57.6 (41.1, 72.5)		27.6 (20.0, 36.8)	36.9 (28.8, 45.9)	35.5 (27.9, 43.8)		30.4 (18.6, 45.6)	40.2 (26.8, 55.2)	29.4 (17.5, 45.0)	
Nondaily (134)	*	*	60.8 (33.8, 82.5)		7.8 (3.6, 16.1)	43.4 (28.5, 59.6)	48.8 (33.1, 64.7)		*	47.6 (25.3, 71.0)	*	
Daily (442)	*	29.1 (14.9, 48.9)	56.0 (35.8, 74.4)		32.5 (23.3, 43.2)	35.3 (26.1, 45.8)	32.2 (24.1, 41.6)		31.1 (18.1, 48.1)	39.2 (24.6, 56.0)	29.7 (16.6, 47.2)	

Table 3B.1 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use[b]	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
White												
Overall (6,971)	22.2 (18.9, 26.0)	25.1 (21.5, 29.2)	52.7 (48.2, 57.0)		23.8 (21.9, 25.8)	39.2 (37.0, 41.4)	37.0 (34.8, 39.3)		36.9 (34.5, 39.3)	48.8 (46.4, 51.3)	14.3 (12.6, 16.1)	
Never (4,100)	26.0 (21.7, 30.9)	24.9 (20.6, 29.7)	49.1 (43.8, 54.4)	.08	25.8 (23.3, 28.5)	42.0 (39.2, 44.9)	32.2 (29.4, 35.1)	<.001	43.3 (39.9, 46.8)	47.0 (43.6, 50.4)	9.7 (7.9, 11.8)	<.001
Former (1,614)	*	26.2 (13.4, 45.0)	57.2 (38.9, 73.8)		20.2 (16.4, 24.8)	43.4 (38.1, 48.8)	36.4 (31.4, 41.7)		29.2 (25.8, 32.9)	53.2 (49.1, 57.3)	17.6 (14.6, 21.1)	
Current (past-year) 1,257)	14.6 (9.3, 22.1)	25.3 (18.2, 34.2)	60.1 (50.8, 68.7)		21.9 (18.3, 25.9)	29.3 (25.0, 34.0)	48.8 (43.9, 53.7)		33.4 (27.2, 40.3)	45.0 (38.1, 52.1)	21.6 (16.5, 27.8)	
Nondaily (273)	*	16.6 (8.2, 30.5)	78.5 (64.3, 88.1)		16.3 (9.8, 25.8)	27.5 (18.8, 38.3)	56.2 (45.0, 66.9)		21.7 (11.1, 38.1)	60.0 (43.3, 74.8)	18.2 (9.4, 32.4)	
Daily (984)	18.4 (11.4, 28.4)	28.9 (19.8, 40.0)	52.7 (41.7, 63.4)		23.0 (19.0, 27.6)	29.7 (24.9, 35.0)	47.3 (41.9, 52.8)		35.4 (28.5, 43.0)	42.4 (35.0, 50.1)	22.2 (16.5, 29.2)	

Note. CI = confidence interval. Black and White participants are non-Hispanic. Data are visualized in Figure 3.1.
[a]*Tobacco* use is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. *Former tobacco use* is defined as use within lifetime but not in the past year. *Never tobacco use* is defined as never use in lifetime. [b]Alcohol use is measured in the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking at least 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[22] and NIAAA's definition of heavy alcohol use.[23] [c]*p* value for bivariate association, obtained through a chi-squared test, between tobacco use categories (never, former, and current [past-year]) and alcohol use categories within each age and race and/or ethnicity group.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.2 Past-Year Hazardous Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Race and/or Ethnicity, Stratified by Age *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
Hispanic, Latino/a, or of Spanish origin																		
Overall (2,734)	70.3 (64.9, 75.1)	29.7 (24.9, 35.1)		89.2 (85.2, 92.3)	10.8 (7.7, 14.8)		79.0 (75.6, 82.1)	21.0 (17.9, 24.4)		95.2 (93.6, 96.4)	4.8 (3.6, 6.4)		91.7 (87.5, 94.6)	8.3 (5.4, 12.5)		98.6 (96.7, 99.4)	*	
Never (2,020)	77.2 (71.7, 81.9)	22.8 (18.1, 28.3)	<.001	92.9 (88.8, 95.6)	7.1 (4.4, 11.2)	<.001	84.5 (80.7, 87.8)	15.5 (12.2, 19.3)	<.001	97.2 (95.6, 98.2)	2.8 (1.8, 4.4)	<.001	92.9 (86.4, 96.4)	7.1 (3.6, 13.6)		99.6 (98.9, 99.9)	*	.02
Former (329)	78.1 (53.0, 91.9)	*		92.3 (77.1, 97.7)	*		74.5 (62.9, 83.5)	25.5 (16.5, 37.1)		96.5 (93.1, 98.3)	3.5 (1.7, 6.9)		90.9 (83.2, 95.2)	9.1 (4.8, 16.8)		98.5 (90.3, 99.8)	*	
Current (past-year) (385)	39.2 (25.6, 54.6)	60.8 (45.4, 74.4)		70.3 (54.3, 82.5)	29.7 (17.5, 45.7)		57.8 (48.4, 66.6)	42.2 (33.4, 51.6)		85.2 (78.1, 90.3)	14.8 (9.7, 21.9)		87.9 (73.8, 94.9)	12.1 (5.1, 26.2)		94.4 (82.4, 98.4)	*	
Nondaily (135)	41.0 (22.8, 61.9)	59.0 (38.1, 77.2)		89.0 (76.1, 95.4)	*		42.7 (28.5, 58.3)	57.3 (41.7, 71.5)		80.7 (64.4, 90.6)	19.3 (9.4, 35.6)		100.0	*		100.0	*	
Daily (250)	37.3 (19.1, 60.0)	62.7 (40.0, 80.9)		50.7 (29.1, 72.0)	49.3 (28.0, 70.9)		63.3 (51.9, 73.4)	36.7 (26.6, 48.1)		86.9 (78.6, 92.2)	13.1 (7.8, 21.4)		86.3 (70.7, 94.3)	13.7 (5.7, 29.3)		93.7 (80.3, 98.2)	*	
Black or African American																		
Overall (3,041)	72.2 (65.9, 77.7)	27.8 (22.3, 34.1)		90.8 (85.8, 94.1)	9.2 (5.9, 14.2)		84.8 (81.5, 87.6)	15.2 (12.4, 18.5)		96.3 (94.5, 97.5)	3.7 (2.5, 5.5)		95.8 (92.8, 97.6)	4.2 (2.4, 7.2)		99.6 (99.2, 99.9)	0.4 (0.2, 0.8)	
Never (2,014)	74.9 (68.4, 80.5)	25.1 (19.5, 31.6)	.004	93.7 (89.9, 96.2)	6.3 (3.8, 10.1)	<.001	88.5 (84.7, 91.5)	11.5 (8.5, 15.3)	.005	97.0 (94.6, 98.4)	3.0 (1.6, 5.4)	.27	98.8 (97.5, 99.5)	1.2 (0.5, 2.5)	<.001	99.5 (98.6, 99.8)	*	.68
Former (434)	97.6 (82.6, 99.7)	*		97.6 (82.6, 99.7)	*		82.9 (67.8, 91.8)	17.1 (8.2, 32.2)		96.5 (88.4, 99.0)	*		97.9 (94.1, 99.2)	*		99.8 (99.0, 100.0)	*	
Current (past-year) (593)	53.4 (36.5, 69.5)	46.6 (30.5, 63.5)		75.2 (54.8, 88.4)	24.8 (11.6, 45.2)		76.1 (68.5, 82.3)	23.9 (17.7, 31.5)		94.4 (90.5, 96.7)	5.6 (3.3, 9.5)		84.0 (70.2, 92.1)	16.0 (7.9, 29.8)		99.7 (97.7, 100.0)	*	
Nondaily (138)	54.7 (27.3, 79.6)	45.3 (20.4, 72.7)		71.5 (35.4, 92.0)	*		66.4 (50.3, 79.5)	33.6 (20.5, 49.7)		88.6 (75.2, 95.2)	*		89.2 (66.6, 97.2)	*		100.0	*	
Daily (455)	52.7 (32.3, 72.3)	47.3 (27.7, 67.7)		77.0 (51.4, 91.4)	*		78.5 (69.8, 85.2)	21.5 (14.8, 30.2)		95.8 (91.9, 97.9)	4.2 (2.1, 8.1)		83.3 (67.6, 92.2)	16.7 (7.8, 32.4)		99.6 (97.4, 100.0)	*	

Table 3B.2 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
White																		
Overall (7,088)	56.8 (52.3, 61.1)	43.3 (38.9, 47.7)		85.0 (81.6, 87.9)	15.0 (12.1, 18.4)		72.2 (70.0, 74.3)	27.8 (25.8, 30.0)		92.5 (91.2, 93.7)	7.5 (6.3, 8.8)		92.5 (91.2, 93.6)	7.5 (6.4, 8.8)		98.7 (98.0, 99.1)	1.3 (1.0, 2.0)	
Never (4,170)	61.2 (55.9, 66.2)	38.8 (33.8, 44.1)	.008	87.6 (83.8, 90.7)	12.4 (9.3, 16.2)	.005	76.5 (73.8, 79.0)	23.5 (21.0, 26.2)	<.001	94.3 (92.6, 95.7)	5.7 (4.3, 7.4)	<.001	95.5 (94.0, 96.6)	4.6 (3.4, 6.0)	<.001	99.1 (98.3, 99.5)	0.9 (0.5, 1.7)	.004
Former (1,641)	57.7 (40.5, 73.2)	42.3 (26.9, 59.5)		91.9 (82.3, 96.5)	8.1 (3.5, 17.7)		74.1 (69.2, 78.4)	26.0 (21.6, 30.8)		95.1 (92.1, 97.0)	4.9 (3.0, 7.9)		91.5 (89.2, 93.3)	8.5 (6.7, 10.8)		99.0 (97.9, 99.5)	1.0 (0.5, 2.1)	
Current (past-year) (1,277)	45.9 (37.0, 55.1)	54.1 (44.9, 63.0)		77.5 (69.1, 84.2)	22.5 (15.8, 30.9)		60.7 (55.7, 65.4)	39.3 (34.6, 44.3)		86.5 (82.9, 89.5)	13.5 (10.5, 17.1)		85.4 (79.9, 89.6)	14.6 (10.4, 20.1)		96.5 (93.1, 98.3)	3.5 (1.7, 6.9)	
Nondaily (274)	28.1 (16.0, 44.5)	71.9 (55.5, 84.0)		76.4 (61.2, 87.0)	23.6 (13.1, 38.8)		56.8 (45.3, 67.6)	43.3 (32.4, 54.7)		87.9 (77.9, 93.8)	12.1 (6.2, 22.1)		89.2 (76.6, 95.4)	10.8 (4.6, 23.4)		98.6 (94.0, 99.7)	1.4 (0.3, 6.0)	
Daily (1,003)	53.0 (42.0, 63.6)	47.1 (36.4, 58.0)		78.0 (67.4, 85.8)	22.0 (14.2, 32.6)		61.4 (56.0, 66.6)	38.6 (33.4, 44.0)		86.2 (82.2, 89.5)	13.8 (10.5, 17.8)		84.8 (78.5, 89.4)	15.3 (10.6, 21.5)		96.2 (92.2, 98.2)	3.8 (1.8, 7.8)	

Note. CI = confidence interval; HID = high intensity drinking. Black and White participants are non-Hispanic.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. [b]Alcohol use is measured in the past year. 5+ drinks is defined as consuming five or more drinks in one day. [c]Alcohol use is measured within the past year. *HID* defined as drinking ≥ 8/12 drinks in one day for women/men. [d]*p* value for bivariate association, obtained through a chi-squared test, between tobacco and alcohol use categories within each age and race and/or ethnicity group.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.3 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), by Race and/or Ethnicity *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Hispanic						
Overall (36,035)	21.6 (20.9, 22.3)	32.6 (31.8, 33.4)	18.8 (18.2, 19.5)	21.2 (20.6, 21.9)	5.8 (5.4, 6.1)	
Never (18,628)	36.8 (35.7, 37.9)	31.3 (30.3, 32.3)	15.8 (15.0, 16.7)	14.1 (13.3, 14.8)	2.1 (1.8, 2.4)	<.001
Former (11,351)	5.1 (4.5, 5.9)	36.5 (35.1, 37.9)	25.5 (24.2, 26.8)	26.2 (25.0, 27.4)	6.7 (6.1, 7.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (3,814)	6.3 (5.2, 7.6)	24.0 (21.8, 26.2)	12.9 (11.3, 14.6)	40.9 (38.5, 43.3)	16.0 (14.4, 17.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (920)	9.3 (6.5, 13.1)	36.0 (31.4, 40.9)	17.0 (13.2, 21.7)	25.1 (21.3, 29.4)	12.6 (10.0, 15.7)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,322)	8.4 (5.8, 12.2)	34.3 (30.5, 38.3)	15.0 (12.3, 18.2)	24.5 (21.1, 28.1)	17.9 (15.0, 21.1)	
Black						
Overall (26,448)	22.5 (21.8, 23.3)	32.1 (31.2, 32.9)	21.1 (20.4, 21.8)	18.1 (17.4, 18.7)	6.3 (5.9, 6.7)	
Never (14,510)	35.9 (34.7, 37.1)	29.8 (28.7, 31.0)	19.3 (18.4, 20.2)	12.6 (11.9, 13.4)	2.3 (2.0, 2.7)	<.001
Former (6,234)	7.2 (6.4, 8.2)	39.5 (37.8, 41.3)	27.2 (25.6, 28.7)	19.4 (18.1, 20.8)	6.7 (5.9, 7.5)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (3,113)	9.8 (8.4, 11.3)	26.8 (24.7, 29.1)	16.0 (14.4, 17.7)	34.4 (32.1, 36.7)	13.1 (11.5, 14.9)	
Current (past-30-day) daily smoking, <300 cigarettes/month (1,104)	8.8 (6.7, 11.3)	28.2 (24.5, 32.1)	21.2 (18.0, 24.7)	26.3 (23.1, 29.9)	15.6 (12.9, 18.8)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,487)	8.6 (6.8, 10.8)	29.3 (26.1, 32.7)	17.4 (15.0, 20.2)	24.4 (21.8, 27.4)	20.2 (17.8, 22.9)	

Table 3B.3 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
White						
Overall (127,134)	9.1 (8.9, 9.3)	31.5 (31.1, 31.9)	31.7 (31.4, 32.1)	16.3 (16.1, 16.6)	11.4 (11.1, 11.6)	
Never (41,808)	21.4 (20.8, 21.9)	29.9 (29.2, 30.5)	32.5 (31.8, 33.2)	11.5 (11.1, 11.9)	4.8 (4.5, 5.1)	<.001
Former (54,507)	3.1 (2.9, 3.3)	31.2 (30.6, 31.7)	36.9 (36.3, 37.5)	16.8 (16.4, 17.3)	12.1 (11.7, 12.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (11,243)	5.1 (4.5, 5.7)	24.0 (22.8, 25.2)	18.3 (17.3, 19.4)	29.0 (27.9, 30.2)	23.7 (22.6, 24.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (3,223)	5.0 (4.0, 6.3)	37.2 (34.8, 39.6)	20.4 (18.4, 22.5)	21.8 (19.8, 23.8)	15.7 (14.1, 17.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (16,353)	4.4 (3.9, 4.9)	40.3 (39.2, 41.3)	18.5 (17.7, 19.4)	18.7 (17.9, 19.5)	18.1 (17.3, 18.9)	
American Indian, Alaska Native, Native Hawaiian, or Other Pacific Islander						
Overall (4,060)	22.6 (20.0, 25.5)	39.2 (36.5, 42.0)	15.1 (13.1, 17.3)	15.8 (14.1, 17.6)	7.3 (6.3, 8.6)	
Never (1,295)	45.2 (40.1, 50.4)	28.8 (24.6, 33.5)	14.1 (10.8, 18.3)	9.3 (7.3, 11.9)	2.5 (1.4, 4.3)	<.001
Former (1,305)	7.8 (4.9, 12.3)	48.7 (43.8, 53.7)	20.1 (16.5, 24.4)	16.7 (13.7, 20.1)	6.7 (5.0, 8.9)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (692)	11.4 (7.9, 16.2)	32.9 (26.9, 39.5)	10.9 (7.4, 15.7)	29.0 (23.9, 34.7)	15.8 (12.3, 20.1)	
Current (past-30-day) daily smoking, <300 cigarettes/month (239)	11.9 (4.5, 27.6)	45.4 (34.0, 57.3)	10.4 (5.3, 19.2)	17.7 (10.2, 29.0)	14.8 (7.5, 26.9)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (529)	8.6 (5.1, 14.3)	49.2 (42.2, 56.2)	9.5 (5.7, 15.4)	19.5 (14.8, 25.3)	13.2 (9.6, 17.8)	

Table 3B.3 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Asian						
Overall (10,095)	33.7 (32.2, 35.2)	26.3 (25.0, 27.6)	25.7 (24.4, 27.0)	11.2 (10.4, 12.1)	3.2 (2.7, 3.8)	
Never (6,563)	46.9 (45.0, 48.8)	25.2 (23.6, 26.8)	20.7 (19.3, 22.3)	6.2 (5.5, 7.1)	1.0 (0.7, 1.3)	<.001
Former (2,501)	7.0 (5.6, 8.7)	29.4 (26.7, 32.2)	39.4 (36.4, 42.5)	18.3 (16.3, 20.6)	5.9 (4.3, 7.9)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (625)	8.2 (5.8, 11.4)	20.4 (15.9, 25.8)	27.8 (22.9, 33.3)	31.5 (26.3, 37.1)	12.1 (9.0, 16.1)	
Current (past-30-day) daily smoking, <300 cigarettes/month (174)	10.2 (4.3, 22.3)	34.6 (23.5, 47.7)	20.9 (13.1, 31.8)	23.0 (15.6, 32.7)	11.2 (6.0, 20.1)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (232)	12.7 (7.3, 21.2)	31.9 (23.4, 41.7)	18.9 (12.4, 27.9)	24.0 (17.0, 32.6)	12.5 (8.2, 18.6)	
Multirace						
Overall (6,912)	11.2 (9.8, 12.8)	37.4 (35.3, 39.6)	25.0 (23.1, 26.9)	16.3 (14.9, 17.8)	10.1 (9.0, 11.4)	
Never (2,419)	26.0 (22.6, 29.7)	33.2 (29.7, 36.8)	28.0 (24.6, 31.6)	9.6 (7.9, 11.6)	3.3 (2.5, 4.3)	<.001
Former (2,353)	4.9 (3.2, 7.2)	42.4 (38.6, 46.2)	28.3 (25.2, 31.7)	16.4 (14.3, 18.9)	8.0 (6.5, 9.8)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (919)	6.0 (3.6, 9.7)	31.4 (26.0, 37.4)	15.4 (12.3, 19.1)	27.2 (22.5, 32.5)	20.0 (15.9, 24.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (304)	2.4 (1.2, 5.1)	34.1 (24.8, 44.8)	13.9 (9.3, 20.4)	26.3 (18.9, 35.4)	23.2 (14.7, 34.6)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (917)	3.7 (1.9, 7.0)	38.6 (33.1, 44.4)	19.8 (15.3, 25.3)	18.6 (14.7, 23.3)	19.3 (15.3, 23.9)	

Note. CI = confidence interval. Categories other than Hispanic are non-Hispanic. Multirace is defined as two or more races. American Indian, Alaska Native, Native Hawaiian, and Other Pacific Islander participants were combined due to small sample sizes and because they are indigenous populations in the United States that have similar drinking patterns. Data are visualized in Figure 3.2. [a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Alcohol use* is defined as use within the past year. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each race and/or ethnicity group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.4 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), by Race and/or Ethnicity *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Hispanic						
Overall (36,074)	21.6 (20.9, 22.3)	32.6 (31.9, 33.4)	18.8 (18.1, 19.4)	21.2 (20.6, 21.9)	5.7 (5.4, 6.1)	
Never (32,609)	23.4 (22.6, 24.1)	33.3 (32.5, 34.1)	18.5 (17.9, 19.2)	19.8 (19.2, 20.5)	5.0 (4.6, 5.3)	<.001
Former (2,874)	3.1 (2.2, 4.5)	26.0 (23.5, 28.6)	22.7 (20.3, 25.2)	35.3 (32.7, 38.1)	12.9 (11.3, 14.7)	
Current (past-30-day) nondaily use (454)	7.7 (4.4, 13.2)	19.5 (14.0, 26.5)	12.1 (8.0, 17.9)	42.3 (35.1, 50.0)	18.3 (13.3, 24.7)	
Current (past-30-day) daily use (137)	*	30.4 (20.5, 42.7)	12.9 (6.9, 22.8)	28.2 (18.4, 40.7)	21.7 (13.1, 33.8)	
Black						
Overall (26,493)	22.5 (21.8, 23.3)	32.1 (31.3, 32.9)	21.0 (20.4, 21.7)	18.1 (17.4, 18.7)	6.3 (5.9, 6.7)	
Never (24,574)	23.5 (22.7, 24.3)	32.1 (31.2, 32.9)	21.2 (20.5, 21.9)	17.4 (16.8, 18.1)	5.8 (5.4, 6.2)	<.001
Former (1,566)	9.6 (7.6, 12.1)	33.0 (29.7, 36.5)	20.9 (18.4, 23.6)	24.7 (22.0, 27.6)	11.8 (9.9, 14.0)	
Current (past-30-day) nondaily use (270)	13.9 (8.9, 21.1)	29.9 (22.5, 38.6)	9.1 (5.8, 14.0)	35.9 (28.3, 44.2)	11.2 (7.2, 17.1)	
Current (past-30-day) daily use (83)	21.7 (11.0, 38.2)	31.4 (19.6, 46.2)	12.1 (5.0, 26.4)	17.4 (9.9, 28.8)	17.4 (9.9, 28.9)	
White						
Overall (127,265)	9.1 (8.9, 9.3)	31.5 (31.1, 31.9)	31.7 (31.3, 32.1)	16.3 (16.1, 16.6)	11.4 (11.1, 11.6)	
Never (95,348)	11.0 (10.7, 11.3)	32.6 (32.2, 33.0)	33.1 (32.7, 33.5)	13.8 (13.5, 14.1)	9.5 (9.3, 9.8)	<.001
Former (24,528)	1.8 (1.5, 2.0)	27.9 (27.1, 28.7)	28.6 (27.8, 29.5)	24.8 (24.0, 25.5)	17.0 (16.3, 17.6)	
Current (past-30-day) nondaily use (3,943)	5.2 (4.2, 6.6)	22.6 (20.7, 24.5)	18.3 (16.5, 20.2)	31.8 (29.9, 33.8)	22.1 (20.4, 23.9)	
Current (past-30-day) daily use (3,446)	3.2 (2.4, 4.2)	29.9 (27.8, 32.2)	19.9 (18.0, 21.9)	26.0 (24.1, 28.0)	21.0 (19.1, 23.1)	
American Indian, Alaska Native, Native Hawaiian, or Other Pacific Islander						
Overall (4,074)	22.6 (20.0, 25.5)	39.3 (36.5, 42.1)	15.0 (13.1, 17.3)	15.7 (14.1, 17.6)	7.3 (6.2, 8.6)	
Never (2,877)	26.2 (23.1, 29.7)	38.2 (35.1, 41.5)	15.5 (13.2, 18.1)	14.4 (12.5, 16.5)	5.7 (4.6, 7.1)	<.001

Table 3B.4 (continued)

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Former (866)	8.1 (5.2, 12.4)	44.8 (38.9, 50.9)	13.9 (10.0, 19.1)	20.6 (16.9, 24.8)	12.6 (9.7, 16.1)	
Current (past-30-day) nondaily use (214)	13.3 (6.9, 24.3)	34.2 (23.3, 47.1)	14.2 (5.8, 30.8)	21.6 (13.9, 31.8)	16.7 (10.2, 26.2)	
Current (past-30-day) daily use (117)	12.4 (6.1, 23.5)	44.7 (29.6, 60.8)	*	21.8 (12.5, 35.2)	13.3 (5.9, 27.4)	
Asian						
Overall (10,100)	33.7 (32.2, 35.2)	26.3 (25.0, 27.6)	25.7 (24.3, 27.0)	11.2 (10.4, 12.1)	3.2 (2.7, 3.8)	
Never (9,509)	35.1 (33.5, 36.6)	26.5 (25.2, 27.9)	25.4 (24.1, 26.8)	10.2 (9.4, 11.0)	2.8 (2.3, 3.5)	<.001
Former (495)	4.7 (3.0, 7.5)	22.6 (17.1, 29.2)	30.6 (23.6, 38.7)	33.0 (26.6, 40.0)	9.1 (6.4, 13.0)	
Current (past-30-day) nondaily use (75)	29.5 (16.0, 47.8)	23.9 (11.0, 44.4)	21.7 (11.2, 37.8)	16.4 (8.4, 29.6)	8.5 (3.9, 17.7)	
Current (past-30-day) daily use (21)	*	*	*	*	*	
Multirace						
Overall (6,925)	11.3 (9.9, 12.9)	37.5 (35.3, 39.7)	24.9 (23.1, 26.9)	16.3 (14.9, 17.7)	10.1 (8.9, 11.3)	
Never (5,414)	13.2 (11.5, 15.2)	38.6 (36.2, 41.1)	25.4 (23.3, 27.7)	15.1 (13.6, 16.7)	7.7 (6.6, 9.0)	<.001
Former (1,173)	2.5 (1.1, 5.3)	33.3 (28.5, 38.5)	23.5 (19.7, 27.9)	22.4 (18.7, 26.5)	18.4 (14.9, 22.4)	
Current (past-30-day) nondaily use (211)	*	19.3 (12.6, 28.4)	20.9 (13.5, 31.0)	20.6 (14.1, 29.0)	26.2 (17.7, 37.1)	
Current (past-30-day) daily use (127)	*	45.4 (26.7, 65.5)	20.4 (11.3, 34.1)	10.8 (5.8, 19.1)	21.3 (10.1, 39.6)	

Note. CI = confidence interval. Categories other than Hispanic are non-Hispanic. Multirace is defined as two or more races. American Indian, Alaska Native, Native Hawaiian, and Other Pacific Islander participants were combined due to small sample sizes and because they are indigenous populations in the United States that have similar drinking patterns.
[a]*Current smokeless tobacco* use is defined as use in the past 30 days. [b]*Alcohol use* is defined as use within the past year. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories within each race and/or ethnicity group.
*Cells with $n < 10$ are suppressed.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.5 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), by Race and/or Ethnicity *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Hispanic						
Overall (36,089)	21.6 (21.0, 22.3)	32.6 (31.9, 33.4)	18.8 (18.2, 19.5)	21.2 (20.6, 21.9)	5.7 (5.4, 6.1)	
Never (27,251)	27.4 (26.6, 28.2)	35.0 (34.1, 35.9)	16.6 (15.9, 17.3)	17.5 (16.8, 18.1)	3.6 (3.3, 3.9)	<.001
Former (7,253)	2.9 (2.3, 3.7)	26.2 (24.7, 27.9)	27.4 (25.8, 29.0)	31.8 (30.2, 33.4)	11.7 (10.7, 12.8)	
Current (past-30-day) cigar smoking (1,585)	5.7 (4.0, 7.9)	19.4 (16.6, 22.6)	16.2 (13.5, 19.3)	41.5 (37.8, 45.2)	17.2 (14.7, 20.1)	
Black						
Overall (26,492)	22.5 (21.8, 23.3)	32.1 (31.3, 32.9)	21.0 (20.3, 21.7)	18.1 (17.5, 18.7)	6.3 (5.9, 6.7)	
Never (19,507)	28.7 (27.8, 29.6)	33.6 (32.6, 34.6)	18.8 (18.1, 19.6)	14.8 (14.1, 15.5)	4.1 (3.8, 4.5)	<.001
Former (4,346)	4.2 (3.5, 5.1)	30.7 (28.7, 32.7)	29.1 (27.3, 31.0)	24.8 (23.1, 26.6)	11.2 (10.0, 12.6)	
Current (past-30-day) cigar smoking (2,639)	4.7 (3.8, 5.8)	21.5 (19.3, 23.8)	24.5 (22.3, 26.7)	33.8 (31.4, 36.3)	15.6 (13.9, 17.6)	
White						
Overall (127,274)	9.1 (8.9, 9.3)	31.5 (31.1, 31.9)	31.7 (31.3, 32.1)	16.3 (16.1, 16.6)	11.4 (11.1, 11.6)	
Never (73,986)	14.1 (13.8, 14.5)	35.4 (34.9, 35.9)	30.6 (30.2, 31.1)	12.1 (11.8, 12.4)	7.8 (7.5, 8.1)	<.001
Former (46,022)	1.6 (1.5, 1.8)	26.7 (26.1, 27.3)	34.3 (33.7, 35.0)	21.5 (21.0, 22.0)	15.9 (15.5, 16.4)	
Current (past-30-day) cigar smoking (7,266)	2.1 (1.7, 2.7)	18.7 (17.4, 20.1)	25.3 (23.8, 26.9)	31.4 (29.9, 32.9)	22.5 (21.1, 23.9)	

Table 3B.5 (continued)

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
American Indian, Alaska Native, Native Hawaiian, or Other Pacific Islander						
Overall (4,075)	22.6 (20.0, 25.5)	39.3 (36.5, 42.1)	15.0 (13.0, 17.3)	15.7 (14.1, 17.6)	7.3 (6.2, 8.6)	
Never (2,869)	28.8 (25.6, 32.2)	40.7 (37.4, 44.1)	12.2 (10.1, 14.7)	13.4 (11.5, 15.4)	5.0 (3.9, 6.3)	<.001
Former (977)	6.2 (2.8, 13.2)	37.3 (32.0, 43.0)	24.1 (19.3, 29.7)	20.3 (16.7, 24.5)	12.1 (9.3, 15.6)	
Current (past-30-day) cigar smoking (229)	9.4 (3.3, 23.7)	27.5 (19.7, 37.0)	14.0 (8.4, 22.7)	29.4 (21.0, 39.4)	19.7 (13.5, 27.9)	
Asian						
Overall (10,102)	33.7 (32.2, 35.2)	26.3 (25.0, 27.6)	25.7 (24.3, 27.0)	11.2 (10.4, 12.1)	3.2 (2.7, 3.8)	
Never (8,389)	39.2 (37.6, 40.9)	27.2 (25.8, 28.7)	22.9 (21.6, 24.3)	8.8 (8.0, 9.7)	1.9 (1.4, 2.5)	<.001
Former (1,515)	5.1 (3.6, 7.4)	22.4 (19.3, 25.7)	40.1 (36.3, 44.0)	22.9 (20.0, 26.0)	9.5 (7.8, 11.7)	
Current (past-30-day) cigar smoking (198)	4.9 (2.5, 9.2)	16.5 (8.5, 29.7)	36.9 (26.3, 49.0)	28.7 (20.5, 38.7)	13.0 (8.2, 19.9)	
Multirace						
Overall (6,926)	11.3 (9.9, 12.9)	37.4 (35.3, 39.6)	24.9 (23.0, 26.8)	16.3 (14.9, 17.7)	10.1 (9.0, 11.3)	
Never (4,019)	17.5 (15.2, 20.0)	41.3 (38.3, 44.3)	24.0 (21.6, 26.6)	11.6 (10.2, 13.3)	5.6 (4.6, 6.8)	<.001
Former (2,279)	2.2 (1.2, 3.7)	32.5 (29.1, 36.1)	27.5 (24.3, 30.9)	22.3 (19.6, 25.3)	15.6 (13.2, 18.2)	
Current (past-30-day) cigar smoking (628)	3.6 (1.5, 8.3)	28.9 (23.2, 35.4)	20.0 (15.5, 25.3)	25.9 (21.0, 31.5)	21.6 (16.4, 27.9)	

Note. CI = confidence interval. Categories other than Hispanic are non-Hispanic. Multirace is defined as two or more races. American Indian, Alaska Native, Native Hawaiian, and Other Pacific Islander participants were combined due to small sample sizes and because they are indigenous populations in the United States that have similar drinking patterns.
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Alcohol use* is defined as use within the past year. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories within each race and/or ethnicity group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.6 Average Past-Year Alcohol Consumption (Overall and Beverage-Specific) by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Race and/or Ethnicity, Stratified by Age

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Alcohol type				*Alcohol type*				*Alcohol type*			
	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits
	Mean number of drinks consumed in past year (95% CI)											
Hispanic, Latino/a, or of Spanish origin												
Never (2,019)	179.8 (114.7, 244.8)	69.2 (35.1, 103.2)	45.1 (29.3, 60.8)	66.2 (38.3, 95.2)	144.9 (114.5, 175.2)	74.5 (50.2, 98.8)	37.2 (29.7, 44.7)	33.5 (24.4, 42.6)	187.0 (42.2, 331.7)	65.6 (0.8, 130.5)	72.8 (8.7, 137.0)	48.6 (-16.8, 114.0)
Former (338)	163.8 (32.0, 295.5)	97.2 (4.7, 189.8)	22.9 (4.4, 41.3)	43.7 (14.7, 72.7)	276.2* (171.7, 380.8)	130.2 (60.4, 199.9)	44.3 (26.0, 62.6)	101.9** (37.7, 166.0)	245.6 (100.9, 390.3)	87.7 (33.4, 142.0)	58.2 (25.1, 91.3)	101.4 (-13.6, 216.5)
Current (past-year) (384)	579.3** (291.3, 867.3)	210.5+ (64.3, 356.7)	48.2 (24.6, 71.7)	320.6* (73.0, 568.2)	464.7** (323.3, 606.1)	266.6** (166.1, 367.1)	69.1** (37.3, 100.9)	132.0** (57.1, 206.9)	293.4 (-42.4, 629.3)	190.3 (-82.3, 462.8)	56.7 (23.6, 136.9)	46.5 (-1.4, 95.3)
Nondaily (135)	420.4+ (160.3, 680.4)	136.6 (42.3, 230.9)	36.2 (16.0, 56.3)	247.6 (-26.3, 521.5)	727.8** (379.7, 1,075.8)	329.5** (159.6, 499.4)	155.5* (60.7, 250.2)	244.4+ (28.2, 460.5)	59.9 (-0.6, 120.4)	25.6 (-9.7, 60.9)	14.5 (-4.5, 33.4)	19.8 (-5.3, 44.9)
Daily (249)	745.2* (213.9, 1,276.4)	287.6 (7.2, 568.1)	60.7 (18.7, 102.7)	396.8 (-27.7, 821.3)	368.4** (225.9, 510.8)	243.3** (120.3, 366.2)	37.2 (14.5, 59.9)	91.0* (28.3, 153.7)	322.7 (-54.9, 700.3)	210.9 (-95.7, 517.5)	62.0 (-28.2, 152.1)	49.8 (-4.0, 103.6)
Black or African American												
Never (2,012)	200.6 (132.7, 268.5)	56.2 (31.3, 81.2)	46.6 (31.0, 62.2)	97.8 (56.0, 139.5)	151.3 (112.7, 190.0)	39.8 (28.0, 51.7)	36.0 (27.7, 44.3)	76.3 (44.6, 107.9)	63.7 (41.2, 86.2)	21.5 (9.2, 33.7)	22.6 (13.4, 31.7)	20.8 (10.7, 30.9)
Former (435)	68.4 (-10.0, 146.7)	19.6 (-6.8, 46.1)	25.8 (-7.1, 58.7)	23.0* (-12.0, 57.8)	186.7 (89.2, 284.3)	50.0 (17.7, 82.2)	50.6 (8.9, 92.3)	87.1 (29.2, 145.0)	59.3 (37.4, 81.2)	24.5 (9.2, 39.8)	18.4 (8.0, 28.8)	16.8 (7.6, 26.1)
Current (past-year) (592)	522.2** (328.4, 716. 0)	210.9** (106.1, 315.8)	91.5+ (40.2, 142.8)	219.8* (108.2, 331.4)	478.2** (295.6, 660.9)	205.8** (126.7, 285.0)	42.4 (28.2, 56.6)	230.3* (105.1, 355.4)	428.3** (159.1, 697.5)	165.6** (61.2, 270.0)	101.3* (22.4, 180.1)	163.9** (51.9, 275.9)
Nondaily (138)	685.3** (354.3, 1,016.3)	333.3** (138.1, 528.6)	117.1* (54.9, 179.4)	234.9+ (94.6, 375.1)	480.8* (200.3, 761.3)	147.8** (72.5, 223.1)	88.6* (39.7, 137.5)	244.3 (9.7, 479.0)	243.1+ (64.8, 421.3)	85.3 (-6.8, 177.4)	106.4* (24.6, 188.1)	51.4 (3.2, 99.5)
Daily (454)	444.6* (210.7, 678.4)	152.6 (37.5, 267.7)	79.3 (10.2, 148.4)	212.6 (61.6, 363.6)	477.6** (260.8, 694.3)	220.0** (123.0, 317.1)	31.1 (18.8, 43.5)	226.8* (82.0, 371.7)	454.0** (147.5, 760.5)	176.8** (58.5, 295.0)	100.5+ (11.5, 189.6)	179.8* (51.3, 208.3)
White												
Never (4,167)	241.3 (190.1, 292.4)	108.8 (80.5, 137.2)	52.3 (37.8, 66.8)	81.9 (61.5, 102.3)	237.6 (205.3, 269.9)	97.5 (83.1, 111.8)	68.0 (57.0, 79.0)	72.6 (57.4, 87.9)	137.2 (114.3, 160.0)	46.8 (33.3, 60.2)	53.4 (43.5, 63.2)	37.3 (24.8, 49.8)
Former (1,639)	555.7 (138.5, 973.0)	226.6+ (56.7, 396.5)	81.6 (28.6, 134.6)	247.6* (-11.0, 506.2)	266.4 (225.2, 307.5)	100.9 (79.4, 122.4)	76.7 (59.7, 93.6)	89.1 (66.2, 112.0)	218.9** (188.3, 249.6)	61.8 (45.7, 78.0)	79.1** (64.1, 94.1)	79.3** (60.8, 97.7)
Current (past-year) (1,276)	480.4** (309.9, 651.0)	252.2** (157.5, 347.0)	66.7 (42.5, 90.8)	161.5+ (71.3, 251.8)	451.5** (335.0, 568.0)	232.6** (143.3, 321.9)	54.8 (41.1, 68.4)	166.6 (105.5, 227.6)	275.8** (196.6, 355.0)	82.9* (50.2, 115.7)	72.5 (35.3, 109.6)	121.0** (65.6, 176.5)
Nondaily (274)	515.8** (318.5, 713.1)	194.7 (82.4, 306.9)	116.4** (68.0, 174.9)	204.7** (124.1, 285.3)	379.5* (255.2, 503.8)	142.6 (80.4, 204.9)	96.8 (55.5, 138.1)	142.4+ (75.6, 209.3)	351.3** (203.3, 499.3)	107.1+ (37.4, 176.8)	84.5 (37.2, 131.7)	160.3** (67.7, 252.9)
Daily (1,002)	466.5* (241.3, 691.6)	275.0** (151.2, 398.8)	47.0 (23.3, 70.7)	144.5 (22.4, 266.6)	465.7** (328.4, 603.1)	250.3** (144.2, 356.3)	46.4* (32.7, 60.1)	171.3** (99.4, 243.3)	263.1** (173.8, 352.4)	78.9+ (42.5, 115.3)	70.4 (27.8, 113.1)	114.5** (51.6, 177.3)

Note. CI = confidence interval. Black and White participants are non-Hispanic. Data are visualized in Figure 3.3.

[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as daily or nondaily use in the past year. *Former tobacco use* is defined as ever use in lifetime but not in the past year. *Never tobacco use* is defined as no lifetime use of tobacco.

+$p < .1$ for association comparing alcohol consumption among tobacco use categories (referent = Never). *$p < .05$ for association comparing alcohol consumption among tobacco use categories (referent = Never). **$p < .01$ for association comparing alcohol consumption among tobacco use categories (referent = Never).

Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.7 Cigarette Smoking History, Categorized by Pack Years, by Lifetime Number of 5+ Drinking Days in the 2014–2015 National Alcohol Survey (*N* = 5,632), by Race and/or Ethnicity, Stratified by Age *(Row Percents)*

Cigarette pack years (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	0	0.1–499	≥500	*p* value[a]	0	0.1–999	≥1,000	*p* value	0	0.1–999	≥1,000	*p* value
	Lifetime number of 5+ drinking days:[b] Weighted % (95% CI)											
Hispanic, Latino/a, or of Spanish origin												
0 (878)	58.8 (48.2, 68.6)	30.2 (21.7, 40.5)	11.0 (5.7, 20.1)	N/A	58.0 (50.1, 65.5)	27.4 (20.8, 35.2)	14.6 (9.9, 21.2)	<.001	85.5 (71.3, 93.4)	3.6 (1.9, 6.8)	10.9 (3.9, 26.8)	<.001
0.1–20 (242)	*	40.7 (21.5, 63.2)	43.2 (24.4, 64.2)		21.1 (12.6, 33.2)	36.0 (24.7, 49.0)	42.9 (31.4, 55.3)		46.3 (26.4, 67.5)	22.0 (7.8, 48.3)	31.7 (15.2, 54.6)	
20.1–40 (32)	*	*	*		*	*	47.7 (20.1, 76.8)		*	*	*	
≥40.1 (85)	*	68.3 (36.2, 89.1)	*		12.3 (4.3, 30.5)	23.6 (10.4, 45.0)	64.2 (40.7, 82.4)		59.6 (27.1, 85.4)	*	29.7 (9.5, 63.0)	
Black or African American												
0 (830)	62.1 (49.6, 73.2)	30.6 (20.6, 42.8)	*	N/A	70.6 (62.4, 77.6)	19.5 (13.6, 27.3)	9.9 (5.9, 16.0)	<.001	90.1 (82.1, 94.7)	4.9 (2.0, 11.6)	5.1 (2.1, 11.8)	<.001
0.1–20 (324)	40.5 (20.6, 64.0)	32.2 (14.4, 57.4)	*		40.6 (29.0, 53.4)	24.2 (14.2, 38.0)	35.3 (24.3, 48.0)		76.9 (58.8, 88.6)	6.7 (2.8, 14.9)	16.5 (6.5, 35.8)	
20.1–40 (82)	*	*	*		37.8 (10.1, 76.6)	*	57.5 (21.4, 87.1)		29.7 (14.5, 51.3)	13.7 (6.3, 27.3)	56.6 (35.2, 75.8)	
≥40.1 (116)	*	*	*		39.8 (18.7, 65.5)	*	28.6 (10.0, 59.2)		74.4 (53.0, 88.3)	12.7 (5.2, 27.7)	12.9 (4.6, 31.1)	
White												
0 (1,446)	37.8 (29.0, 47.5)	52.0 (42.3, 61.5)	10.2 (5.7, 17.7)	<.001	34.8 (30.4, 39.5)	48.4 (43.4, 53.4)	16.8 (13.2, 21.3)	<.001	74.5 (69.6, 78.8)	17.0 (13.4, 21.2)	8.6 (5.9, 12.3)	<.001
0.1–20 (591)	*	55.8 (41.8, 69.0)	35.2 (23.1, 49.6)		13.2 (9.4, 18.3)	40.8 (33.7, 48.3)	46.0 (38.6, 53.5)		59.0 (50.2, 67.4)	22.2 (15.8, 30.4)	18.7 (12.7, 26.8)	
20.1–40 (253)	*	*	*		13.7 (7.2, 24.4)	41.7 (30.0, 54.3)	44.7 (33.1, 56.9)		36.1 (26.8, 46.5)	35.6 (25.6, 47.0)	28.3 (19.1, 39.8)	
≥40.1 (398)	*	*	*		19.2 (12.3, 28.8)	39.4 (29.0, 50.8)	41.4 (30.9, 52.8)		39.3 (31.8, 47.5)	25.2 (19.0, 32.7)	35.5 (27.9, 43.8)	

Note. CI = confidence interval; N/A = not applicable. Black and White participants are non-Hispanic.
[a]*p* value for bivariate association, obtained through a chi-squared test, between pack year and alcohol use categories within each age and race and/or ethnicity group. *p* value could not be calculated for groups that included categories where *n* = 0. [b]*Lifetime 5+ drinking days* is defined as the number of days over lifetime in which participants reported drinking 5 or more drinks on 1 occasion.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 U.S. National Alcohol Survey.

Table 3B.8 Past-Year Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Education, Stratified by Age *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use	Past-year alcohol use within guidelines[b]	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
High school or less												
Overall (3,946)	41.7 (37.0, 46.6)	26.0 (22.1, 30.4)	32.3 (27.9, 37.0)		37.5 (34.5, 40.6)	35.4 (32.4, 38.6)	27.1 (24.1, 30.3)		52.9 (49.0, 56.8)	35.1 (31.4, 39.0)	12.0 (9.6, 14.9)	
Never (2,300)	52.1 (46.4, 57.8)	25.1 (20.6, 30.1)	22.8 (18.4, 27.9)	<.001	44.4 (40.0, 48.8)	36.4 (32.2, 40.8)	19.2 (15.6, 23.4)	<.001	64.4 (58.9, 69.6)	29.2 (24.5, 34.5)	6.3 (3.9, 10.0)	<.001
Former (699)	*	*	52.0 (30.3, 73.0)		32.3 (24.9, 40.6)	43.3 (34.8, 52.2)	24.4 (17.4, 33.1)		42.3 (36.1, 48.8)	44.2 (37.6, 50.9)	13.5 (9.5, 18.8)	
Current (past-year) (947)	20.7 (13.5, 30.5)	27.5 (19.4, 37.5)	51.8 (41.5, 61.8)		29.3 (24.5, 34.7)	30.1 (25.1, 35.6)	40.6 (34.9, 46.6)		42.3 (33.4, 51.8)	33.5 (24.9, 43.4)	24.1 (17.0, 33.0)	
Nondaily (139)	*	31.8 (15.1, 55.1)	47.5 (26.4, 69.5)		25.3 (13.7, 41.9)	33.4 (19.9, 50.4)	41.3 (26.9, 57.3)		*	56.7 (27.3, 82.1)	*	
Daily (808)	20.7 (12.8, 31.8)	26.8 (18.0, 37.9)	52.5 (41.1, 63.5)		29.8 (24.6, 35.6)	29.6 (24.4, 35.5)	40.5 (34.4, 46.9)		43.8 (34.3, 53.7)	30.8 (22.3, 40.9)	25.4 (17.7, 35.1)	
Some college												
Overall (4,062)	22.2 (18.4, 26.5)	29.6 (25.1, 34.6)	48.2 (43.0, 53.4)		25.1 (22.5, 27.8)	40.6 (37.6, 43.6)	34.4 (31.4, 37.4)		36.1 (32.5, 39.8)	49.1 (45.4, 52.9)	14.8 (12.3, 17.7)	
Never (2,372)	28.5 (23.4, 34.1)	31.0 (25.6, 36.9)	40.5 (34.7, 46.6)	<.001	28.8 (25.2, 32.6)	43.9 (39.9, 48.0)	27.3 (23.6, 31.2)	<.001	43.7 (38.4, 49.1)	46.8 (41.6, 52.1)	9.5 (7.0, 12.7)	<.001
Former (804)	*	35.7 (20.6, 54.5)	39.3 (22.5, 59.1)		21.2 (15.6, 28.1)	40.3 (33.6, 47.5)	38.5 (31.6, 45.8)		28.1 (23.0, 33.8)	52.3 (46.0, 58.6)	19.6 (14.6, 25.7)	
Current (past-year) (886)	5.8 (2.7, 12.0)	21.6 (13.7, 32.5)	72.5 (61.5, 81.4)		20.0 (15.5, 25.4)	32.7 (27.0, 39.0)	47.3 (41.1, 53.6)		31.0 (22.7, 40.6)	49.2 (39.9, 58.6)	19.8 (13.6, 28.0)	
Nondaily (224)	*	14.9 (7.2, 28.2)	80.2 (66.8, 89.0)		9.2 (4.8, 17.0)	25.0 (16.1, 36.8)	65.7 (53.5, 76.2)		*	55.9 (34.3, 75.4)	*	
Daily (662)	*	28.4 (15.8, 45.7)	64.8 (47.7, 78.8)		22.5 (17.1, 28.9)	34.5 (27.9, 41.8)	43.0 (36.2, 50.1)		31.9 (22.9, 42.4)	48.3 (38.3, 58.5)	19.8 (13.1, 28.7)	

Table 3B.8 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	No past-year alcohol use	Past-year alcohol use within guidelines[b]	Past-year alcohol use exceeding guidelines	*p* value[c]	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value	No past-year alcohol use	Past-year alcohol use within guidelines	Past-year alcohol use exceeding guidelines	*p* value
	Alcohol use: Weighted % (95% CI)											
College degree												
Overall (5,516)	14.6 (11.5, 18.5)	23.0 (18.8, 27.8)	62.4 (57.1, 67.3)		20.3 (18.3, 22.4)	44.4 (41.8, 47.0)	35.4 (32.8, 38.0)		27.8 (25.1, 30.7)	55.6 (52.5, 58.7)	16.5 (14.4, 19.0)	
Never (4,1014)	16.0 (12.4, 20.4)	23.5 (18.9, 28.7)	60.6 (54.8, 66.1)	.38	22.4 (20.0, 25.0)	46.7 (43.8, 49.7)	30.9 (28.0, 33.8)	<.001	32.1 (28.4, 36.0)	54.4 (50.4, 58.4)	13.5 (11.0, 16.4)	.001
Former (960)	*	*	64.2 (33.1, 86.6)		14.1 (10.2, 19.2)	43.8 (36.7, 51.0)	42.1 (35.3, 49.3)		21.0 (17.0, 25.6)	58.5 (53.0, 63.9)	20.5 (16.3, 25.5)	
Current (past-year) (542)	*	19.1 (10.0, 33.2)	67.5 (52.1, 79.9)		14.8 (9.8, 21.8)	30.0 (22.6, 38.7)	55.2 (46.4, 63.6)		26.9 (17.6, 38.9)	50.3 (39.4, 61.2)	22.8 (15.6, 32.1)	
Nondaily (229)	*	16.3 (6.5, 35.2)	76.7 (57.0, 89.1)		10.9 (5.5, 20.3)	32.7 (21.0, 47.0)	56.4 (42.6, 69.3)		*	62.4 (45.9, 76.4)	21.1 (10.4, 38.1)	
Daily (313)	*	22.4 (8.8, 46.2)	56.8 (33.0, 77.8)		17.2 (10.3, 27.2)	28.4 (19.4, 39.6)	54.4 (43.1, 65.3)		31.6 (19.3, 47.3)	44.8 (31.3, 59.1)	23.6 (14.8, 35.3)	

Note. CI = confidence interval. Data are visualized in Figure 3.4.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. [b]*Alcohol use* is defined as use within the past year. *Alcohol use exceeding guidelines* is defined as exceeding 7/14 drinks per week or drinking at least 4/5 drinks per day for women/men, per the *Dietary Guidelines for Americans, 2020–2025*[22] and NIAAA's definition of heavy alcohol use.[23] [c]*p* value for bivariate association, obtained through a chi-squared test, between tobacco and alcohol use categories within each age and education group.
*Cells with $n < 10$ are suppressed.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.9 Past-Year Hazardous Alcohol Use by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Education, Stratified by Age *(Row Percents)*

Tobacco use status[a] (unweighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
High school or less																		
Overall (4,073)	75.2 (70.9, 79.2)	24.8 (20.8, 29.1)		90.5 (87.3, 93.0)	9.5 (7.0, 12.7)		79.5 (76.6, 82.2)	20.5 (17.8, 23.4)		93.5 (91.6, 95.0)	6.5 (5.0, 8.4)		92.4 (90.0, 94.3)	7.6 (5.7, 10.0)		98.5 (97.3, 99.2)	1.5 (0.8, 2.7)	
Never (2,384)	83.4 (78.9, 87.1)	16.6 (12.9, 21.1)	<.001	95.4 (93.1, 97.0)	4.6 (3.0, 6.9)	<.001	85.4 (81.5, 88.6)	14.6 (11.4, 18.5)	<.001	96.2 (93.6, 97.7)	3.8 (2.3, 6.4)	<.001	97.5 (94.9, 98.8)	2.5 (1.2, 5.1)	<.001	99.8 (99.4, 99.9)	*	<.001
Former (719)	72.2 (47.8, 88.0)	*		95.0 (81.4, 98.8)	*		83.1 (75.5, 88.6)	16.9 (11.4, 24.5)		96.1 (89.6, 98.6)	3.9 (1.4, 10.4)		90.8 (85.9, 94.1)	9.2 (5.9, 14.1)		98.6 (96.5, 99.4)	*	
Current (past-year) (970)	56.4 (46.2, 66.1)	43.6 (33.9, 53.8)		78.3 (68.6, 85.6)	21.7 (14.4, 31.4)		68.4 (62.6, 73.6)	31.6 (26.4, 37.4)		88.1 (83.8, 91.3)	11.9 (8.7, 16.2)		82.5 (74.3, 88.5)	17.5 (11.5, 25.7)		95.0 (89.2, 97.7)	5.0 (2.3, 10.8)	
Nondaily (143)	56.0 (33.5, 76.3)	44.0 (23.7, 66.5)		79.5 (48.0, 94.2)	*		65.5 (49.7, 78.5)	34.5 (21.5, 50.3)		88.6 (73.5, 95.6)	11.4 (4.4, 26.5)		100.0	*		100.0	*	
Daily (827)	56.5 (45.2, 67.1)	43.5 (32.9, 54.8)		78.1 (67.7, 85.9)	21.9 (14.1, 32.3)		68.8 (62.5, 74.4)	31.2 (25.6, 37.5)		88.0 (83.4, 91.5)	12.0 (8.5, 16.6)		80.5 (71.6, 87.1)	19.5 (12.9, 28.4)		94.4 (88.1, 97.4)	5.6 (2.6, 11.9)	
Some college																		
Overall (4,176)	59.7 (54.5, 64.7)	40.3 (35.3, 45.5)		86.3 (82.2, 89.6)	13.7 (10.4, 17.8)		74.8 (72.0, 77.5)	25.2 (22.5, 28.0)		92.8 (91.1, 94.2)	7.2 (5.8, 8.9)		94.1 (92.4, 95.5)	5.9 (4.5, 7.6)		99.1 (98.2, 99.5)	0.9 (0.5, 1.8)	
Never (2,442)	67.2 (61.2, 72.7)	32.8 (27.3, 38.8)	<.001	91.1 (86.9, 94.1)	8.9 (5.9, 13.1)	<.001	80.1 (76.5, 83.2)	19.9 (16.8, 23.5)	<.001	94.3 (92.0, 95.9)	5.7 (4.1, 8.0)	.02	96.1 (93.8, 97.6)	3.9 (2.4, 6.2)	.03	99.0 (97.5, 99.6)	*	.96
Former (829)	68.1 (48.9, 82.7)	31.9 (17.3, 51.1)		92.1 (81.6, 96.9)	*		73.2 (66.3, 79.1)	26.8 (20.9, 33.7)		94.1 (90.1, 96.6)	5.9 (3.4, 9.9)		93.2 (89.9, 95.4)	6.8 (4.6, 10.1)		99.1 (97.2, 99.7)	*	
Current (past-year) (905)	35.7 (25.5, 47.3)	64.3 (52.7, 74.5)		73.6 (62.0, 82.7)	26.4 (17.3, 38.0)		64.1 (57.9, 69.8)	35.9 (30.2, 42.1)		89.3 (85.2, 92.4)	10.7 (7.6, 14.8)		90.5 (84.3, 94.5)	9.5 (5.5, 15.7)		99.2 (97.4, 99.8)	*	
Nondaily (228)	28.6 (16.6, 44.5)	71.4 (55.5, 83.4)		80.5 (66.9, 89.4)	19.5 (10.6, 33.1)		53.8 (41.0, 66.1)	46.2 (33.9, 59.0)		87.9 (78.4, 93.5)	12.1 (6.5, 21.6)		89.1 (64.3, 97.4)	*		100.0	*	
Daily (677)	42.8 (27.6, 59.6)	57.2 (40.4, 72.4)		66.7 (48.6, 80.9)	33.3 (19.1, 51.4)		66.5 (59.6, 72.8)	33.5 (27.2, 40.4)		89.6 (84.9, 93.0)	10.4 (7.0, 15.1)		90.7 (84.0, 94.8)	9.3 (5.2, 16.0)		99.1 (97.0, 99.7)	*	

Table 3B.9 (continued)

Tobacco use status[a] (unweighted *n*)	Ages 18–29						Ages 30–59						Ages 60 and older					
	5+ drinking[b]			HID[c]			5+ drinking			HID			5+ drinking			HID		
	No past-year 5+ drinking	Past-year 5+ drinking	*p* value[d]	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value	No past-year 5+ drinking	Past-year 5+ drinking	*p* value	No past-year HID	Past-year HID	*p* value
	Alcohol use: Weighted % (95% CI)																	
College degree																		
Overall (5,613)	51.7 (46.3, 57.1)	48.3 (42.9, 53.7)		84.9 (80.6, 88.3)	15.1 (11.7, 19.4)		73.8 (71.3, 76.2)	26.2 (23.8, 28.7)		94.8 (93.4, 95.9)	5.2 (4.1, 6.6)		91.3 (89.4, 92.8)	8.7 (7.2, 10.6)		98.9 (98.2, 99.4)	1.1 (0.6, 1.8)	
Never (4,086)	55.1 (49.1, 60.9)	44.9 (39.1, 50.9)	.11	85.1 (80.2, 89.0)	14.9 (11.0, 19.8)	.52	78.2 (75.4, 80.8)	21.8 (19.2, 24.6)	<.001	95.8 (94.1, 97.0)	4.2 (3.0, 5.9)	<.001	92.7 (90.2, 94.6)	7.3 (5.4, 9.8)	.04	98.8 (97.6, 99.4)	1.2 (0.6, 2.4)	.01
Former (980)	*	62.4 (32.4, 85.2)		89.4 (72.0, 96.5)	*		68.9 (61.8, 75.2)	31.1 (24.8, 38.2)		95.9 (92.9, 97.7)	4.1 (2.3, 7.1)		90.2 (86.7, 92.8)	9.8 (7.2, 13.3)		99.7 (99.0, 99.9)	*	
Current (past-year) (547)	37.8 (24.5, 53.3)	62.2 (46.7, 75.5)		80.2 (66.6, 89.1)	19.8 (10.9, 33.4)		51.3 (42.6, 60.0)	48.7 (40.0, 57.4)		86.1 (78.9, 91.1)	13.9 (8.9, 21.1)		85.0 (76.4, 90.9)	15.0 (9.1, 23.6)		96.9 (92.1, 98.9)	*	
Nondaily (230)	32.1 (17.0, 52.1)	67.9 (47.9, 83.0)		78.4 (60.4, 89.6)	21.6 (10.4, 39.6)		53.3 (39.5, 66.6)	46.7 (33.4, 60.5)		86.7 (71.9, 94.3)	13.3 (5.7, 28.1)		81.7 (64.0, 91.8)	*		96.6 (86.4, 99.2)	*	
Daily (317)	44.6 (23.2, 68.2)	55.4 (31.8, 76.8)		82.3 (58.7, 93.8)	*		50.2 (39.1, 61.2)	49.8 (38.8, 60.9)		85.7 (77.0, 91.5)	14.3 (8.5, 23.0)		86.6 (76.1, 92.9)	13.4 (7.1, 23.9)		97.1 (90.0, 99.2)	*	

Note. CI = confidence interval; HID = high intensity drinking.
[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as use in the past year. [b]*Alcohol use* is measured in the past year. *5+ drinks* is defined as consuming 5 or more drinks on a single occasion. [c]Alcohol use is measured in the past year. *HID* is defined as drinking ≥ 8/12 drinks in one day for women/men. [d]*p* value for bivariate association, obtained through a chi-squared test, between tobacco and alcohol use categories within each age and education group.
Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.10 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), by Education *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
High school or less						
Overall (82,700)	21.8 (21.4, 22.3)	37.9 (37.4, 38.4)	16.1 (15.7, 16.5)	16.5 (16.2, 16.9)	7.6 (7.4, 7.9)	
Never (33,685)	42.5 (41.7, 43.3)	31.6 (30.8, 32.3)	12.8 (12.3, 13.4)	10.7 (10.2, 11.2)	2.5 (2.2, 2.8)	<.001
Former (24,145)	7.8 (7.3, 8.4)	45.6 (44.6, 46.5)	21.9 (21.2, 22.7)	17.0 (16.4, 17.6)	7.7 (7.2, 8.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (9,581)	10.4 (9.5, 11.4)	32.3 (30.9, 33.7)	11.7 (10.8, 12.7)	31.9 (30.5, 33.3)	13.7 (12.7, 14.7)	
Current (past-30-day) daily smoking, <300 cigarettes/month (3,313)	9.0 (7.6, 10.6)	39.9 (37.4, 42.4)	15.0 (13.2, 16.9)	24.3 (22.2, 26.4)	11.9 (10.5, 13.6)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (11,976)	7.1 (6.3, 7.9)	40.9 (39.7, 42.2)	14.6 (13.7, 15.5)	20.2 (19.2, 21.3)	17.2 (16.3, 18.2)	
Some college						
Overall (70,884)	10.4 (10.1, 10.8)	32.0 (31.5, 32.6)	28.6 (28.1, 29.2)	18.6 (18.2, 19.0)	10.4 (10.0, 10.7)	
Never (27,772)	23.8 (23.0, 24.6)	31.9 (31.0, 32.7)	26.9 (26.1, 27.7)	13.6 (13.1, 14.2)	3.8 (3.5, 4.2)	<.001
Former (26,715)	2.8 (2.5, 3.1)	32.8 (32.0, 33.6)	33.8 (33.0, 34.7)	19.8 (19.2, 20.5)	10.8 (10.3, 11.3)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (7,230)	3.5 (2.9, 4.2)	21.0 (19.5, 22.5)	20.6 (19.2, 22.0)	31.6 (30.1, 33.1)	23.4 (22.0, 24.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (2,059)	5.0 (3.5, 7.2)	30.7 (27.9, 33.7)	23.4 (20.8, 26.2)	22.4 (20.0, 24.9)	18.5 (16.2, 21.0)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (7,108)	2.7 (2.1, 3.3)	38.3 (36.7, 39.9)	21.2 (19.8, 22.6)	19.3 (18.1, 20.5)	18.6 (17.4, 19.9)	

Table 3B.10 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
College degree						
Overall (57,100)	9.0 (8.7, 9.4)	23.8 (23.3, 24.3)	40.7 (40.1, 41.3)	16.1 (15.7, 16.5)	10.4 (10.0, 10.7)	
Never (23,766)	19.8 (19.0, 20.5)	25.8 (25.0, 26.6)	39.1 (38.2, 40.0)	11.2 (10.7, 11.7)	4.2 (3.8, 4.5)	<.001
Former (27,391)	1.6 (1.4, 1.9)	22.6 (21.9, 23.4)	44.9 (44.0, 45.7)	18.1 (17.5, 18.6)	12.8 (12.3, 13.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (3,595)	2.0 (1.4, 2.8)	13.9 (12.1, 15.9)	23.0 (21.1, 25.0)	33.7 (31.6, 35.9)	27.5 (25.5, 29.5)	
Current (past-30-day) daily smoking, <300 cigarettes/month (592)	2.2 (1.1, 4.2)	27.4 (22.2, 33.3)	28.5 (23.3, 34.2)	22.3 (18.0, 27.3)	19.7 (15.8, 24.2)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,756)	3.1 (2.0, 4.6)	31.7 (28.7, 34.8)	26.5 (23.7, 29.5)	17.4 (15.3, 19.8)	21.3 (18.9, 23.9)	

Note. CI = confidence interval. Data are visualized in Figure 3.5.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each age and education group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.11 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), by Education *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
High school or less						
Overall (82,898)	21.8 (21.4, 22.3)	38.0 (37.5, 38.5)	16.1 (15.7, 16.5)	16.5 (16.2, 16.9)	7.6 (7.4, 7.9)	
Never (66,945)	25.1 (24.6, 25.6)	38.1 (37.5, 38.6)	15.7 (15.3, 16.1)	15.1 (14.8, 15.5)	6.0 (5.8, 6.3)	<.001
Former (11,502)	4.7 (4.2, 5.4)	38.6 (37.3, 40.0)	18.9 (17.9, 20.0)	23.2 (22.1, 24.3)	14.6 (13.7, 15.5)	
Current (past-30-day) nondaily use (2,494)	10.8 (8.9, 13.1)	31.6 (28.9, 34.5)	13.5 (11.5, 15.8)	27.2 (24.8, 29.7)	16.8 (14.8, 19.1)	
Current (past-30-day) daily use (1,957)	6.6 (5.1, 8.6)	36.0 (32.9, 39.3)	16.3 (13.9, 19.0)	21.9 (19.6, 24.5)	19.2 (16.8, 21.8)	
Some college						
Overall (70,932)	10.4 (10.1, 10.8)	32.0 (31.5, 32.6)	28.6 (28.1, 29.1)	18.6 (18.2, 19.0)	10.3 (10.0, 10.7)	
Never (56,484)	12.4 (12.0, 12.8)	33.3 (32.7, 33.9)	29.0 (28.4, 29.6)	16.6 (16.2, 17.0)	8.7 (8.3, 9.0)	<.001
Former (11,242)	1.5 (1.2, 1.9)	27.6 (26.4, 28.9)	28.2 (27.0, 29.5)	26.0 (24.9, 27.2)	16.6 (15.7, 17.6)	
Current (past-30-day) nondaily use (1,779)	3.8 (2.4, 5.9)	17.8 (15.2, 20.7)	21.1 (18.3, 24.3)	34.7 (31.8, 37.9)	22.6 (20.1, 25.2)	
Current (past-30-day) daily use (1,427)	1.4 (0.5, 3.7)	26.3 (23.2, 29.7)	22.5 (19.5, 25.8)	28.5 (25.4, 31.9)	21.3 (18.4, 24.6)	

Table 3B.11 (continued)

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
College degree						
Overall (57,101)	9.0 (8.7, 9.4)	23.8 (23.3, 24.4)	40.7 (40.1, 41.3)	16.1 (15.7, 16.5)	10.4 (10.0, 10.7)	
Never (46,902)	10.5 (10.1, 10.9)	25.0 (24.5, 25.6)	41.9 (41.3, 42.6)	13.7 (13.3, 14.1)	8.9 (8.5, 9.3)	<.001
Former (8,758)	1.0 (0.7, 1.4)	17.7 (16.6, 19.0)	35.8 (34.3, 37.3)	28.2 (26.9, 29.5)	17.4 (16.3, 18.4)	
Current (past-30-day) nondaily use (894)	2.4 (1.3, 4.4)	11.5 (8.6, 15.2)	19.4 (16.0, 23.3)	39.7 (35.3, 44.3)	27.0 (23.2, 31.2)	
Current (past-30-day) daily use (547)	*	21.7 (17.0, 27.3)	23.0 (18.3, 28.5)	29.4 (24.6, 34.8)	25.0 (20.0, 30.6)	

Note. CI = confidence interval.
[a]*Current smokeless tobacco use* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories within each education group.
*Cells with $n < 10$ are suppressed.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.12 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), by Education *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
High school or less						
Overall (82,910)	21.8 (21.4, 22.3)	38.0 (37.5, 38.5)	16.1 (15.7, 16.5)	16.5 (16.2, 16.9)	7.6 (7.4, 7.9)	
Never (58,317)	28.6 (28.1, 29.2)	38.3 (37.7, 38.9)	14.2 (13.7, 14.6)	13.7 (13.3, 14.1)	5.2 (4.9, 5.5)	<.001
Former (18,925)	4.2 (3.8, 4.7)	39.1 (38.0, 40.2)	21.8 (21.0, 22.8)	22.0 (21.2, 22.8)	12.9 (12.3, 13.6)	
Current (past-30-day) cigar smoking (5,668)	5.5 (4.7, 6.5)	28.0 (26.3, 29.8)	17.4 (16.0, 18.9)	31.6 (29.8, 33.4)	17.5 (16.2, 19.0)	
Some college						
Overall (70,939)	10.4 (10.1, 10.8)	32.0 (31.5, 32.6)	28.6 (28.1, 29.1)	18.6 (18.2, 19.0)	10.4 (10.0, 10.7)	
Never (44,540)	15.5 (15.0, 16.0)	35.7 (35.0, 36.4)	27.4 (26.8, 28.1)	14.7 (14.2, 15.1)	6.8 (6.4, 7.1)	<.001
Former (21,895)	1.5 (1.2, 1.7)	27.2 (26.3, 28.1)	31.5 (30.6, 32.4)	24.3 (23.5, 25.1)	15.6 (14.9, 16.3)	
Current (past-30-day) cigar smoking (4,504)	1.9 (1.4, 2.6)	16.6 (15.0, 18.3)	26.1 (24.2, 28.2)	32.2 (30.3, 34.2)	23.1 (21.4, 25.0)	
College degree						
Overall (57,109)	9.0 (8.7, 9.4)	23.8 (23.3, 24.4)	40.7 (40.1, 41.3)	16.1 (15.7, 16.5)	10.4 (10.0, 10.7)	
Never (33,164)	14.3 (13.8, 14.9)	28.3 (27.6, 29.0)	39.9 (39.1, 40.6)	11.1 (10.7, 11.5)	6.4 (6.1, 6.8)	<.001
Former (21,572)	1.1 (0.9, 1.3)	17.9 (17.1, 18.7)	42.8 (41.9, 43.8)	22.3 (21.6, 23.1)	15.9 (15.2, 16.6)	
Current (past-30-day) cigar smoking (2,373)	1.1 (0.7, 1.9)	9.3 (7.6, 11.4)	32.6 (29.8, 35.5)	36.3 (33.6, 39.0)	20.7 (18.6, 22.9)	

Note. CI = confidence interval.
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories within each education group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.13 Average Past-Year Alcohol Consumption (Overall and Beverage-Specific) by Tobacco Use Status in the 2014–2015 and 2019–2020 National Alcohol Surveys (*N* = 14,125), by Education, Stratified by Age

Tobacco use status[a] (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	Alcohol type				*Alcohol type*				*Alcohol type*			
	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits	Overall	Beer	Wine	Spirits
	Mean number of drinks consumed in past year (95% CI)											
High school or less												
Never (2,382)	156.1 (91.3, 221.0)	60.2 (31.4, 89.0)	33.4 (15.8, 50.9)	63.7 (36.4, 90.9)	154.3 (119.7, 189.0)	66.7 (46.6, 86.8)	33.6 (24.8, 42.4)	54.3 (33.3, 75.2)	62.4 (37.8, 87.0)	28.5 (10.6, 46.4)	16.0 (9.4, 22.5)	18.1 (4.5, 31.7)
Former (719)	271.9 (92.5, 451.3)	168.5+ (58.0, 278.9)	53.5 (-8.2, 115.1)	49.9 (9.9, 90.0)	189.6 (128.0, 251.2)	81.2 (47.6, 114.7)	30.4 (16.0, 44.8)	78.8 (41.7, 115.8)	158.3** (110.9, 205.7)	59.7* (33.8, 85.5)	37.4* (21.1, 53.8)	63.2** (32.8, 93.5)
Current (past-year) (967)	432.7** (292.8, 572.7)	237.0** (135.0, 339.0)	43.4 (24.6, 62.2)	152.4* (74.7, 230.0)	451.1** (298.2, 604.1)	290.9** (161.1, 420.7)	34.7 (23.4, 46.0)	131.1** (77.7, 184.6)	275.2** (152.9, 397.4)	115.7** (51.5, 179.9)	44.3* (16.4, 72.1)	116.0* (30.3, 201.7)
Nondaily (143)	354.4+ (147.3, 561.6)	157.2 (36.6, 277.7)	67.5 (10.0, 125.0)	129.8 (49.2, 210.4)	308.9* (163.6, 454.3)	137.1* (70.4, 203.9)	79.7+ (30.9, 128.5)	92.5 (14.9, 170.0)	69.9 (3.6, 136.3)	23.4 (5.1, 41.8)	37.5 (20.5, 95.4)	9.1 (-1.5, 19.7)
Daily (824)	445.7** (286.6, 604.8)	250.2** (133.6, 366.8)	39.4 (19.7, 59.0)	156.1* (66.6, 245.6)	469.3** (298.1, 640.4)	310.8** (164.8, 456.7)	28.8 (17.9, 39.7)	136.1** (76.6, 195.6)	298.9** (163.6, 434.3)	126.4** (54.9, 197.9)	45.1+ (14.7, 75.4)	128.5* (33.2, 223.7)
Some college												
Never (2,440)	180.6 (133.0, 228.1)	70.1 (42.5, 97.7)	42.5 (30.0, 55.1)	68.2 (44.7, 91.6)	185.3 (146.5, 224.1)	76.8 (60.2, 93.4)	48.5 (33.8, 63.1)	61.4 (43.7, 79.1)	135.8 (96.3, 175.4)	56.1 (28.9, 83.2)	46.2 (32.8, 59.5)	34.5 (13.6, 55.5)
Former (827)	596.5 (29.2, 1163.8)	186.5 (-40.4, 413.4)	73.1 (14.3, 131.8)	336.9 (-14.6, 688.5)	255.6+ (196.1, 315.2)	105.7 (66.5, 144.8)	72.5+ (50.0, 95.0)	77.5 (51.4, 103.5)	194.8* (150.3, 239.4)	57.0 (34.1, 79.9)	79.5* (54.6, 104.5)	59.3 (33.0, 85.5)
Current (past-year) (905)	598.5** (332.3, 864.8)	268.7** (142.4, 395.0)	77.3* (44.1, 110.4)	252.6* (93.4, 411.8)	381.9** (277.0, 486.9)	147.5** (108.0, 186.9)	50.7 (34.7, 66.8)	185.1** (93.0, 277.2)	282.4** (173.4, 391.3)	91.6 (45.9, 137.2)	102.9+ (38.2, 167.6)	88.8* (46.1, 131.6)
Nondaily (228)	489.0** (305.5, 672.4)	181.6* (75.5, 287.4)	77.6 (35.3, 119.9)	229.8* (99.3, 360.3)	499.0** (301.3, 696.7)	204.7* (105.8, 303.6)	85.3 (41.1, 129.6)	210.0* (78.7, 341.3)	386.4* (187.6, 585.1)	143.2 (18.3, 268.1)	99.2 (27.2, 171.3)	144.0+ (29.1, 258.9)
Daily (677)	709.6* (211.9, 1,207.3)	357.0* (131.8, 582.2)	76.9 (25.8, 128.0)	275.7 (-15.5, 566.8)	354.4** (233.7, 475.0)	134.0* (91.5, 176.6)	42.6 (26.0, 59.1)	179.2* (69.6, 288.8)	269.1* (149.2, 389.0)	84.9 (36.3, 133.5)	103.4 (31.0, 174.8)	81.8* (36.3, 127.3)
College degree												
Never (4,082)	294.7 (249.4, 340.1)	124.2 (94.5, 153.9)	73.2 (59.5, 86.9)	99.5 (79.8, 119.2)	243.5 (207.9, 279.2)	92.8 (77.1, 108.5)	79.0 (67.5, 90.5)	72 (55.5, 88.6)	204.7 (154.7, 254.6)	53.1 (38.1, 68.2)	95.3 (69.0, 121.6)	56.6 (32.5, 80.7)
Former (978)	419.7 (54.0, 785.5)	112.8 (42.1, 183.5)	60.3 (7.9, 112.8)	246.5 (-86.2, 579.2)	342.2** (280.9, 403.6)	119.0* (88.7, 149.2)	104.9+ (75.9, 133.9)	118.9* (78.5, 159.3)	276.0* (231.4, 320.5)	55.3 (39.9, 70.6)	112.6 (90.0, 135.2)	109.1** (79.7, 138.5)
Current (past-year) (546)	520.4* (330.5, 710.4)	238.1* (138.3, 337.8)	109.8 (53.3, 166.2)	172.6+ (90.0, 255.1)	500.1** (352.8, 647.3)	179.7** (127.1, 232.2)	111.2+ (73.3, 149.1)	212.7** (110.8, 314.6)	339.0+ (211.4, 466.6)	70.0 (29.7, 110.2)	82.2 (45.4, 119.0)	187.1** (106.9, 267.3)
Nondaily (229)	665.8* (367.8, 963.9)	254.0+ (116.7, 391.2)	162.0+ (66.9, 257.2)	249.8* (111.2, 388.4)	467.1* (299.6, 634.7)	149.3 (81.9, 216.6)	143.4+ (79.7, 207.1)	180.2* (85.8, 274.5)	476.2* (265.1, 687.3)	119.3 (22.8, 215.8)	101.9 (49.5, 154.4)	256.4* (99.6, 413.2)
Daily (317)	348.7 (179.2, 518.1)	219.3 (76.7, 361.8)	48.1 (19.2, 77.0)	81.3 (42.0, 120.5)	519.8** (306.2, 733.3)	197.6** (123.8, 271.5)	92.1 (46.8, 137.5)	231.8* (79.3, 384.3)	277.3 (127.2, 427.3)	48 (11.9, 84.1)	73.4 (26.3, 120.4)	155.9* (67.7, 244.1)

Note. CI = confidence interval. Data are visualized in Figure 3.6.

[a]*Tobacco use* is defined as use of tobacco products other than e-cigarettes (due to survey question wording). *Current tobacco use* is defined as daily or nondaily use in the past year. *Former tobacco use* is defined as ever use in lifetime but not in the past year. *Never tobacco use* is defined as no lifetime use of tobacco.

+$p < .1$ for association comparing alcohol consumption among tobacco use categories (referent = Never). *$p < .05$ for association comparing alcohol consumption among tobacco use categories (referent = Never). **$p < .01$ for association comparing alcohol consumption among tobacco use categories (referent = Never).

Source: 2014–2015 and 2019–2020 U.S. National Alcohol Surveys.

Table 3B.14 Cigarette Smoking History, Categorized by Pack Years, by Lifetime Number of 5+ Drinking Days in the 2014–2015 National Alcohol Survey (*N* = 5,632), by Education, Stratified by Age *(Row Percents)*

Cigarette pack years (unweighted *n*)	Ages 18–29				Ages 30–59				Ages 60 and older			
	0	0.1–499	≥500	*p* value	0	0.1–999	≥1,000	*p* value	0	0.1–999	≥1,000	*p* value[b]
	Lifetime number of 5+ drinking days:[a] Weighted % (95% CI)											
High school or less												
0 (1,175)	73.4 (63.9, 81.0)	17.7 (11.5, 26.2)	9.0 (4.6, 16.6)	<.001	58.4 (51.0, 65.5)	30.5 (23.9, 38.0)	11.1 (7.2, 16.8)	<.001	80.8 (73.7, 86.3)	9.7 (5.9, 15.6)	9.5 (5.7, 15.4)	<.001
0.1–20 (503)	25.1 (14.7, 39.4)	37.1 (24.3, 51.9)	37.9 (24.6, 53.3)		25.6 (19.2, 33.3)	34.9 (26.6, 44.3)	39.5 (31.2, 48.3)		62.8 (51.1, 73.2)	13.0 (6.9, 23.2)	24.1 (15.5, 35.6)	
20.1–40 (163)	*	*	*		20.2 (10.3, 35.9)	29.8 (17.0, 46.8)	50.0 (34.9, 65.1)		40.9 (27.6, 55.7)	26.6 (14.0, 44.7)	32.5 (20.3, 47.5)	
≥40.1 (272)	*	61.9 (30.3, 85.9)	*		23.8 (14.4, 37.3)	25.9 (15.2, 40.4)	50.3 (36.0, 64.7)		43.3 (32.3, 55.0)	18.4 (11.7, 27.7)	38.4 (27.4, 50.7)	
Some college												
0 (820)	47.8 (37.1, 58.7)	40.1 (29.9, 51.2)	12.1 (6.5, 21.6)	<.001	40.2 (33.7, 47.2)	40.7 (33.8, 48.0)	19.1 (13.9, 25.6)	<.001	76.4 (68.6, 82.7)	16.5 (11.3, 23.4)	7.2 (3.8, 13.2)	<.001
0.1–20 (330)	*	65.4 (47.1, 80.0)	26.1 (13.5, 44.4)		16.5 (10.6, 24.8)	42.3 (32.4, 52.9)	41.2 (31.3, 51.9)		60.5 (45.4, 73.8)	18.4 (9.8, 31.7)	21.1 (10.7, 37.4)	
20.1–40 (104)	*	*	*		*	38.3 (21.9, 57.8)	51.4 (32.9, 69.5)		27.1 (15.1, 43.8)	29.3 (16.8, 45.9)	43.6 (27.1, 61.8)	
≥40.1 (183)	*	46.1 (21.5, 72.9)	*		23.7 (12.2, 41.0)	34.9 (19.9, 53.7)	41.4 (24.8, 60.1)		38.0 (26.2, 51.5)	36.6 (24.6, 50.5)	25.4 (16.2, 37.5)	
College degree												
0 (1,323)	27.0 (19.0, 36.9)	65.1 (54.8, 74.2)	7.9 (3.9, 15.2)	<.001	40.7 (35.7, 45.9)	45.7 (40.4, 51.1)	13.6 (10.0, 18.2)	<.001	73.7 (67.1, 79.5)	19.3 (14.5, 25.3)	7.0 (4.0, 11.9)	<.001
0.1–20 (369)	*	52.3 (29.4, 74.2)	47.7 (25.8, 70.6)		13.9 (8.9, 21.1)	38.5 (29.2, 48.8)	47.6 (37.4, 58.0)		47.3 (36.1, 58.8)	33.5 (22.9, 46.0)	19.3 (9.6, 35.2)	
20.1–40 (114)	*	*	*		22.0 (7.2, 50.5)	44.4 (23.8, 67.1)	33.7 (17.8, 54.4)		32.3 (19.8, 48.0)	48.4 (33.5, 63.5)	19.4 (9.6, 35.2)	
≥40.1 (170)	*	*	*		14.0 (6.0, 29.3)	54.4 (37.1, 70.6)	31.6 (18.2, 49.1)		40.9 (29.2, 53.7)	28.7 (19.4, 40.4)	30.4 (19.4, 44.2)	

Note. CI = confidence interval.
[a]*Lifetime 5+ drinking days* is defined as the number of days over lifetime in which participants reported drinking five or more drinks on one occasion. [b]*p* value for bivariate association, obtained through a chi-squared test, between pack year and alcohol use categories within each age and education group.
*Cells with *n* < 10 are suppressed.
Source: 2014–2015 U.S. National Alcohol Survey.

Table 3B.15 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), by Annual Income *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
<$20,000						
Overall (41,895)	24.0 (23.3, 24.6)	37.3 (36.5, 38.0)	14.4 (13.9, 14.9)	16.7 (16.2, 17.2)	7.8 (7.4, 8.1)	
Never (18,156)	44.0 (42.9, 45.2)	30.1 (29.0, 31.1)	12.3 (11.6, 13.0)	11.0 (10.4, 11.7)	2.6 (2.3, 3.0)	<.001
Former (10,537)	9.4 (8.4, 10.4)	46.9 (45.4, 48.3)	19.1 (18.0, 20.3)	17.2 (16.3, 18.2)	7.5 (6.8, 8.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (5,780)	11.0 (9.7, 12.4)	31.5 (29.7, 33.4)	11.5 (10.3, 12.7)	30.6 (28.9, 32.4)	15.5 (14.2, 16.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (1,844)	9.0 (7.2, 11.2)	40.2 (36.8, 43.7)	16.3 (13.8, 19.1)	22.5 (19.8, 25.4)	12.0 (10.1, 14.3)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (5,578)	8.1 (7.0, 9.4)	43.9 (42.0, 45.8)	13.4 (12.1, 14.8)	18.7 (17.3, 20.3)	15.9 (14.6, 17.3)	
$20,000–$49,999						
Overall (65,577)	17.1 (16.6, 17.5)	37.1 (36.6, 37.7)	21.6 (21.2, 22.1)	16.0 (15.6, 16.4)	8.2 (7.9, 8.5)	
Never (26,489)	34.5 (33.6, 35.4)	33.0 (32.1, 33.9)	18.4 (17.8, 19.1)	11.3 (10.8, 11.9)	2.8 (2.5, 3.1)	<.001
Former (21,993)	5.5 (5.1, 6.0)	42.0 (41.0, 43.0)	27.5 (26.7, 28.4)	16.5 (15.8, 17.1)	8.5 (8.0, 9.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (6,836)	7.6 (6.6, 8.6)	27.5 (25.9, 29.2)	16.8 (15.5, 18.2)	30.5 (28.9, 32.2)	17.7 (16.4, 19.0)	
Current (past-30-day) daily smoking, <300 cigarettes/month (2,253)	7.0 (5.5, 9.0)	39.0 (36.0, 42.1)	17.3 (15.2, 19.7)	23.3 (21.0, 25.8)	13.3 (11.5, 15.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (8,006)	4.8 (4.1, 5.7)	41.4 (39.9, 43.0)	17.6 (16.5, 18.9)	18.5 (17.4, 19.7)	17.6 (16.5, 18.8)	

Table 3B.15 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
$50,000–$74,999						
Overall (32,987)	12.2 (11.7, 12.8)	32.3 (31.5, 33.0)	29.1 (28.3, 29.8)	17.1 (16.6, 17.7)	9.3 (8.9, 9.8)	
Never (12,900)	26.7 (25.6, 27.9)	31.2 (30.0, 32.4)	27.4 (26.2, 28.5)	11.8 (11.0, 12.6)	3.0 (2.6, 3.4)	<.001
Former (13,402)	3.3 (2.9, 3.8)	34.1 (32.9, 35.2)	34.3 (33.2, 35.5)	18.3 (17.4, 19.2)	10.1 (9.3, 10.8)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (2,806)	3.6 (2.7, 4.9)	22.4 (20.0, 24.9)	18.2 (16.3, 20.3)	32.8 (30.3, 35.3)	23.0 (20.7, 25.5)	
Current (past-30-day) daily smoking, <300 cigarettes/month (771)	6.7 (3.9, 11.2)	29.1 (24.7, 33.9)	25.0 (20.6, 30.0)	22.6 (18.7, 27.1)	16.6 (13.2, 20.7)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (3,108)	4.3 (3.2, 5.8)	36.0 (33.7, 38.4)	19.7 (17.7, 21.8)	20.2 (18.4, 22.3)	19.8 (18.0, 21.8)	
$75,000+						
Overall (70,225)	8.8 (8.5, 9.1)	24.8 (24.3, 25.3)	37.7 (37.1, 38.2)	17.9 (17.5, 18.3)	10.9 (10.6, 11.2)	
Never (27,678)	20.1 (19.4, 20.8)	26.4 (25.6, 27.2)	36.8 (36.0, 37.6)	12.2 (11.7, 12.7)	4.5 (4.1, 4.9)	<.001
Former (32,319)	1.8 (1.6, 2.0)	23.6 (22.9, 24.3)	42.1 (41.4, 42.9)	19.6 (19.0, 20.2)	12.9 (12.4, 13.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (4,984)	2.1 (1.6, 2.6)	16.3 (14.8, 18.0)	21.9 (20.2, 23.6)	35.2 (33.4, 37.1)	24.5 (22.9, 26.2)	
Current (past-30-day) daily smoking, <300 cigarettes/month (1,096)	3.4 (2.1, 5.4)	25.9 (22.3, 30.0)	24.2 (20.6, 28.3)	25.0 (21.7, 28.7)	21.4 (18.3, 24.9)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (4,148)	2.9 (2.2, 3.9)	32.3 (30.3, 34.4)	22.9 (21.1, 24.8)	21.7 (20.1, 23.4)	20.2 (18.6, 21.9)	

Note. CI = confidence interval. Data are visualized in Figure 3.7.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each income group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.16 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), by Annual Income *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
<$20,000						
Overall (42,012)	23.9 (23.3, 24.6)	37.3 (36.6, 38.0)	14.3 (13.8, 14.8)	16.7 (16.2, 17.2)	7.8 (7.4, 8.1)	
Never (35,426)	26.5 (25.8, 27.3)	37.3 (36.5, 38.1)	14.3 (13.7, 14.8)	15.3 (14.8, 15.9)	6.6 (6.3, 7.0)	<.001
Former (4,981)	6.1 (5.1, 7.4)	38.8 (36.6, 41.0)	16.2 (14.6, 17.8)	24.7 (23.0, 26.5)	14.2 (12.9, 15.6)	
Current (past-30-day) nondaily use (1,125)	12.1 (8.8, 16.3)	29.2 (25.1, 33.7)	7.5 (5.4, 10.2)	33.1 (29.0, 37.5)	18.2 (15.3, 21.4)	
Current (past-30-day) daily use (480)	12.5 (8.4, 18.3)	39.8 (33.1, 46.9)	13.4 (9.2, 19.1)	15.9 (12.3, 20.3)	18.5 (13.6, 24.5)	
$20,000–$49,999						
Overall (65,656)	17.1 (16.6, 17.5)	37.1 (36.6, 37.7)	21.6 (21.1, 22.1)	16.0 (15.7, 16.4)	8.2 (7.9, 8.5)	
Never (53,968)	19.4 (18.9, 19.9)	37.4 (36.7, 38.0)	21.7 (21.2, 22.2)	14.9 (14.4, 15.3)	6.7 (6.4, 7.0)	<.001
Former (8,959)	3.3 (2.7, 4.0)	37.1 (35.6, 38.6)	22.1 (20.8, 23.4)	22.1 (21.0, 23.3)	15.4 (14.4, 16.4)	
Current (past-30-day) nondaily use (1,536)	9.3 (7.2, 12.1)	26.0 (22.9, 29.4)	17.3 (14.7, 20.4)	26.8 (23.9, 30.0)	20.5 (17.9, 23.3)	
Current (past-30-day) daily use (1,193)	4.7 (2.9, 7.5)	35.7 (31.8, 39.8)	17.8 (14.8, 21.4)	22.9 (19.8, 26.4)	18.8 (15.9, 22.1)	

Table 3B.16 (continued)

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
$50,000–$74,999						
Overall (33,017)	12.2 (11.7, 12.8)	32.3 (31.5, 33.0)	29.0 (28.3, 29.8)	17.1 (16.6, 17.7)	9.3 (8.9, 9.8)	
Never (26,222)	14.4 (13.7, 15.0)	33.1 (32.2, 33.9)	29.9 (29.1, 30.7)	15.1 (14.5, 15.7)	7.6 (7.2, 8.1)	<.001
Former (5,223)	2.1 (1.6, 2.9)	29.0 (27.2, 30.8)	26.4 (24.7, 28.1)	26.2 (24.5, 27.9)	16.4 (15.0, 17.8)	
Current (past-30-day) nondaily use (800)	4.8 (2.7, 8.6)	23.0 (18.9, 27.6)	18.9 (15.0, 23.5)	33.7 (29.4, 38.3)	19.7 (16.1, 23.8)	
Current (past-30-day) daily use (772)	2.4 (1.3, 4.3)	31.6 (26.8, 36.8)	19.9 (16.4, 23.9)	24.7 (20.8, 29.0)	21.5 (17.5, 26.1)	
$75,000+						
Overall (70,246)	8.8 (8.5, 9.1)	24.8 (24.3, 25.3)	37.6 (37.1, 38.2)	17.9 (17.5, 18.3)	10.9 (10.6, 11.2)	
Never (54,715)	10.6 (10.2, 11.0)	25.9 (25.4, 26.5)	39.1 (38.5, 39.7)	15.2 (14.8, 15.6)	9.2 (8.8, 9.5)	<.001
Former (12,339)	1.0 (0.8, 1.3)	19.9 (18.9, 21.0)	34.0 (32.8, 35.3)	28.0 (26.9, 29.0)	17.1 (16.2, 18.0)	
Current (past-30-day) nondaily use (1,706)	3.1 (2.1, 4.6)	17.7 (15.1, 20.6)	21.4 (18.6, 24.4)	35.1 (32.1, 38.3)	22.7 (20.1, 25.6)	
Current (past-30-day) daily use (1,486)	1.9 (1.0, 3.4)	23.8 (20.8, 27.0)	22.0 (19.1, 25.3)	29.9 (26.9, 33.1)	22.5 (19.6, 25.7)	

Note. CI = confidence interval.
[a]*Current smokeless tobacco use* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories within each income group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.17 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), by Annual Income *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
<$20,000						
Overall (42,017)	24.0 (23.3, 24.6)	37.3 (36.6, 38.0)	14.3 (13.8, 14.8)	16.7 (16.2, 17.2)	7.8 (7.4, 8.1)	
Never (29,594)	30.9 (30.1, 31.7)	38.0 (37.2, 38.9)	12.6 (12.0, 13.2)	13.4 (12.9, 14.0)	5.1 (4.7, 5.4)	<.001
Former (9,163)	4.5 (3.8, 5.2)	37.7 (36.1, 39.2)	19.5 (18.3, 20.8)	23.7 (22.4, 24.9)	14.7 (13.7, 15.8)	
Current (past-30-day) cigar smoking (3,260)	6.1 (4.8, 7.6)	28.2 (26.0, 30.7)	17.6 (15.8, 19.6)	31.5 (29.3, 33.8)	16.6 (14.9, 18.4)	
$20,000–$49,999						
Overall (65,667)	17.1 (16.6, 17.5)	37.1 (36.6, 37.7)	21.6 (21.1, 22.1)	16.0 (15.7, 16.4)	8.2 (7.9, 8.5)	
Never (44,442)	23.1 (22.5, 23.7)	38.5 (37.9, 39.2)	19.7 (19.1, 20.2)	13.4 (13.0, 13.9)	5.3 (5.0, 5.6)	<.001
Former (17,309)	3.1 (2.7, 3.6)	35.3 (34.3, 36.4)	27.0 (26.0, 28.0)	20.9 (20.1, 21.8)	13.7 (13.0, 14.4)	
Current (past-30-day) cigar smoking (3,916)	3.7 (3.0, 4.6)	25.2 (23.2, 27.4)	20.9 (19.1, 22.9)	29.0 (27.0, 31.0)	21.2 (19.3, 23.1)	
$50,000–$74,999						
Overall (33,025)	12.2 (11.7, 12.8)	32.3 (31.5, 33.0)	29.0 (28.3, 29.8)	17.1 (16.6, 17.7)	9.3 (8.9, 9.8)	
Never (20,849)	18.0 (17.2, 18.8)	35.1 (34.1, 36.0)	27.6 (26.8, 28.5)	13.2 (12.5, 13.8)	6.2 (5.7, 6.7)	<.001
Former (10,458)	1.7 (1.3, 2.1)	28.7 (27.4, 30.0)	32.3 (31.0, 33.6)	23.0 (21.9, 24.2)	14.4 (13.5, 15.4)	
Current (past-30-day) cigar smoking (1,718)	2.5 (1.6, 4.0)	16.8 (14.2, 19.8)	26.5 (23.5, 29.8)	34.0 (30.8, 37.3)	20.2 (17.6, 23.0)	

Table 3B.17 (continued)

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
$75,000+						
Overall (70,249)	8.8 (8.5, 9.1)	24.8 (24.3, 25.3)	37.7 (37.1, 38.2)	17.9 (17.5, 18.3)	10.9 (10.6, 11.2)	
Never (41,136)	14.0 (13.5, 14.5)	29.1 (28.5, 29.8)	36.8 (36.1, 37.5)	13.0 (12.6, 13.4)	7.1 (6.7, 7.5)	<.001
Former (25,462)	1.1 (1.0, 1.3)	19.3 (18.6, 20.0)	40.0 (39.2, 40.9)	23.7 (23.0, 24.4)	15.9 (15.3, 16.5)	
Current (past-30-day) cigar smoking (3,651)	1.5 (1.0, 2.0)	11.2 (9.8, 12.8)	29.3 (27.1, 31.6)	36.4 (34.2, 38.7)	21.6 (19.8, 23.6)	

Note. CI = confidence interval.
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories within each income group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.18 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), by Metropolitan Status *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Non-metropolitan						
Overall (41,243)	14.9 (14.3, 15.5)	38.2 (37.4, 38.9)	23.0 (22.3, 23.6)	15.1 (14.6, 15.6)	8.9 (8.5, 9.3)	
Never (14,061)	32.9 (31.6, 34.1)	32.8 (31.6, 34.0)	21.0 (20.0, 22.1)	9.9 (9.2, 10.6)	3.5 (3.0, 4.0)	<.001
Former (15,603)	5.8 (5.2, 6.4)	41.2 (40.0, 42.4)	28.7 (27.7, 29.9)	15.1 (14.3, 15.9)	9.2 (8.6, 9.9)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month, (4,057)	9.2 (7.8, 10.9)	32.4 (30.1, 34.8)	14.9 (13.2, 16.7)	27.9 (25.8, 30.2)	15.5 (14.0, 17.2)	
Current (past-30-day) daily smoking, <300 cigarettes/month (1,428)	8.6 (6.3, 11.7)	40.3 (36.3, 44.3)	17.5 (14.5, 21.0)	20.5 (17.7, 23.7)	13.1 (10.7, 16.0)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (6,094)	5.7 (4.8, 6.9)	43.7 (41.9, 45.6)	15.7 (14.3, 17.1)	19.4 (18.0, 20.9)	15.5 (14.3, 16.9)	
Metropolitan						
Overall (169,441)	14.1 (13.9, 14.4)	30.5 (30.2, 30.9)	28.6 (28.3, 28.9)	17.3 (17.1, 17.6)	9.4 (9.2, 9.6)	
Never (71,162)	29.2 (28.7, 29.7)	29.3 (28.8, 29.8)	26.2 (25.7, 26.7)	11.9 (11.6, 12.3)	3.4 (3.2, 3.6)	<.001
Former (62,648)	3.5 (3.3, 3.8)	31.3 (30.8, 31.9)	35.4 (34.9, 36.0)	18.8 (18.4, 19.2)	10.9 (10.6, 11.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (16,349)	5.8 (5.3, 6.4)	23.4 (22.4, 24.4)	17.4 (16.6, 18.3)	32.9 (31.9, 33.9)	20.5 (19.6, 21.4)	
Current (past-30-day) daily smoking, <300 cigarettes/month (4,536)	6.3 (5.3, 7.5)	34.0 (32.0, 36.1)	20.0 (18.4, 21.8)	24.0 (22.3, 25.7)	15.6 (14.2, 17.2)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (14,746)	4.9 (4.4, 5.5)	37.7 (36.6, 38.8)	18.9 (18.0, 19.8)	19.7 (18.8, 20.5)	18.9 (18.1, 19.8)	

Note. CI = confidence interval. The 2015–2019 National Survey on Drug Use and Health categorizes participants' counties of residence as large metropolitan, small metropolitan, and nonmetropolitan based on the 2013 Rural/Urban Continuum Codes[81]; large and small metropolitan counties were defined as metropolitan in this analysis. Data are visualized in Figure 3.8.
[a]*Current cigarette* smoking is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within metropolitan status group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.19 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), by Metropolitan Status *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Non-metropolitan						
Overall (41,322)	14.9 (14.3, 15.5)	38.2 (37.5, 38.9)	22.9 (22.3, 23.6)	15.1 (14.6, 15.6)	8.9 (8.5, 9.3)	
Never (29,824)	18.3 (17.6, 19.0)	39.0 (38.2, 39.9)	23.3 (22.5, 24.0)	12.6 (12.1, 13.2)	6.8 (6.4, 7.2)	<.001
Former (8,208)	3.9 (3.2, 4.7)	36.6 (35.0, 38.2)	23.6 (22.2, 25.1)	21.5 (20.2, 22.9)	14.4 (13.4, 15.6)	
Current (past-30-day) nondaily use (1,615)	7.3 (5.2, 10.1)	29.5 (26.1, 33.1)	14.7 (12.1, 17.8)	29.5 (26.3, 32.8)	19.1 (16.5, 22.0)	
Current (past-30-day) daily use (1,675)	6.0 (4.2, 8.5)	36.0 (32.4, 39.7)	19.6 (16.8, 22.8)	23.1 (20.3, 26.1)	15.3 (13.1, 17.8)	
Metropolitan						
Overall (169,609)	14.1 (13.9, 14.4)	30.6 (30.2, 30.9)	28.6 (28.3, 28.9)	17.3 (17.1, 17.6)	9.4 (9.2, 9.6)	
Never (140,507)	16.3 (16.0, 16.6)	31.4 (31.1, 31.8)	28.9 (28.6, 29.3)	15.5 (15.2, 15.7)	7.9 (7.7, 8.1)	<.001
Former (23,294)	2.1 (1.8, 2.4)	26.3 (25.4, 27.1)	28.3 (27.5, 29.2)	26.7 (25.9, 27.5)	16.6 (15.9, 17.2)	
Current (past-30-day) nondaily use (3,552)	6.6 (5.4, 8.1)	21.0 (19.0, 23.0)	18.0 (16.2, 20.0)	33.1 (31.0, 35.3)	21.3 (19.6, 23.2)	
Current (past-30-day) daily use (2,256)	2.7 (1.9, 3.9)	27.2 (24.7, 29.9)	19.5 (17.2, 21.9)	26.7 (24.4, 29.1)	23.9 (21.4, 26.5)	

Note. CI = confidence interval. The 2015–2019 National Survey on Drug Use and Health categorizes participants' counties of residence as large metropolitan, small metropolitan, and nonmetropolitan based on the 2013 Rural/Urban Continuum Codes[81]; large and small metropolitan counties were defined as metropolitan in this analysis.
[a]*Current smokeless tobacco use* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories within metropolitan status group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.20 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), by Metropolitan Status *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Non-metropolitan						
Overall (41,317)	14.9 (14.3, 15.5)	38.2 (37.5, 38.9)	22.9 (22.3, 23.6)	15.1 (14.6, 15.6)	8.9 (8.5, 9.3)	
Never (26,094)	21.2 (20.4, 22.0)	39.8 (38.8, 40.7)	21.2 (20.5, 22.0)	11.6 (11.0, 12.1)	6.2 (5.8, 6.7)	<.001
Former (12,896)	3.0 (2.5, 3.5)	36.6 (35.3, 37.9)	26.9 (25.7, 28.1)	20.5 (19.5, 21.6)	13.0 (12.2, 13.9)	
Current (past-30-day) cigar smoking (2,327)	4.5 (3.3, 6.1)	25.3 (22.8, 28.0)	20.7 (18.0, 23.6)	30.8 (28.1, 33.6)	18.8 (16.6, 21.2)	
Metropolitan						
Overall (169,641)	14.1 (13.9, 14.4)	30.6 (30.2, 30.9)	28.6 (28.3, 28.9)	17.3 (17.1, 17.6)	9.4 (9.2, 9.6)	
Never (109,927)	20.4 (20.1, 20.8)	33.8 (33.4, 34.2)	26.3 (26.0, 26.7)	13.5 (13.2, 13.8)	6.0 (5.8, 6.2)	<.001
Former (49,496)	1.9 (1.8, 2.1)	25.3 (24.7, 25.9)	34.2 (33.6, 34.9)	23.3 (22.8, 23.8)	15.3 (14.8, 15.7)	
Current (past-30-day) cigar smoking (10,218)	3.1 (2.6, 3.6)	18.9 (17.7, 20.0)	24.5 (23.2, 25.8)	33.2 (31.9, 34.5)	20.4 (19.3, 21.5)	

Note. CI = confidence interval. The 2015–2019 National Survey on Drug Use and Health categorizes participants' counties of residence as large metropolitan, small metropolitan, and nonmetropolitan based on the 2013 Rural/Urban Continuum Codes[81]; large and small metropolitan counties were defined as metropolitan in this analysis.
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories within each metropolitan status group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.21 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), Overall and Among Sexual Minority Adults *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall Sample						
Overall (210,684)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (85,223)	29.6 (29.1, 30.1)	29.7 (29.3, 30.2)	25.6 (25.1, 26.0)	11.7 (11.4, 12.0)	3.4 (3.2, 3.6)	<.001
Former (78,251)	3.9 (3.7, 4.1)	32.7 (32.3, 33.2)	34.5 (34.0, 35.0)	18.3 (17.9, 18.7)	10.7 (10.3, 11.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (20,406)	6.3 (5.8, 6.8)	24.6 (23.7, 25.6)	17.1 (16.3, 17.8)	32.2 (31.3, 33.1)	19.8 (19.0, 20.6)	
Current (past-30-day) daily smoking, <300 cigarettes/month (5,964)	6.7 (5.8, 7.8)	35.1 (33.3, 37.0)	19.6 (18.1, 21.2)	23.4 (21.9, 24.9)	15.2 (14.0, 16.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (20,840)	5.1 (4.6, 5.6)	39.0 (38.1, 40.0)	18.2 (17.4, 18.9)	19.6 (18.9, 20.3)	18.1 (17.4, 18.9)	
Sexual minority						
Overall (14,730)	9.7 (9.0, 10.5)	27.5 (26.4, 28.6)	27.1 (26.0, 28.3)	21.2 (20.3, 22.2)	14.5 (13.6, 15.4)	
Never (4,706)	24.7 (22.8, 26.7)	27.1 (25.1, 29.1)	27.4 (25.4, 29.4)	15.3 (13.9, 16.8)	5.6 (4.7, 6.8)	<.001
Former (5,117)	2.4 (1.8, 3.2)	28.0 (26.2, 30.0)	33.3 (31.3, 35.3)	21.4 (19.9, 23.1)	14.8 (13.4, 16.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (2,242)	3.4 (2.3, 5.0)	19.3 (17.1, 21.8)	18.1 (15.8, 20.7)	33.6 (30.9, 36.5)	25.5 (22.9, 28.3)	
Current (past-30-day) daily smoking, <300 cigarettes/month (753)	6.8 (4.3, 10.5)	27.3 (22.9, 32.1)	18.7 (15.2, 22.9)	27.5 (23.3, 32.0)	19.8 (16.3, 23.9)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,912)	3.1 (2.1, 4.5)	35.2 (32.2, 38.4)	19.6 (17.0, 22.5)	20.4 (18.0, 23.1)	21.7 (19.1, 24.4)	

Note. CI = confidence interval. *Sexual minority* is defined as participants who identified as "lesbian or gay" or "bisexual". Data are visualized in Figure 3.9.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.22 Current (Past-30-Day) Alcohol Use by Smokeless Tobacco Use Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,931), Overall and Among Sexual Minority Adults *(Row Percents)*

Smokeless tobacco use status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall Sample						
Overall (210,931)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (170,331)	16.5 (16.3, 16.8)	32.4 (32.1, 32.7)	28.2 (27.9, 28.5)	15.1 (14.9, 15.3)	7.8 (7.6, 7.9)	<.001
Former (31,502)	2.4 (2.2, 2.7)	28.2 (27.5, 29.0)	27.5 (26.7, 28.2)	25.7 (25.1, 26.4)	16.2 (15.6, 16.7)	
Current (past-30-day) nondaily use (5,167)	6.8 (5.7, 8.1)	23.0 (21.3, 24.8)	17.2 (15.7, 18.9)	32.2 (30.5, 34.1)	20.8 (19.3, 22.4)	
Current (past-30-day) daily use (3,931)	3.9 (3.0, 5.0)	30.3 (28.3, 32.5)	19.5 (17.7, 21.4)	25.4 (23.6, 27.3)	20.8 (19.1, 22.8)	
Sexual minority individuals						
Overall (14,743)	9.7 (9.0, 10.5)	27.5 (26.4, 28.6)	27.1 (26.0, 28.3)	21.2 (20.3, 22.2)	14.4 (13.6, 15.3)	
Never (12,441)	10.9 (10.0, 11.8)	27.9 (26.7, 29.1)	27.6 (26.4, 28.9)	20.7 (19.7, 21.7)	13.0 (12.1, 14.0)	<.001
Former (2,004)	1.5 (0.8, 2.7)	26.1 (23.2, 29.2)	25.4 (22.6, 28.4)	24.4 (21.9, 27.2)	22.6 (20.1, 25.4)	
Current (past-30-day) nondaily use (222)	17.2 (8.2, 32.6)	17.2 (11.5, 25.0)	13.8 (8.8, 20.9)	28.2 (20.3, 37.7)	23.6 (16.5, 32.6)	
Current (past-30-day) daily use (76)	*	33.9 (20.8, 50.2)	19.7 (11.0, 32.9)	19.2 (7.6, 40.5)	26.2 (15.4, 41.0)	

Note. CI = confidence interval. *Sexual minority* is defined as participants who identified as "lesbian or gay" or "bisexual".
[a]*Current smokeless tobacco use* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between smokeless tobacco and alcohol use categories.
*Cells with *n* < 10 are suppressed.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.23 Current (Past-30-Day) Alcohol Use by Cigar Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,958), Overall and Among Sexual Minority Adults *(Row Percents)*

Cigar smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall Sample						
Overall (210,958)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (136,021)	20.5 (20.2, 20.8)	34.6 (34.2, 35.0)	25.6 (25.3, 26.0)	13.2 (13.0, 13.5)	6.0 (5.8, 6.2)	<.001
Former (62,392)	2.1 (1.9, 2.3)	27.0 (26.4, 27.5)	33.2 (32.6, 33.7)	22.9 (22.4, 23.3)	14.9 (14.5, 15.3)	
Current (past-30-day) cigar smoking (12,545)	3.3 (2.8, 3.7)	19.7 (18.6, 20.8)	24.0 (22.8, 25.2)	32.9 (31.7, 34.1)	20.2 (19.2, 21.2)	
Sexual minority individuals						
Overall (14,742)	9.7 (9.0, 10.5)	27.5 (26.4, 28.6)	27.1 (26.0, 28.3)	21.2 (20.3, 22.2)	14.5 (13.6, 15.3)	
Never (8,924)	15.3 (14.1, 16.5)	30.3 (28.8, 31.8)	25.9 (24.5, 27.3)	19.1 (18.0, 20.3)	9.5 (8.6, 10.5)	<.001
Former (4,569)	1.0 (0.7, 1.4)	24.0 (22.1, 25.9)	30.1 (28.1, 32.2)	23.5 (21.9, 25.3)	21.4 (19.7, 23.2)	
Current (past-30-day) cigar smoking (1,249)	2.3 (1.3, 3.8)	20.2 (17.3, 23.5)	24.0 (20.7, 27.8)	28.8 (25.5, 32.4)	24.7 (21.3, 28.4)	

Note. CI = confidence interval. *Sexual minority* is defined as participants who identified as "lesbian or gay" or "bisexual".
[a]*Current cigar smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigar smoking and alcohol use categories.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.24 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), Overall and Among Veterans *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall Sample						
Overall (210,684)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (85,223)	29.6 (29.1, 30.1)	29.7 (29.3, 30.2)	25.6 (25.1, 26.0)	11.7 (11.4, 12.0)	3.4 (3.2, 3.6)	<.001
Former (78,251)	3.9 (3.7, 4.1)	32.7 (32.3, 33.2)	34.5 (34.0, 35.0)	18.3 (17.9, 18.7)	10.7 (10.3, 11.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (20,406)	6.3 (5.8, 6.8)	24.6 (23.7, 25.6)	17.1 (16.3, 17.8)	32.2 (31.3, 33.1)	19.8 (19.0, 20.6)	
Current (past-30-day) daily smoking, <300 cigarettes/month (5,964)	6.7 (5.8, 7.8)	35.1 (33.3, 37.0)	19.6 (18.1, 21.2)	23.4 (21.9, 24.9)	15.2 (14.0, 16.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (20,840)	5.1 (4.6, 5.6)	39.0 (38.1, 40.0)	18.2 (17.4, 18.9)	19.6 (18.9, 20.3)	18.1 (17.4, 18.9)	
Veterans						
Overall (11,695)	6.6 (6.0, 7.2)	36.9 (35.8, 38.1)	33.8 (32.7, 35.0)	13.6 (12.8, 14.4)	9.1 (8.4, 9.8)	
Never (2,654)	17.6 (15.7, 19.7)	32.9 (30.6, 35.4)	34.2 (31.8, 36.6)	10.6 (9.1, 12.2)	4.8 (3.8, 6.0)	<.001
Former (6,289)	3.0 (2.4, 3.6)	38.8 (37.2, 40.4)	37.8 (36.2, 39.4)	12.6 (11.6, 13.7)	7.9 (7.1, 8.8)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (874)	3.7 (2.4, 5.7)	27.8 (23.6, 32.5)	21.6 (18.1, 25.6)	28.1 (24.2, 32.4)	18.7 (15.3, 22.8)	
Current (past-30-day) daily smoking, <300 cigarettes/month (283)	6.6 (3.4, 12.5)	38.3 (30.7, 46.4)	29.1 (22.0, 37.5)	14.8 (10.2, 20.9)	11.2 (7.4, 16.8)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,595)	3.7 (2.6, 5.4)	39.7 (36.5, 43.1)	20.2 (17.6, 23.1)	17.9 (15.6, 20.4)	18.5 (16.1, 21.1)	

Note. CI = confidence interval. *Veterans* are participants who indicated they are "now separated or retired from the military". Data are visualized in Figure 3.10.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.25 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), Overall and by Health Condition *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Overall Sample						
Overall (210,684)	14.2 (14.0, 14.5)	31.6 (31.3, 31.9)	27.8 (27.5, 28.1)	17.0 (16.8, 17.2)	9.3 (9.2, 9.5)	
Never (85,223)	29.6 (29.1, 30.1)	29.7 (29.3, 30.2)	25.6 (25.1, 26.0)	11.7 (11.4, 12.0)	3.4 (3.2, 3.6)	<.001
Former (78,251)	3.9 (3.7, 4.1)	32.7 (32.3, 33.2)	34.5 (34.0, 35.0)	18.3 (17.9, 18.7)	10.7 (10.3, 11.0)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (20,406)	6.3 (5.8, 6.8)	24.6 (23.7, 25.6)	17.1 (16.3, 17.8)	32.2 (31.3, 33.1)	19.8 (19.0, 20.6)	
Current (past-30-day) daily smoking, <300 cigarettes/month (5,964)	6.7 (5.8, 7.8)	35.1 (33.3, 37.0)	19.6 (18.1, 21.2)	23.4 (21.9, 24.9)	15.2 (14.0, 16.5)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (20,840)	5.1 (4.6, 5.6)	39.0 (38.1, 40.0)	18.2 (17.4, 18.9)	19.6 (18.9, 20.3)	18.1 (17.4, 18.9)	
Cancer						
Overall (7,548)	9.9 (9.0, 10.8)	37.8 (36.3, 39.2)	35.9 (34.5, 37.3)	9.2 (8.4, 10.1)	7.3 (6.6, 8.1)	
Never (2,370)	22.9 (20.7, 25.2)	32.5 (30.0, 35.0)	34.6 (32.1, 37.2)	6.6 (5.5, 8.0)	3.4 (2.6, 4.5)	<.001
Former (4,015)	3.4 (2.7, 4.2)	38.3 (36.4, 40.3)	39.8 (37.9, 41.8)	9.8 (8.7, 11.0)	8.7 (7.6, 9.9)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (333)	7.4 (4.1, 12.8)	39.5 (32.3, 47.2)	20.0 (14.9, 26.4)	20.1 (14.6, 27.0)	13.1 (9.0, 18.5)	
Current (past-30-day) daily smoking, <300 cigarettes/month (143)	7.0 (3.4, 13.9)	52.8 (41.3, 63.9)	17.6 (10.1, 28.9)	14.1 (8.0, 23.5)	8.6 (4.7, 15.2)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (687)	4.1 (2.6, 6.3)	54.0 (49.0, 58.9)	20.3 (16.5, 24.7)	11.1 (8.5, 14.5)	10.5 (8.0, 13.8)	
Hypertension						
Overall (25,838)	13.2 (12.6, 13.8)	38.3 (37.5, 39.1)	29.1 (28.4, 29.9)	11.2 (10.7, 11.7)	8.1 (7.7, 8.6)	
Never (8,902)	28.2 (27.0, 29.5)	34.8 (33.4, 36.1)	26.0 (24.8, 27.3)	7.3 (6.7, 8.0)	3.7 (3.1, 4.3)	<.001
Former (12,267)	4.5 (4.0, 5.0)	39.8 (38.7, 41.0)	34.5 (33.3, 35.6)	12.0 (11.3, 12.8)	9.2 (8.5, 9.9)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (1,480)	6.5 (4.9, 8.5)	32.5 (29.2, 36.0)	19.5 (16.9, 22.4)	24.0 (21.1, 27.2)	17.5 (14.9, 20.5)	
Current (past-30-day) daily smoking, <300 cigarettes/month (560)	8.4 (5.4, 12.8)	37.9 (32.4, 43.7)	23.3 (18.4, 29.0)	17.8 (14.0, 22.3)	12.7 (9.5, 16.8)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (2,629)	5.8 (4.6, 7.4)	47.1 (44.5, 49.7)	17.0 (15.1, 19.1)	14.9 (13.1, 16.7)	15.2 (13.5, 17.2)	

Table 3B.25 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Heart condition						
Overall (13,958)	12.4 (11.7, 13.1)	41.5 (40.4, 42.6)	29.9 (28.9, 31.0)	9.4 (8.8, 10.0)	6.8 (6.3, 7.4)	
Never (4,567)	27.5 (25.7, 29.3)	36.0 (34.0, 37.9)	27.7 (25.9, 29.6)	6.4 (5.5, 7.3)	2.5 (2.0, 3.2)	<.001
Former (6,673)	5.2 (4.6, 5.9)	43.6 (42.1, 45.2)	34.6 (33.1, 36.1)	9.6 (8.8, 10.5)	7.0 (6.3, 7.8)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (928)	8.5 (6.3, 11.4)	36.9 (32.5, 41.5)	15.9 (13.0, 19.4)	19.2 (16.0, 22.9)	19.4 (16.0, 23.4)	
Current (past-30-day) daily smoking, <300 cigarettes/month (300)	8.8 (5.2, 14.4)	47.2 (39.4, 55.2)	16.2 (11.4, 22.6)	16.8 (11.8, 23.3)	11.0 (6.7, 17.6)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,490)	5.8 (4.3, 7.9)	49.5 (46.0, 53.1)	20.1 (17.3, 23.2)	11.7 (9.7, 14.1)	12.8 (10.8, 15.2)	
Diabetes						
Overall (14,329)	18.5 (17.6, 19.4)	44.6 (43.5, 45.7)	22.2 (21.3, 23.2)	10.4 (9.8, 11.1)	4.3 (3.9, 4.7)	
Never (5,398)	36.6 (34.8, 38.4)	36.8 (35.1, 38.6)	17.3 (16.0, 18.7)	7.6 (6.7, 8.5)	1.8 (1.3, 2.3)	<.001
Former (6,091)	6.5 (5.7, 7.4)	50.2 (48.6, 51.9)	28.1 (26.6, 29.6)	10.7 (9.8, 11.7)	4.5 (3.8, 5.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (933)	11.0 (8.4, 14.4)	40.3 (35.9, 44.9)	13.7 (11.1, 16.8)	22.2 (18.8, 26.2)	12.8 (9.8, 16.6)	
Current (past-30-day) daily smoking, <300 cigarettes/month (344)	11.7 (7.2, 18.3)	43.3 (36.0, 50.9)	23.8 (18.1, 30.5)	12.3 (8.8, 17.0)	9.0 (5.7, 13.8)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (1,563)	6.9 (5.2, 9.0)	52.0 (48.6, 55.4)	17.7 (15.1, 20.5)	14.7 (12.5, 17.3)	8.8 (7.1, 10.8)	

Note. CI = confidence interval. *Self-reported health conditions* are defined as a condition that a doctor or other health care professional has ever told participants that they had, including "cancer," "any kind of heart condition or heart disease," "diabetes or sugar diabetes," and "hypertension, also called high blood pressure." Data are visualized in Figure 3.11.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each health condition group.
Source: 2015–2019 National Survey on Drug Use and Health.

Table 3B.26 Current (Past-30-Day) Alcohol Use by Cigarette Smoking Status in the 2015–2019 National Survey on Drug Use and Health (*N* = 210,684), Among People with Mental Distress *(Row Percents)*

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
None/low (Score of 0–4 on Kessler K6 non-specific distress scale)						
Overall (128,719)	15.0 (14.7, 15.2)	31.2 (30.8, 31.6)	29.8 (29.4, 30.2)	15.9 (15.6, 16.2)	8.2 (8.0, 8.4)	
Never (55,297)	29.9 (29.3, 30.5)	29.5 (29.0, 30.1)	26.6 (26.0, 27.1)	10.9 (10.6, 11.3)	3.1 (2.9, 3.3)	<.001
Former (49,707)	3.9 (3.7, 4.2)	32.3 (31.7, 32.9)	36.7 (36.1, 37.3)	17.3 (16.9, 17.8)	9.8 (9.4, 10.2)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month, (10,207)	7.3 (6.6, 8.1)	25.6 (24.3, 27.0)	17.7 (16.7, 18.9)	32.0 (30.7, 33.3)	17.4 (16.3, 18.4)	
Current (past-30-day) daily smoking, <300 cigarettes/month (3,083)	8.4 (7.0, 10.1)	34.9 (32.5, 37.4)	20.7 (18.6, 23.0)	22.4 (20.5, 24.6)	13.6 (11.9, 15.4)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (10,425)	5.8 (5.1, 6.5)	37.1 (35.8, 38.5)	20.1 (19.0, 21.2)	19.4 (18.4, 20.5)	17.6 (16.6, 18.7)	
Moderate (Score of 5–12 on Kessler K6 non-specific distress scale)						
Overall (64,921)	12.6 (12.2, 13.1)	31.8 (31.3, 32.4)	24.7 (24.2, 25.2)	19.2 (18.8, 19.7)	11.6 (11.3, 12.0)	
Never (24,488)	28.7 (27.8, 29.6)	29.7 (28.8, 30.6)	23.9 (23.1, 24.7)	13.5 (12.9, 14.1)	4.3 (3.9, 4.7)	<.001
Former (23,527)	3.6 (3.2, 4.0)	33.2 (32.3, 34.1)	30.1 (29.3, 31.0)	20.5 (19.8, 21.2)	12.7 (12.1, 13.3)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (7,406)	4.4 (3.8, 5.2)	22.8 (21.4, 24.3)	16.7 (15.5, 18.0)	32.4 (30.9, 34.0)	23.6 (22.3, 25.1)	
Current (past-30-day) daily smoking, <300 cigarettes/month (2,086)	4.4 (3.3, 5.9)	34.1 (31.1, 37.2)	19.4 (17.1, 22.0)	24.7 (22.2, 27.4)	17.3 (15.2, 19.7)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (7,414)	4.0 (3.3, 4.9)	40.6 (39.0, 42.2)	16.3 (15.1, 17.6)	20.2 (18.9, 21.4)	18.9 (17.7, 20.2)	

Table 3B.26 (continued)

Cigarette smoking status[a] (unweighted *n*)	Never alcohol use[b]	Former alcohol use	Past-30-day alcohol use within guidelines[c]	Past-30-day binge, no risky weekly use[d]	Past-30-day risky weekly use[e]	*p* value[f]
	Alcohol use: Weighted % (95% CI)					
Severe (Score of 13–24 on Kessler K6 non-specific distress scale)						
Overall (17,044)	12.8 (12.0, 13.6)	35.5 (34.4, 36.6)	18.8 (17.9, 19.7)	20.3 (19.5, 21.2)	12.6 (11.9, 13.3)	
Never (5,438)	30.0 (28.2, 31.9)	33.4 (31.6, 35.4)	18.8 (17.3, 20.5)	13.8 (12.5, 15.2)	3.9 (3.3, 4.7)	<.001
Former (5,017)	4.8 (3.9, 5.9)	36.8 (34.7, 39.0)	24.0 (22.2, 25.8)	21.4 (19.8, 23.1)	13.1 (11.8, 14.4)	
Current (past-30-day) nondaily smoking, <300 cigarettes/month (2,793)	7.0 (5.6, 8.7)	25.2 (22.8, 27.6)	14.8 (12.9, 16.8)	32.4 (29.9, 35.1)	20.7 (18.6, 22.9)	
Current (past-30-day) daily smoking, <300 cigarettes/month (795)	4.2 (2.6, 6.9)	39.2 (34.1, 44.5)	14.2 (11.2, 17.7)	24.5 (20.5, 29.0)	18.0 (14.5, 22.0)	
Current (past-30-day) daily or nondaily smoking, ≥300 cigarettes/month (3,001)	5.0 (3.8, 6.6)	43.6 (41.0, 46.2)	14.2 (12.6, 16.0)	18.8 (17.0, 20.8)	18.4 (16.6, 20.4)	

Note. CI = confidence interval. *Mental distress* levels are based on scores on the Kessler K6 non-specific distress scale, where a score of 0–4 = none/low, 5–12 = moderate, and 13–24 = severe. Data are visualized in Figure 3.12.
[a]*Current cigarette smoking* is defined as use in the past 30 days. [b]*Never* and *former alcohol use* are based on lifetime alcohol use and use in the past 30 days. [c]*Alcohol use within guidelines* is defined as alcohol use without binge drinking or risky weekly use in the past 30 days. [d]*Binge, no risky weekly use* is defined as drinking ≥4/5 drinks on an occasion for women/men without exceeding 7/14 drinks per week for women/men in the past 30 days. [e]*Risky weekly use* is defined as drinking more than 7/14 drinks per week for women/men in the past 30 days. [f]*p* value for bivariate association, obtained through a chi-squared test, between cigarette smoking and alcohol use categories within each K6 category.
Source: 2015–2019 National Survey on Drug Use and Health.

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Chapter 4
Cancer Epidemiology of Tobacco and Alcohol Co-Use

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Chapter 4
Cancer Epidemiology of Tobacco and Alcohol Co-Use

Introduction

Tobacco and alcohol independently cause cancer at numerous sites, and their combined use results in additive or multiplicative increases in risk for some types of cancer.[1-4] This chapter summarizes the epidemiologic research examining associations between the use of tobacco and alcohol products with cancer risk, as well as interactions between tobacco and alcohol that influence cancer risk, including established synergistic increases in risk for cancers of the aerodigestive tract. For tobacco use, this chapter focuses on cigarette smoking because most of the extant literature pertaining to the cancer-related risks of combined tobacco and alcohol use has focused on this product. This does not diminish the importance of understanding the cancer-related risks posed by other tobacco products in the context of alcohol use, and research is needed to investigate this area. In this chapter, "alcohol" is used both to refer to alcohol-containing beverages and to ethanol, the psychoactive ingredient and primary carcinogen contained in alcoholic beverages.

One important motivation for studying tobacco and alcohol co-use is the presence of synergy between tobacco and alcohol exposures in risk of aerodigestive tract cancers (and potentially for other sites).[5-7] Synergy occurs when the interaction between two factors (e.g., tobacco and alcohol use) results in a total effect (e.g., cancer risk) that is greater than the sum of the individual effects.[8,9] Synergy can be assessed with statistical tests for interaction in regression models[9] and is sometimes examined by calculating a synergy index.[5-7,10-12] Further details concerning different types of synergy, synergy indices, and statistical approaches to synergy are available elsewhere.[10]

The strong epidemiological evidence for the presence of synergy makes it especially important to address tobacco and alcohol co-use for cancer prevention and control. As discussed in detail in chapter 5, plausible mechanisms underlying synergy between tobacco and alcohol include increased exposure to acetaldehyde derived both from tobacco smoke and ethanol metabolism, alcohol-induced increases in membrane permeability that increase exposure to carcinogens from tobacco and alcohol and increased reactive oxygen species and chronic inflammation. Furthermore, successful efforts to reduce tobacco and/or alcohol use would be especially beneficial in the context of co-use. For example, a 2024 systematic review conducted by Burton and colleagues[7] aimed to quantify the independent and joint associations of exposure to alcohol consumption, cigarette smoking, and excess weight across health outcomes and identify whether these associations are synergistic. They reported a synergy index for smoking and alcohol use on head and neck cancer death/disease that was 3.78 times greater than the additive effect of each exposure (95% confidence interval [CI] = 2.61–5.48). Their findings suggest that true population-level risk may be underestimated and that, compared to individuals with one risk factor, those with multiple risk factors would experience a greater absolute risk reduction for an intervention that targets a single exposure. Tobacco cessation alone is known to reduce cancer risk at multiple sites, and reviews published as of 2026 indicate that reduction/cessation of

alcohol use also reduces risk of head and neck cancers (see the section "Reduction in Alcohol Consumption and Cancer Risk" in this chapter; smoking cessation in the context of alcohol use is discussed in detail in chapter 7).

Major reports and reviews have examined and summarized existing research on the associations between alcohol use, tobacco use, and cancer risk, including the 2014 Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*,[3] the International Agency for Research on Cancer (IARC) *Monographs on the Evaluation of Carcinogenic Risks to Humans*[1,2,13-15] and *Handbooks of Cancer Prevention*,[16] and the American Institute for Cancer Research (AICR) and World Cancer Research Fund (WCRF) Continuous Update Project.[4] This chapter summarizes research with attention to (1) the scientific evidence on the synergistic risk of cancer associated with the use of both tobacco and alcohol products and (2) different population subgroups and types of alcohol or tobacco products used. The literature review search terms and methods used throughout this chapter are described in Appendix 4A.

This chapter seeks to identify gaps in the understanding of the cancer epidemiology of tobacco and alcohol use in relation to changes in prevalence of use and product types. As previously noted, most of the epidemiological research summarized in this chapter examines cigarettes, as there are far fewer epidemiological studies of tobacco and alcohol cancer risk for other tobacco products. Additionally, over the past 20 years, there have been considerable changes in both the tobacco and alcohol marketplaces, with many new products being added to the market.[17-19] In addition to cigarettes, cigars, and smokeless tobacco products, the tobacco product market now includes products such as e-cigarettes, which are available in thousands of flavors; nicotine pouches; and heated tobacco products (e.g., IQOS).[19,20] Although products such as beer, wine, and distilled spirits continue to predominate, the alcohol market has also expanded in the past 20 years with the addition of low-calorie options (many of which feature sweeteners like stevia and erythritol), craft products, and alcohol-containing seltzers and beer-fruit infusions.[21] Like newer tobacco products, many of these newer alcohol products come in appealing flavors and are relatively inexpensive compared to wine and spirits, making them especially attractive to young people.[22,23] Tobacco and alcohol products also vary in the amount of nicotine and alcohol they contain, respectively. For example, FDA-authorized e-cigarette products contain nicotine concentrations ranging from 2% to 5% and come in tobacco and menthol flavors,[24,25] but there are many e-cigarette products in the marketplace that come in large capacity cartridges with 5% or higher nicotine concentration.[17,26] As of 2026, the nicotine concentration in FDA-authorized nicotine pouches range from 3 mg to 9 mg, but companies have sought authorization for products with nicotine concentrations of up to 12 mg.[25,27] With regard to alcohol content, beer, hard seltzers, and canned cocktails typically contain 4%–6% alcohol by volume (ABV),[28,29] but products are available at both higher levels (e.g., canned cocktails with >20% ABV)[30] and lower levels (e.g., beer with 2%–3% alcohol).[28] There are also high-alcohol-content products that particularly appeal to youth, such as Four Loko.[31,32] Alcohol per volume of spirits is typically 40%.[33] Furthermore, similar to how tobacco products have been marketed for decades, the marketing of alcohol products has expanded to more explicitly target specific demographic subgroups, such as women and sexual minorities.[17-19,34,35] Some alcohol marketing has also involved health-related claims concerning calories, gluten, sugar, and health outcomes

(including diabetes). In 2022, the U.S. Alcohol and Tobacco Tax and Trade Bureau (TTB) released a reminder that "TTB advertising regulations [for alcohol products] prohibit any health-related statement that is untrue in any particular or tends to create a misleading impression as to the effects of consumption on health."[36]

The internet, social media, mobile apps, and other related technologies have changed consumer behavior and increased the capacity for targeted marketing for consumer products, including tobacco and alcohol products.[37-39] Changes in the product market are accompanied by changes in the populations using these products, including the disproportionate use of tobacco products (particularly cigarettes) among people in medically underserved communities, the increase and substantial prevalence of e-cigarette use among youth and young adults, and changes in the alcohol market with significant decreases in alcohol use among adolescents and young adults in the past 20 years (see chapter 2).[40] Understanding the circumstances and motivations that are driving product use changes, such as targeted marketing by the tobacco industry to promote cigarette smoking among specific subpopulations[41] and increased health consciousness among young adults,[42] are essential to understanding the patterns of use of these products and their association with cancer risk. Addressing the needs of underserved populations is an important priority across the medical and research communities, although more research is needed to understand and address disparities in health behaviors and in cancer incidence and mortality.

This chapter does not present new meta-analyses or a formal systematic review. Rather, the main aims are (1) to summarize existing literature concerning the associations between tobacco and alcohol and cancer and (2) to assess whether synergy between alcohol and tobacco is present for a given cancer site. Knowledge of underlying biological mechanisms is critical to interpreting the results from epidemiology studies. As such, this chapter uses the mechanisms presented in chapter 5 to help interpret the empirical evidence from cancer epidemiology studies and assess whether tobacco and alcohol are associated with each cancer site and whether they together pose synergistic risk.

Cancer Epidemiology of Tobacco, Alcohol, and Their Co-Use

The causal relationship between the use of tobacco and alcohol individually and the risk of cancer is well established and includes increased risk for many of the most common cancers including lung, colon, female breast, head and neck, and bladder cancer. Although their combined effects have not been as well studied, convincing evidence demonstrates synergistic increases in the risk of aerodigestive tract cancers with tobacco and alcohol co-use. This section reviews the extensive epidemiological evidence regarding the relationship between tobacco and alcohol use, separately and together, and the risk of cancer. To further contextualize the magnitude of impact that tobacco and alcohol have on population-level health in relation to cancer, the incidence rates for cancer sites causally related to tobacco and/or alcohol use are presented in Table 4.1.

Table 4.1 Rates of Cancer Incidence and Mortality per 100,000

Disease site	Cancer incidence (age-adjusted) Rate per 100,000 (95% CI), 2018–2022			Cancer mortality (age-adjusted) Rate per 100,000 (95% CI), 2019–2023		
	Overall	Female	Male	Overall	Female	Male
Esophagus	4.2 (4.1, 4.2)	1.7 (1.7, 1.8)	7.1 (7.0, 7.2)	3.7 (3.7, 3.7)	1.4 (1.4, 1.4)	6.5 (6.4, 6.5)
Larynx	2.5 (2.5, 2.6)	1.0 (0.9, 1.0)	4.4 (4.3, 4.5)	0.9 (0.9, 0.9)	0.3 (0.3, 0.3)	1.6 (1.5, 1.6)
Oral cavity and pharynx	11.6 (11.6, 11.7)	6.6 (6.5, 6.7)	17.4 (17.2, 17.5)	2.7 (2.6, 2.7)	1.4 (1.4, 1.5)	4.1 (4.0, 4.1)
Colon and rectum	37.1 (37.0, 37.2)	32.6 (32.4, 32.8)	42.3 (42.1, 42.5)	12.9 (12.8, 12.9)	10.8 (10.7, 10.9)	15.3 (15.2, 15.4)
Liver and intrahepatic bile duct	9.4 (9.4, 9.5)	5.5 (5.5, 5.6)	13.9 (13.8, 14.0)	6.6 (6.6, 6.7)	4.3 (4.2, 4.3)	9.4 (9.3, 9.5)
Stomach	7.3 (7.2, 7.3)	5.7 (5.6, 5.8)	9.2 (9.1, 9.3)	2.7 (2.7, 2.7)	2.0 (2.0, 2.1)	3.5 (3.5, 3.6)
Urinary bladder	18.0 (17.9, 18.1)	7.7 (7.6, 7.8)	31.2 (31.0, 31.4)	4.1 (4.1, 4.2)	2.0 (2.0, 2.1)	7.1 (7.1, 7.2)
Acute myeloid leukemia	4.3 (4.2, 4.3)	3.6 (3.6, 3.7)	5.1 (5.1, 5.2)	2.7 (2.7, 2.7)	2.1 (2.1, 2.1)	3.4 (3.4, 3.5)
Cervix uteri	-	7.7 (7.6, 7.8)	-	-	2.2 (2.1, 2.2)	-
Kidney and renal pelvis	17.5 (17.5, 17.6)	12.0 (11.9, 12.1)	23.8 (23.7, 24.0)	3.4 (3.4, 3.5)	2.1 (2.1, 2.1)	5.1 (5.0, 5.1)
Lung and bronchus	47.8 (47.7, 48.0)	43.9 (43.7, 44.1)	53.1 (52.9, 53.4)	31.5 (31.4, 31.6)	27.1 (27.0, 27.2)	37.2 (37.0, 37.3)
Pancreas	13.8 (13.7, 13.8)	12.3 (12.2, 12.4)	15.5 (15.4, 15.6)	11.3 (11.2, 11.3)	9.9 (9.8, 9.9)	12.9 (12.8, 13.0)
Breast	-	130.8 (130.4, 131.1)	1.2 (1.2, 1.3)	-	19.2 (19.1, 19.3)	0.3 (0.3, 0.3)

Note. CI = confidence interval. Rates are per 100,000 and age-adjusted to the 2000 U.S. Standard Population. Cancer sites are listed in the same order as in Table 4.3. A dash (-) indicates that data are not available.
Source: SEER*Explorer.[43]

Epidemiology of Tobacco Use and Cancer Risk

The evidence summarizing the health risks from smoking and the benefits of cessation on cancer outcomes are documented in the 2014 Surgeon General's report, *The Health Consequences of Smoking-50 Years of Progress*[3]; the 2020 Surgeon General's report, *Smoking Cessation: A Report of the Surgeon General*[44]; National Cancer Institute (NCI) *Monograph 23: Treating Smoking Cessation in Cancer Patients*[45]; and the National Toxicology Program's *15th Report on Carcinogens*.[46] Conclusive evidence demonstrates that cigarette smoking causes cancers at multiple sites, including cancers of the oral cavity, pharynx, larynx, esophagus, lung (including trachea and bronchus), stomach, liver, pancreas, kidney and ureter, bladder, cervix, colorectum, and blood (acute myeloid leukemia).[3] Cigarette smoking is estimated to have caused about 170,000 cancer deaths, or approximately 28.5% of all cancer deaths, in the United States in

2019; it is estimated that in 2019 about 19.3% of incident cancers, or about 350,000 cases, were attributable to cigarette smoking.[47] Globally, an estimated 2.5 million cancer-related deaths were attributable to cigarette smoking in 2019.[48] Additionally, cigarette smoking also causes many other conditions including cardiovascular disease, respiratory disease, adverse reproductive outcomes, and diabetes.[3,47] Furthermore, exposure to secondhand smoke causes many diseases, as well as increasing the risk of lung cancer among people who do not smoke and asthma among youth.[3] Tobacco smoke contains thousands of harmful chemicals, including a complex mixture of carcinogens, that contribute to the development of many serious health conditions.[3,47,49,50]

Tobacco cessation improves health status and quality of life and decreases risks for morbidity and mortality at any age.[44] The 2020 Surgeon General's report[44] concluded that smoking cessation reduces the risk of premature death and can add as much as a decade to life expectancy. The report also concluded that quitting smoking reduces the risk of many adverse health effects, including adverse reproductive health outcomes, cardiovascular diseases, chronic obstructive pulmonary disease, and cancer. Regarding cancer, the evidence is sufficient to infer that smoking cessation reduces the risk of cancers of the oral cavity, pharynx, larynx, esophagus, lung (including trachea and bronchus), stomach, liver, pancreas, kidney and ureter, bladder, cervix, colorectum, and blood (acute myeloid leukemia).[44] The 2020 Surgeon General's report also found that evidence is sufficient to infer that the relative risk (RR) of lung cancer decreases steadily after smoking cessation when compared to people who continue to smoke. For example, at 10 to 15 years after smoking cessation, the lung cancer risk of individuals who quit is about half that of individuals of the same age who continue to smoke.[44]

Tobacco and Cancer by Demographic Subgroup

Tobacco use prevalence has slowly trended downward among the general population in the United States over many decades (see chapter 2). In 2024, a record low of 9.9% of U.S. adults reported current use of cigarettes compared with 20.9% in 2005.[51,52] However, many population subgroups continue to have a higher prevalence of tobacco use and associated diseases relative to the population overall, including people living in rural communities, military veterans, sexual minorities, people with lower educational attainment or income level, people without health insurance, people who have mental or behavioral health conditions, people with alcohol and other substance use disorders, and certain racial and/or ethnic groups, most notably American Indian and Alaska Native populations.[53] For example, the cigarette smoking prevalence among African American and Hispanic populations in 2022 differed by sex compared to White populations, with higher prevalence of smoking among African American men compared to non-Hispanic White men, but lower prevalence among African American women and Hispanic men and women than White women.[54]

As described in NCI Tobacco Control Monograph 22, *A Socioecological Approach to Addressing Tobacco-Related Health Disparities*, many factors contribute to the prevalence of tobacco use, including the interaction of factors at the individual, interpersonal, community/neighborhood, societal, and policy level.[55] Tobacco and alcohol industry practices, including marketing and product formulation also influence prevalence and product type used by different demographic groups.[55-57] These factors continue to influence tobacco use prevalence in these population groups, helping to maintain higher levels of tobacco use and negative health outcomes, and lower rates of decline in prevalence, compared with the general population.

Similarly, key indicators of smoking cessation, including quit attempts, receiving advice to quit from a health professional, and use of evidence-based cessation therapies, also vary across populations, with lower prevalence across certain subgroups defined by educational attainment, poverty status, age, health insurance status, race, ethnicity, and geography.[44,53,58]

Tobacco and Cancer by Tobacco Product

Although attention has focused primarily on cigarette smoking, other tobacco products, including cigars, cigarillos, pipes, and hookah, contain nicotine and toxic compounds and expose the user and the bystander to carcinogens and other harmful constituents. Cigars and pipes are established causes of cancer[2,3,15] and substantial evidence supports that cigarillo and hookah use are also causes of cancer.[2,59-61] Hookah smoking was traditionally viewed as a relatively safe form of tobacco use,[62] but studies demonstrate that hookah use results in exposure to significant levels of carbon monoxide[63] and many harmful toxicants, including carcinogens.[2,3,59,61,64,65] A growing body of epidemiological evidence links hookah use to lung, oral, and esophageal cancers.[66,67] Smokeless tobacco products also cause cancer, including cancers of the oral cavity, esophagus, and pancreas.[3,14,68,69] Research on the cancer risk of alcohol in combination with tobacco products other than cigarettes is very limited.

Surveys from 2013 to 2016 indicated that a substantial proportion of individuals, particularly young people, engaged in polytobacco use (i.e., the use of multiple tobacco products).[70,71] Results from the 2024 National Youth Tobacco Survey found that 8.1% of middle and high school students in the United States reported current use of any tobacco product; 3.0% reported use of multiple tobacco products, and e-cigarettes were the most frequently reported product currently used (5.9%).[72] The Population Assessment of Tobacco and Health Study in the United States found that among participants who smoked cigarettes, polytobacco use was more common among youth (12–17 years) and young adults (18–24 years), than among adults 25 years and older.[70] Mattingly and colleagues (2021)[73] examined adult (18 years and older) tobacco use patterns using data from the 2014–2015 and 2018–2019 Tobacco Use Supplement to the Current Population Survey and the 2015–2019 National Health Interview Survey, 2 nationally representative U.S. surveys. They found that both dual use of electronic nicotine delivery systems (ENDS) and cigarettes, as well as cigarettes with polytobacco use (ENDS, cigars/cigarillos/little filtered cigars, and pipe or hookah) decreased over time; in contrast polytobacco use without cigarettes increased over time. These complex trends also varied by age, sex, and race and/or ethnicity. These data are important for understanding tobacco use trends across the population; however, as of 2026, little evidence exists concerning the effects of co-use of e-cigarettes, or polytobacco use and alcohol, on cancer risk.

Epidemiology of Alcohol Use and Cancer Risk

The two major authoritative sources for summary and adjudication of evidence concerning the causal and dose–response relationships between alcohol and cancer are IARC and the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR). IARC develops monographs assessing the mechanistic and epidemiological evidence for whether an exposure is

related to cancer.[74] IARC monographs do not assess dose–response relationships, though this topic is discussed as part of their assessment of causality. By contrast the WCRF/AICR Continuous Update Project focuses on the epidemiological evidence and includes meta-analyses assessing dose–response relationships.[4]

Globally, about 741,000, or 4.1%, of all new cases of cancer in 2020 were attributable to alcohol consumption.[75] In 2019 in the United States, 96,730 (5.4%) of incident cancers were estimated to be caused by alcohol consumption with 42,400 of these in men and 54,330 in women.[47] For cancer mortality, an estimated 20,216 deaths were attributable to alcohol during 2020–2021, with 14,562 in men and 5,654 in women.[76] Alcohol is the third leading modifiable risk factor for cancer in the United States, after cigarette smoking and excess bodyweight, highlighting the importance of efforts to reduce alcohol consumption for cancer prevention.[47]

The large body of evidence concerning alcohol and cancer demonstrates that consumption of alcoholic beverages causes increased risk for oral and pharyngeal cancer, laryngeal cancer, esophageal cancer (specifically, squamous cell carcinoma),[77] female breast cancer in women, colorectal cancer, and liver cancer.[1] WCRF/AICR also judges the evidence linking stomach cancer and alcohol to be strong.[4] Importantly, the shape of the dose–response curves for alcohol and the risk of specific cancers appears to differ by site. For example, very strong evidence of increased risk is observed even at low levels of drinking for female breast cancer, one of the most common cancers in the United States.[78] Evidence is also growing that head and neck cancer risk increases with consumption of <1 drink per day[79] and a 2023 meta-analysis reports elevated risk for colorectal cancer with reported consumption of <1 U.S. standard drink per day.[80]

Two meta-analyses of alcohol consumption and liver cancer reported increased risk at one or more drinks per day.[81,82] In contrast, the WCRF/AICR concluded that risk for liver and stomach cancer does not increase until three drinks per day.[4] This suggests that low levels of alcohol consumption may increase the risk for some, but not all, cancers related to alcohol consumption.[1] A 2021 meta-analysis reported a linear dose–response relationship for stomach cancer and alcohol consumption with risk increasing linearly beginning at consumption of 5 grams per day of alcohol.[83] Finally, a large 2023 meta-analysis found that less than one U.S. standard drink per day was associated with increased risk of esophageal, colorectal, and female breast cancers, as well as increased risk of other cancers at higher levels of alcohol consumption.[6]

Some studies have found an association between alcohol and an increased risk for other cancer sites, including ovary, prostate, uterus, and bladder, but the evidence for these associations is deemed inconclusive.[2] More evidence is needed to understand the mechanisms potentially underlying associations between alcohol and increased risk of skin cancers (both melanoma and nonmelanoma skin cancers) as well as cancers of the prostate and pancreas. In 2010, the evidence linking alcohol and pancreatic cancer was rated as "limited."[2] In 2025, a study that used a large, pooled analysis of individual-level data from 30 cohorts with >10,000 incident cases of pancreatic cancer found significant increases in risk associated with consumption of 30–59 grams of ethanol per day and even greater risk at higher levels of consumption.[84] Evidence for associations between alcohol consumption and melanoma also appears to be strengthening,[85] but the relationship with prostate cancer remains unclear, with meta-analyses published in 2020[86] and 2023[80] reporting somewhat different results based on alcohol type consumed and cancer type.

More research is also needed on mechanisms underlying associations between alcohol consumption and increased risk for liver and female breast cancer, although major aspects of these mechanisms have been identified.[87] Alcohol consumption is associated with decreased risk of kidney cancer (Strong Evidence/Probable grading by WCRF/AICR)[4] and non-Hodgkin lymphoma.[4,88] Inverse associations with kidney cancer could be related to alcohols' diuretic effects, insulin sensitivity, or perhaps poorly measured or unmeasured confounding; further epidemiological studies could help clarify the potential importance of these mechanisms.[89-91] More research is also needed to examine potential mechanisms for an inverse association between alcohol use and non-Hodgkin lymphoma.[92] Nevertheless, the reduced number of cases of kidney cancer and non-Hodgkin lymphoma attributable to alcohol consumption are much lower than the increased number of cancers observed at other sites.

Compared with people who do not drink, regular consumption of about 50 grams of ethanol per day is associated with an approximately 3-fold increase in risk for oropharyngeal cancers, a 2-fold risk for cancers of the larynx and esophagus, and a risk ratio (RR) of 1.5 for female breast cancer and 1.4 for colorectal cancer.[1] For reference, 50 grams of alcohol per day corresponds to about 3.5 U.S. standard drinks per day.[1,33] Ten grams of ethanol per day is associated with a RR of 1.15 (95% CI, 1.09–1.22) for oral cavity cancer, 1.13 (95% CI, 1.05–1.21) for pharynx cancer, 1.09 (95% CI, 1.05–1.13) for larynx cancer, 1.07 (95% CI, 1.05–1.08) for colon cancer, 1.04 (95% CI, 1.01–1.08) for premenopausal female breast cancer, and 1.08 (95% CI, 1.05–1.10) for postmenopausal female breast cancer,[75] providing further evidence that low level alcohol use is carcinogenic. Table 4.2 summarizes results of the 2010 IARC monograph, *Monographs on the Evaluation of Carcinogenic Risks to Humans: Alcohol Consumption and Ethyl Carbamate (Volume 96).* Rumgay and colleagues (2021)[87] provide a thorough review of the epidemiology and biological mechanisms associated with alcohol use and cancer. Their review highlights the evolving nature of the evidence linking alcohol and some of the cancer sites, including sparse or inconsistent evidence. For example, the WCRF/AICR Continuous Update Project concluded that there was limited—but suggestive—evidence that alcoholic drinks increase risk of lung, pancreatic, and skin cancers.[4]

Trends in Alcohol Use by Demographic Subgroup

Research on alcohol consumption consistently shows variations in drinking by race and/or ethnicity, sex, age, socioeconomic status (SES), and other demographic characteristics. Associations between demographic characteristics and alcohol use differ from tobacco in several important ways. Most notably, people of higher SES report drinking more alcohol than those of lower SES, but those of lower SES who drink tend to report more heavy drinking and greater negative alcohol-related consequences.[93-95] Patterns of beverage consumption are changing in association with reductions in alcohol consumption, the COVID-19 pandemic, and changes in types of products available along with marketing practices and price.[21,96-100] Detailed descriptions of tobacco and alcohol use and co-use for the population overall and for specific demographic groups are presented in chapters 2 and 3.

Secular trends in alcohol consumption in the population overall and by demographic group will influence the future incidence of cancers related to alcohol and to the co-use of tobacco and alcohol. Efforts at modeling the effects of these changing patterns of use with approaches such as those used by the Cancer Intervention and Surveillance Modeling Network (CISNET) project for

tobacco and cancer screening would be valuable for understanding future cancer trends and developing appropriate cancer control strategies.[101] These modeling tools have not yet been applied specifically to alcohol and cancer or tobacco and alcohol co-use.

Alcohol and Cancer by Alcohol Product

Sufficient evidence demonstrates (1) the consumption of alcoholic beverages is causally related to several types of cancer and (2) ethanol in alcoholic beverages and its metabolic product, acetaldehyde, are carcinogenic.[1] Unlike tobacco products that can have different strengths of association with cancer and other diseases (e.g., cigarettes compared with smokeless tobacco), cancer risk does not vary by the type of alcoholic beverage consumed (e.g., wine, beer, or spirits), with the prime determinant of cancer risk being the amount and frequency of ethanol consumed.[1] Although some studies report differences in risk for different types of alcoholic beverages, these are most often attributed to chance or more commonly to confounding. For example, people who report largely or entirely wine consumption are likely to have different and healthier dietary intakes as well as reporting higher levels of physical activity than those who report largely beer or spirits consumption.[102-104]

Table 4.2 Findings From the 2010 International Agency for Research on Cancer Monograph, *Evaluation of Carcinogenic Risks to Humans: Alcohol Consumption and Ethyl Carbamate (Volume 96)*

Findings regarding alcohol consumption and cancer	
There is sufficient causal evidence of an association between alcohol consumption and risk for cancer of the—	• Oral cavity/pharynx • Larynx • Esophagus • Liver • Colon and rectum • Female breast
There is insufficient causal evidence of an association between alcohol consumption and risk for cancer of the—	• Stomach • Lung
There is evidence of no increase in risk between—	• Consumption of alcoholic beverages and renal-cell cancer – In several studies, increasing intake of alcoholic beverages was associated with a significantly lower risk for kidney cancer.
There is evidence of an inverse association or no association between—	• Consumption of alcoholic beverages and the risk for non-Hodgkin lymphoma – Most studies of non-Hodgkin lymphoma showed a lower risk for drinkers compared with nondrinkers.

Table 4.2 (continued)

There is sparse and/or inconsistent evidence for an association between consumption of alcoholic beverages and risk for cancer of the—	• Pancreas • Cervix • Endometrium • Ovary • Vulva and vagina • Male breast • Urinary bladder • Prostate • Testis • Brain • Thyroid • Skin melanoma • Hodgkin disease, leukemias • Multiple myeloma

Source: Adapted from IARC 2010.[1]

Reduction in Alcohol Consumption and Cancer Risk

In 2023, IARC convened a working group of 15 scientists from 8 countries to qualitatively evaluate the strength of published epidemiologic evidence on the potential for alcohol reduction or cessation to reduce alcohol-related cancer risk. They also evaluated mechanistic evidence on the potential effects of alcohol reduction or cessation to reduce alcohol-related carcinogenesis.[16] A summary of this IARC report by the authors[105] supports findings from previous IARC and WCRF/AICR reports suggesting that sufficient evidence shows that alcohol reduction or cessation reduces alcohol-related risk for cancers of the oral cavity and esophagus, while only limited evidence exists regarding the impact of alcohol reduction or cessation on reduction of risks for cancers of the larynx, colorectum, and female breast, and inadequate evidence exists regarding risks for cancers of the pharynx and liver. Differences in the conclusions about the effects of alcohol consumption reflect limitations in the available epidemiologic data by cancer site. Research on mechanisms linking alcohol and cancer support the idea that cessation or reductions in consumption should reduce risk of alcohol-related carcinogenesis across sites. However, for some sites, such as colon and rectum, there are few epidemiological studies, some of them small, and results are mixed.[105]

A 2024 study estimated that 20,216 cancer deaths in the United States in 2020–2021 were attributable to alcohol consumption, and 16,800 of these could have been prevented if no adults exceeded the 2020–2025 U.S. Dietary Guidelines recommended intake of no more than 1 (for women) or 2 (for men) drinks per day.[76] Worldwide, of 741,000 cancer cases attributable to alcohol, approximately 86% are associated with self-reported consumption of 20+ grams per day (~1.5 standard drinks).[75] Together, these studies support addressing alcohol as a target for cancer prevention and show that the greatest reductions in cancer mortality could be achieved by preventing consumption of larger amounts of alcohol.

Mechanisms of Carcinogenesis for Tobacco and Alcohol

Detailed review of the extensive literature on mechanisms of carcinogenesis of tobacco and alcohol can be found in chapter 5. Briefly, there are many mechanisms by which tobacco products cause cancer. These underlying mechanisms vary by the type of tobacco product used, with the strongest risks found for combusted tobacco products such as cigarettes, pipes, and cigars.[50,106] Tobacco smoke contains more than 7,000 compounds and at least 80 carcinogens.[107] These compounds cause cancer via diverse mechanisms including direct exposure to carcinogens; DNA damage; oxidative stress; inflammation; and changes in cell proliferation, immune response, and cellular metabolism.[50]

For alcohol, a primary mechanism for carcinogenesis, particularly for cancers of the aerodigestive tract, is the formation of acetaldehyde, a carcinogenic byproduct of alcohol metabolism.[1,87,108] Other mechanisms thought to contribute to carcinogenesis from alcohol include changes in DNA methylation, induction of the cytochrome P450 2E1, and increased inflammation and oxidative stress.[87,109] Additionally, alcohol consumption affects estrogen regulation, which is thought to play a role in the relationship between alcohol consumption and female breast cancer.[87]

As described in chapter 5, several potential mechanisms have been proposed for how tobacco and alcohol act synergistically to cause cancer, including that tobacco and alcohol expose users to some of the same carcinogens. Acetaldehyde, the most important of these overlapping exposures, is produced by the metabolism of ethanol in alcoholic beverages in the oral cavity and elsewhere in the digestive system by alcohol dehydrogenase (ADH) enzymes that are found endogenously and in the oral microbiome.[110,111] Acetaldehyde is also one of the major volatile organic compounds found in tobacco smoke, primarily produced by the combustion of polysaccharides.[112] Tobacco smoke also contains a wide variety of carcinogenic polycyclic aromatic hydrocarbons (PAHs), and some alcoholic beverages also contain PAHs,[113] potentially increasing exposure among people who both smoke tobacco and drink alcohol. Additionally, ethanol might promote cancer development by acting as a solvent for other carcinogenic agents, such as chemicals in tobacco and tobacco smoke.[87] Lastly, alcohol consumption can increase membrane permeability in the aerodigestive system, especially the oral cavity, pharynx, larynx, and esophagus increasing uptake of carcinogens from both the alcohol itself and from tobacco smoke.[114]

Studies of individuals with aldehyde dehydrogenase 2 (ALDH2) deficiency, also known as alcohol flushing syndrome, have provided important evidence concerning mechanisms of carcinogenesis related to alcohol (see chapter 5). The ALDH2 gene codes for a key enzyme involved in the detoxification of acetaldehyde [115] ALDH2 deficiency affects approximately 8% of the world's population and is very common in East Asian people, with an allele frequency of 35% to 45%.[116,117] Alcohol consumption by individuals with ALDH2 deficiency results in facial flushing, tachycardia, nausea, and headache, due to a rapid increase in circulating acetaldehyde concentrations.[115,116] ALDH2 deficiency is associated with increased risk of aerodigestive tract cancers, including cancers of the oral cavity, pharynx, larynx, and esophagus.[116-118] Among those who chronically drink alcohol, ALDH2 deficiency is one of the highest risk factors for cancer, with risk increasing with the extent of drinking.[116,119]
For example, a meta-analysis and Mendelian randomization approach of six case-control studies

by Boccia and colleagues (2009)[120] found that, compared with the ALDH2*1/*1 polymorphism (rs671), the ALDH2*1/*2 was associated with an increased risk of head and neck cancers among people who reported moderate and heavy drinking, but not among people who reported never drinking. The authors also noted that substantial increases in blood acetaldehyde levels were observed in people heterozygous for ALDH2 with administration of ethanol.[120] Further, a 2022 review indicated that risk for esophageal cancer was significantly higher among those with ALDH2 deficiency who consumed alcohol, with the risk increasing 50-fold among those who are ALDH2 deficient, drink alcohol, and also smoke cigarettes.[116]

The dramatic increase in risk observed for people with the ALDH2 deficiency who consume alcohol provides strong evidence for the causal role of acetaldehyde in head and neck cancers.[117,121] Furthermore, these physiological processes provide a clear mechanism for the synergistic effects of tobacco and alcohol on head and neck cancers.[122] Thus, established biological factors could account for both additive and multiplicative effects of tobacco and alcohol on cancer risk. Combined use of tobacco and alcohol in the context of ALDH2 deficiency could also influence behavioral factors related to risk, such as the probability of binge drinking or the specific temporal pattern of co-use.[117,121,123,124] More research is needed to enhance prevention of cancers of the aerodigestive system addressing behavior, exposure, and the biological factors relate to use and co-use of tobacco and alcohol.

Historically, the unpleasant effects of alcohol consumption have been a deterrent against heavy drinking among people with ALDH2 deficiency, resulting in protective effects against heavy alcohol use and resulting disease.[119] However, influenced by social, cultural, and economic factors, alcohol use has become more common among men in some populations that include large number of ALDH2 deficient individuals.[119] Absent intervention, increased rates of cancers and other alcohol-associated disease among people with ALDH2 deficiency who drink can be expected in the future. Relatively little research has been carried out in the United States addressing alcohol use among people with ALDH2 deficiency[125] or the combined effects of ALDH2 variants and tobacco use.[126] Efforts to improve surveillance of alcohol use in subgroups who are at increased risk, such as East Asian communities, could increase awareness and bring important public health benefits.[127]

Epidemiology of Synergistic Cancer Risk for Joint Tobacco and Alcohol Exposures

Comprehensive reviews by IARC and WCRF/AICR of the alcohol literature examine the question of whether cancer risk is synergistically affected by combined tobacco smoking and alcohol exposures.[1,2,4] These reports systematically explore combined effects and provide a comprehensive assessment of the literature. The WCRF/AICR report, based on epidemiological evidence, identified “amplified” risk for tobacco and alcohol exposures for cancers of the oral cavity, pharynx, larynx, and esophagus.[4] The 2010 and 2012 IARC reports, based on epidemiological, animal, and mechanistic evidence, reached similar conclusions and showed that the joint effects were multiplicative rather than additive for cancer of the esophagus, larynx, and oral cavity.[1,2] Other cancer sites, such as lung, produced less clear evidence of synergy. A summary of the cancer risk from tobacco smoking, alcohol use, and synergistic cancer risk is shown in Table 4.3.

Table 4.3 Summary of Literature on Independent and Synergistic Cancer Risk Related to Tobacco Smoking and Alcohol

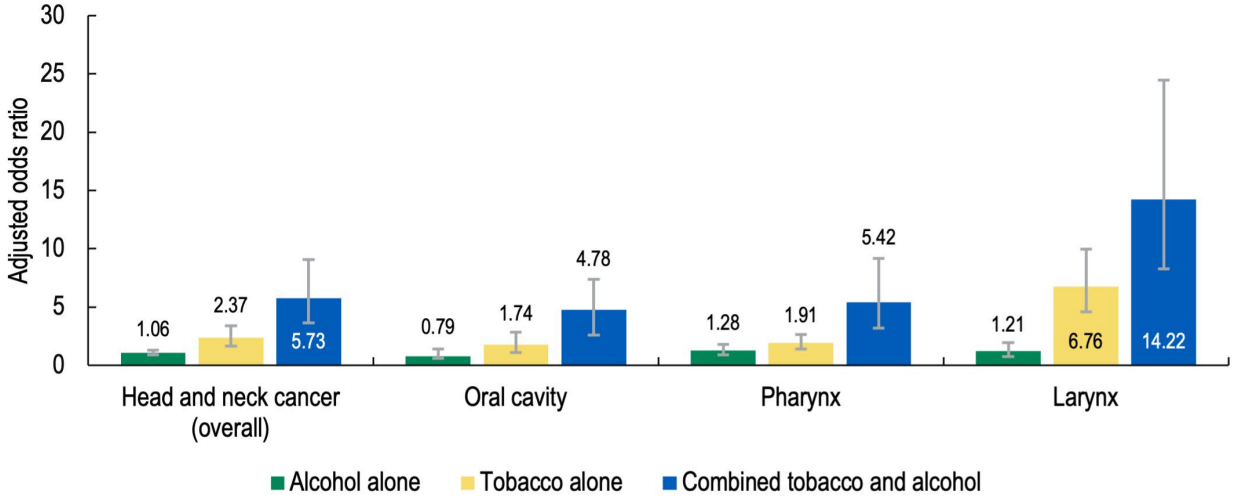
Disease site	Caused by tobacco	Caused by alcohol	Evidence of synergy
Aerodigestive tract	Yes[a,b]	Yes[c,d]	Yes[b,c,d]
Esophagus (squamous)	Yes[a,b]	Yes[c,d]	Yes[b,c,d]
Larynx	Yes[a,b]	Yes[c,d]	Yes[b,c,d]
Oral cavity and pharynx	Yes[a,b]	Yes[c,d]	Yes[b,c,d]
Colon and rectum	Yes[a,b]	Yes[c,d]	-
Liver	Yes[a,b]	Yes[c,d]	-
Stomach	Yes[a,b]	Yes[d]	-
Bladder	Yes[a,b]	-	-
Blood	Yes[a,b]	-	-
Acute myeloid leukemia	Yes[a,b]	-	-
Hodgkin lymphoma	-	-	-
Myeloma	-	-	-
Non-Hodgkin lymphoma	-	*	-
Cervix	Yes[a,b]	-	-
Kidney and renal pelvis	Yes[a,b]	*	-
Lungs, bronchus, trachea	Yes[a,b]	-	-
Pancreas	Yes[a,b]	-	-
Breast (female)	-	Yes[c,d]	-
Endometrium	-	-	-
Ovary	-	-	-
Prostate	-	-	-
Skin	-	-	-
Thyroid	-	*	-

Note. Cancers are presented in order of evidence of synergy (caused by tobacco and alcohol), caused by tobacco but not alcohol, caused by alcohol but not tobacco, or caused by neither; they are then listed alphabetically within those clusters. A dash (-) indicates that as of 2026, there is no conclusive evidence regarding a causal relationship with tobacco or alcohol use or synergy related to tobacco and alcohol use.
[a]USDHHS 2014,[3] [b]IARC 2012,[2] [c]IARC 2010,[1] [d]World Cancer Research Fund/American Institute for Cancer Research 2018.[4]
*Inverse associations with alcohol consumption are observed for kidney cancer, non-Hodgkin lymphoma, and thyroid cancer (see sections "Epidemiology of Alcohol Use and Cancer Risk" and "Thyroid Cancer" in this chapter).

Aerodigestive Tract Cancers

Based on the IARC and WCRF/AICR reports, a synergistic relationship between tobacco use and alcohol use and cancer risk on a multiplicative scale has been observed for squamous cell cancers of the oral cavity, larynx, pharynx, and esophagus.[1,4] A 2009 pooled analysis of 18 case-control studies, with 11,221 cases and 16,168 controls, reported greater than multiplicative joint effects of tobacco smoking and alcohol for head and neck cancers overall and separately for cancers of the oral cavity and pharynx.[5] Results were positive but not statistically significant for the larynx. These interactions are illustrated in Figure 4.1.

Figure 4.1 Risk of Cancer at Different Sites for Alcohol, Tobacco, and Combined Use



Note. Adjusted odds ratios are the results of random effects logistic regression models comparing ever use of tobacco and/or alcohol compared to never use of tobacco and/or alcohol, adjusted for age, sex, education, race/ethnicity, and study center.
Source: Hashibe et al. 2009.[5]

An important early study examining levels of tobacco smoking and drinking in a population with a wide range of consumption levels showed much more dramatic increases in risk of esophageal cancer for those who smoked and drank heavily, providing evidence of synergistic effects on cancer risk.[128] Castellsagué and colleagues (1999)[128] analyzed risk of esophageal cancer in 5 hospital-based, case-control studies of 830 cases and 1,779 controls in Argentina, Brazil, Paraguay, and Uruguay. The study was coordinated by IARC, and the 5 studies took place from 1986 to 1992. Compared with men who reported never smoking or drinking, men who reported using 1–7 cigarettes per day and 1–24 ml of ethanol per day had an adjusted odds ratio (aOR) of 4.15, whereas men who reported use of ≥25 cigarettes per day and ≥150 ml of ethanol had an aOR of 50.85 (95% CI, 22.80–113.39). The interaction between tobacco and alcohol use for esophageal cancer risk was not statistically significant, likely reflecting the modest sample sizes for the highest levels of exposure.

Most past research on interactions between tobacco and alcohol use and cancer risk has focused on combusted tobacco products, especially cigarettes. A systematic review and meta-analysis of 2 cohort studies and 31 case-control studies examined the risk of oral squamous cell carcinoma in people who used smoked tobacco, smokeless tobacco, and alcohol.[129] An elevated risk of oral squamous cell carcinoma was found for both co-use of smoked tobacco and alcohol (odds ratio [OR] = 4.74, 95% CI = 3.51–6.40) as well as co-use of smokeless tobacco and alcohol (OR = 7.78, 95% CI = 2.86–21.14). The highest relative risk of oral squamous cell carcinoma was observed for combined use of alcohol, smoked tobacco, and smokeless tobacco of (OR = 16.17, 95% CI = 7.97–32.79). Elevated risks for oral squamous cell carcinoma were also observed for use of each of the three products separately. Formal tests for interaction between products were not presented. Given the growing global market for smokeless tobacco products, further studies of interactions between smokeless tobacco and alcohol use are urgently needed.

Overall, these synergistic increases in risk observed in many studies of head and neck cancers strongly support efforts to address tobacco and alcohol co-use for cancer prevention.

A few studies have examined potential for differential cancer risk among those who consume tobacco and alcohol products by demographic factors. Three studies examined key differences for specific oral cancers by several factors, including sex, race, age, geographic region, and human papillomavirus (HPV) status (Table 4.4). In a narrative review of studies conducted in the Americas, Europe, and Asia, Pelucchi and colleagues (2006)[130] concluded that the evidence supported the presence of synergistic increases in risk of oral, laryngeal, and esophageal squamous cell cancer associated with co-use of tobacco and alcohol. For liver cancer they concluded that tobacco and alcohol use appeared to be independent risk factors.

Hashibe and colleagues (2009),[5] in a pooled analysis of case-control studies, evaluated differences between esophageal and head and neck cancers, respectively, by tobacco and alcohol use. They reported multiplicative joint effects for cancer risk among male and female participants who used both tobacco and alcohol (multiplicative interaction parameters for men: 2.36 [95% CI, 1.66–3.36] and women: 2.05 [95% CI, 1.35–3.11]) and in different age groups (multiplicative interaction parameters for <45 years = 2.93 [95% CI, 1.42–6.02], 45–60 years = 1.93 [95% CI, 1.40–2.66], >60 years = 2.19 [95% CI, 1.46–3.29]). Brown and colleagues (1994)[131] conducted a case-control study of 373 patients with squamous cell esophageal cancer (124 White men; 249 Black men) and 1,364 control subjects (750 White men; 614 Black men) to explore racial disparities in rates of squamous cell esophageal cancer. At every level of tobacco-alcohol co-use, the aOR for esophageal squamous cell carcinoma was higher among Black men than among White men. At the highest levels of exposure to tobacco and alcohol, the aORs for esophageal squamous cell carcinoma (adjusted for age, geographic area, and income) were 149.2 (95% CI, 39.2–367.4) among Black men, compared to 35.4 (95% CI, 10.0–125.5) among White men. The authors suggested that these differences in risk may be attributed to qualitative differences in alcohol consumption, differences in other environmental exposures, or genetic differences in susceptibility to these factors.

Examination of the development of head and neck squamous cell carcinoma and interactions between established risk factors, including tobacco smoking, alcohol drinking, and human papillomavirus (HPV) in a modest sized case-control study (N = 145 cases), suggested that the effects of tobacco, alcohol, and their interaction could be weaker in patients positive for high-risk HPV genotypes than in patients negative for high-risk HPV genotypes.[132] When stratified by high-risk HPV status, the effect estimates for tobacco smoking, alcohol drinking, and their additive and multiplicative interactions were consistently smaller and non-significant in the high-risk HPV-positive subsample, whereas patients positive for HPV but negative for high-risk HPV had significant, positive effect estimates for tobacco and alcohol effects and for the additive interaction. This may be related to the observation that HPV is a strong initiator of carcinogenesis in oropharyngeal squamous cell carcinoma via a specific molecular mechanism independent of alcohol or tobacco. However, alcohol consumption can increase the probability of risky sexual behavior, thus increasing the likelihood of HPV infection.[133-137]

Table 4.4 Evidence From Selected Studies of Differences in Synergistic Effect by Demographics

Cancer type	Study	Study design	Sample	Results	Key demographic differences
Squamous cell esophageal cancer	Brown et al. 1994[131]	Population-based, case-control study	U.S. adult male participants from 3 geographic areas; 124 White and 249 Black male cases; 750 White and 614 Black male controls	• aOR for esophageal squamous cell carcinoma was higher among Black men (aOR = 149.2, 95% CI = 39.2–567.4) and White men (aOR = 35.4, 95% CI = 10.0–125.5) who reported heavier tobacco and alcohol use compared with men of those racial groups who reported no or light amounts of tobacco and alcohol use.	Race
Head and neck	Hashibe et al. 2009[5]	Pooled analysis, case-control studies	International Head and Neck Cancer Epidemiology (INHANCE) consortium; Since 2004; 18 studies; 12,282 cases and 17,189 controls	• A greater than multiplicative joint effect between ever-tobacco and -alcohol use was observed for head and neck cancer risk. • Multiplicative joint effects for cancer risk estimated for men and for women (multiplicative interaction parameters of 2.36 [95% CI, 1.66–3.36] and 2.05 [95% CI, 1.35–3.11] respectively) and differences by age (multiplicative interaction parameters for <45 years = 2.93 [95% CI, 1.42–6.02], 45–60 years = 1.93 [95% CI, 1.40–2.66], >60 years = 2.19 [95% CI, 1.46–3.29])	Sex (male/female); age (<45/45–59/60+); geographic region

Table 4.4 (continued)

Cancer type	Study	Study design	Sample	Results	Key demographic differences
Head and neck squamous cell carcinoma (HNSCC)	Auguste et al. 2020[132]	Population-based case-control study	145 cases and 405 controls	• Current smoking (aOR = 11.6, 95% CI = 6.7–20.1), drinking >5 glasses of alcohol per day (aOR = 2.4, 95% CI = 1.2–4.7), and oral infection with high-risk HPV (aOR = 2.4, 95% CI = 1.1–5.0) were significantly associated with HNSCC. • The combined exposure to tobacco and alcohol produced a significant additive synergistic effect on the incidence of HNSCC (RERI = 9.8 [95% CI, 3.2–16.6]), but the multiplicative synergistic effect was not significant (multiplicative interaction parameter = 2.0 [95% CI, 0.8–5.4]). • The effects of tobacco, alcohol, and the combined exposure of tobacco and alcohol on HNSCC were consistently lower in high-risk HPV-positive than in high-risk HPV-negative individuals, though the three-way interaction of tobacco, alcohol, and high-risk HPV status was not explicitly tested.	HPV-positive versus HPV-negative HNSCC

Note. aOR = adjusted odds ratio; CI = confidence interval; HPV = human papillomavirus; RERI = relative excess risk due to interaction.

Synergistic Effects Between Tobacco and Alcohol Use for Other Cancer Sites

Convincing evidence supports the presence of synergistic interactions between tobacco and alcohol use and cancers of the aerodigestive system. Epidemiological evidence for these interactions is also supported by strong mechanistic evidence (see chapter 5). This section reviews evidence of a synergistic tobacco/alcohol exposure relationship with cancer sites not already identified by IARC or WCRF/AICR. Separate subsections address studies of cancers of the lung, liver and colon, female breast, thyroid and other sites. This section focuses on studies with more rigorous designs; however, a formal quality or risk of bias assessment was not conducted. Further details of the literature search are described in Appendix 4A, and selected studies are summarized in Appendix 4B. Overall, literature published since the 2010 and 2012 IARC reports and the 2018 WCRF/AICR report suggest additional potential examples of synergy between tobacco and alcohol consumption and cancer risk.

This section further describes evidence of differential synergistic relationships across population subgroups (e.g., sex, race, and ethnicity) for any cancer site, although few studies by population subgroup were identified. As noted previously, this chapter seeks to identify studies that assessed synergistic relationships by exposure to specific types of tobacco products (i.e., cigars, e-cigarettes, smokeless tobacco) or specific alcoholic beverages (i.e., wine, liquor, beer). However, few studies by tobacco product type or alcoholic beverage type were identified. The studies reviewed primarily address cigarettes and other combusted tobacco products. Research has also focused on the combined effects of smokeless tobacco products and alcohol use on head and neck cancer risk, because smokeless tobacco is associated with increased risk of these cancers.

Lung Cancer

Brenner and colleagues (2019)[138] published a pooled analysis examining the potential effect of alcohol consumption on lung cancer risk, using data from the International Lung Cancer Consortium and Synergy Consortium Studies. Their study included 21 case-control studies and 1 cohort study from North America (11), Europe (8), and Asia (3) with data on participants' alcohol intake. The interaction analysis between tobacco smoking and drinking, including 19,149 cases and 362,340 controls, suggested lung cancer risk was associated with higher levels of tobacco consumption (i.e., pack years), with an interaction observed for those in the highest tobacco and alcohol consumption groups. The effect estimate deviated from that expected on the additive scale among those who consumed ≥20 grams/day of alcohol and ≥10 pack years of tobacco (OR = 11.23, 95% CI = 10.29–12.26). The test for a multiplicative interaction between alcohol consumption and smoking intensity was also significant ($p < .001$). This study found an overall positive interaction between high pack years of smoking and elevated levels of drinking. However, the authors of the study noted:

> This may reflect some type of biological interaction of the combination of the two exposures, or it may also reflect a different exposure profile across drinking categories, where those at higher levels of both tobacco and alcohol consumption may be exposed to other factors, such as second-hand smoke from time spent in bars.

Furthermore, analyses from the Alberta's Tomorrow prospective cohort of 26,607 adults did not find evidence for an alcohol effect on lung cancer or interactions with tobacco use.[139] Further mechanistic research could help determine whether these epidemiological results are replicable and if they reflect a biological interaction related to carcinogenesis rather than unmeasured aspects of exposure.

Overall, there is inadequate evidence to support an association between alcohol consumption and lung cancer risk.[1] Ongoing epidemiological research has had mixed results,[140] with some studies suggesting an increased risk of lung cancer associated with drinking alcohol[141] and others reporting protective effects.[142]

Hepatocellular Carcinoma

Marrero and colleagues (2005)[143] examined the associations between cigarette smoking and alcohol use on hepatocellular carcinoma (HCC) in a case-control study of 70 patients with HCC, 70 patients with cirrhosis, and 70 patients without liver disease. Patients with cirrhosis and those without liver disease were included because cirrhosis is among the most important risk factors for development of HCC.[144] This study found that both tobacco and alcohol use were independent, dose-dependent risk factors for HCC.[143] When patients with hepatocellular carcinoma were compared to patients with cirrhosis, HCC risk increased nearly 6-fold for more than 1,500 grams of alcohol consumed per year (aOR = 5.7, 95% CI = 2.4–13.7), and nearly 5-fold for more than 20 pack years of smoking (aOR = 4.9, 95% CI = 2.2–10.6). Adjusted odds ratios for smoking and alcohol were far higher when patients with HCC were compared with patients without liver disease. The study also looked for an interaction effect between exposure to smoking and alcohol on HCC risk. When patients with HCC were compared to patients with cirrhosis, the risk for HCC was higher when patients were exposed to both cigarette smoking and alcohol (aOR = 7.2, 95% CI = 2.2–14.1), with a synergistic index >1, compared with exposure to cigarette smoking alone (aOR = 2.5, 95% CI = 1.7–15.0). or alcohol alone (aOR = 1.4, 95% CI = 1.1–4.0). The study concluded that alcohol and smoking are independent risk factors for HCC, and they also interact synergistically.[143]

A further study in 2000 reported an interaction between tobacco and alcohol consumption for liver cancer in a case-control study in Greece of 333 patients with cancer and 360 controls.[145] More recently, a 2024 case-control study in Korea of 15,352 patients with liver cancer and 92,112 matched controls reported a synergistic interaction between light-to-moderate alcohol consumption and heavy smoking with a significant synergy index (2.75, 95% CI = 1.85–3.66).[146] Despite its importance, few studies have addressed the combined effects of tobacco and alcohol use on liver cancer. These analyses are complicated by the fact that patients with a diagnosis of cirrhosis of the liver, a common precursor to liver cancer, are strongly advised to abstain from alcohol consumption,[147,148] highlighting the need for more detailed assessments of alcohol consumption over the life course, especially where circumstances are likely to lead to changes in consumption.

A 2025 study assessed the individual and interactive effects of cigarette smoking and alcohol consumption on HCC risk, using a nested case-control analysis of 108 HCC cases and 108 matched controls.[149] The analysis found a positive relationship between serum smoking biomarkers and HCC risk, serum alcohol biomarkers and HCC risk, smoking and HCC risk, and

alcohol use and HCC risk.[149] However, there was no evidence of synergistic effects between smoking and alcohol use on HCC risk, as interactions between self-reported smoking and alcohol use (synergy index = 1.27, 95% CI = 0.98–1.66), and interactions between smoking and alcohol biomarkers were not significant in predicting HCC (synergy index for 2-hydroxy-3-methylbutyric acid and cotinine = 1.59, 95% CI = 0.58–4.33; synergy index for 2-hydroxy-3-methylbutyric acid and nicotine = 1.36, 95% CI = 0.60–3.10).[149]

Female Breast Cancer

Alcohol use causes female breast cancer. IARC has concluded that the "occurrence of malignant tumors of the female breast is causally related to the consumption of alcoholic beverages,"[1] and the WCRF/AICR review concluded that alcohol consumption is a "probable cause" for premenopausal and a "convincing cause" for postmenopausal breast cancer.[4] Furthermore, alcohol alters some hormone levels that are associated with breast cancer risk, providing a plausible biological mechanism for the association (see chapter 5).[87,150,151] Risk of breast cancer related to alcohol use may be increased for estrogen receptor positive women. Some research on the association between alcohol consumption and breast cancer also suggests that the time between menarche to first pregnancy may represent a period when breast tissue is particularly susceptible to carcinogens.[150]

The potential for an association between active smoking, passive smoking (i.e., exposure to secondhand smoke), and female breast cancer has been addressed in several previous Surgeon General's reports.[3,152,153] The 2014 Surgeon General's report reviewed epidemiologic studies and studies of biological mechanisms to understand associations between exposure to tobacco smoke and female breast cancer. This report concluded that the evidence was suggestive but not sufficient to infer a causal relationship between tobacco smoke and female breast cancer or between active smoking and female breast cancer and between exposure to secondhand tobacco smoke and female breast cancer.[3] Similar conclusions were reached by a 2012 IARC review.[2]

However, some recent studies suggest a potential association. For example, a 2023 meta-analysis of 169 case-control or cohort studies of the relationship between cigarette smoking and female breast cancer reported a pooled RR of 1.07 (95% CI, 1.05–1.10) for women who currently smoked, compared with women who never smoked.[154] These authors also found that breast cancer risk increased with both the intensity of cigarette smoking (i.e., number of cigarettes smoked per day) and with increased duration of smoking.[154] The authors concluded, "this meta-analysis convincingly supports the contention that cigarette smoking is a risk factor for breast cancer."[154] However, concerns remain in the field regarding possible poorly measured and unmeasured confounding, the timing of exposure, and the possibility of etiologic differences across the major subtypes of breast cancer.[155] Even after decades of study, further large and robust studies are needed to resolve the relationship between tobacco use and female breast cancer.

Moderate and heavy alcohol consumption during the developmental window between menarche to first pregnancy could potentially increase the lifelong risk of breast cancer.[156,157] Such windows of susceptibility may also be present for smoking, as research examining the mechanism for smoking increasing female breast cancer risk suggests a potential increase in risk among women who initiated smoking before first childbirth.[158] However, this association was

only noted when the analyses did not adjust for age at first pregnancy and was attenuated by adjustment for pack years of smoking. The 2014 Surgeon General's report concluded that there was not clear evidence for a greater risk of female breast cancer associated with initiating smoking during adolescence or young adulthood than with initiating smoking at an older age.[3]

Several studies have reported significant multiplicative interactions for cigarette smoking and drinking alcohol in relation to female breast cancer risk,[139,155,159,160] but in other studies, apparent interactions were not significant or not tested directly.[158,161] Gaudet and colleagues (2017)[155] conducted a pooled analysis of 14 cohort studies that included 36,060 invasive female breast cancer cases and reported statistically significant positive interactions between alcohol consumption and several measures of smoking, including smoking status, cigarettes per day and years of smoking among those who currently smoked, and years of smoking and years since quitting in those who formerly smoked. The large sample size in this study is a notable strength. Zeinomar and colleagues (2019)[161] found a significant 3-way multiplicative interaction between familial risk profile, alcohol consumption, and cigarette smoking ($p = .005$), primarily driven by women with very high familial risk profile, who saw a 30% increase in overall breast cancer risk compared to women who never smoked. Similarly, Li and colleagues (2020)[160] reported a significant synergistic increase in breast cancer risk for carriers of the *BRCA2* mutation in a retrospective analysis among women who consume alcohol and who also smoke cigarettes (i.e., at least 1 glass per month for 1 year plus at least 1 pack of cigarettes per month for 1 year; aHR = 1.43, 95% CI = 1.13–1.81; $p = .003$). Additionally, Viner and colleagues (2019)[139] found a "U-shaped curve" for the association with breast cancer, observing a lower cancer incidence among those who reported moderate alcohol consumption and minimal cigarette smoking, changing to a multiplicative interaction with increased consumption of cigarettes and alcohol, compared to people who did not smoke or drink.

Other studies have not found a synergistic relationship between alcohol consumption, smoking, and female breast cancer risk.[162-164] As with other cancer sites, this may be due in part to differences in definitions of tobacco and alcohol use and measurement, as well as differences in specific outcome measures. Additional studies are needed to clarify the role of tobacco and alcohol in female breast cancer risk, with attention to the potential role of genetics and family history. Increases in alcohol-induced deaths among women in the United States emphasizes the importance of this area of research.[165]

Thyroid Cancer

The relationship between cigarette smoking, alcohol consumption, and thyroid cancer risk is unclear. A pooled analysis in the United States[166] and a large study in Korea[167] examined the individual and combined risk of these substances on thyroid cancer risk. The pooled analysis of 5 U.S.-based cohort studies by Kitahara and colleagues (2012)[166] included more than 740,000 participants and 1,000 incident cases of thyroid cancer and was conducted to examine the association between cigarette smoking, alcohol consumption, and the risk of thyroid cancer. This study found that current smoking was associated with a significantly reduced risk of thyroid cancer (adjusted hazard ratio [aHR] = 0.68, 95% CI = 0.55–0.85) compared with never smoking.

Furthermore, the risk of thyroid cancer decreased with greater smoking intensity and duration. Among people who formerly smoked, the risk was lowest among those who quit more recently (<5 years), but not as low as for people who currently smoked. The risk for people who quit smoking 5 or more years ago was similar to people who never smoked.[166]

Kitahara and colleagues also found that alcohol consumption was associated with a significant reduction in thyroid cancer risk, which was highest among those who consumed ≥7 drinks/week (aHR = 0.72, 95% CI = 0.58–0.90).[166] Additionally, compared to never smoking and nondrinking, current smoking and consumption of ≥1 drinks per week was associated with 36% reduced risk of thyroid cancer (joint aHR = 0.64, 95% CI = 0.47–0.86). The joint aHR for current smoking among participants who did not drink alcohol was weaker (aHR = 0.46, 95% CI = 0.29–0.73), while the joint aHR among participants who reported never smoking and reported ≥1 drink per week was similar (aHR = 0.70, 95% CI = 0.56–0.87). Overall, the reduced risk for current smoking was most pronounced among people who never drank, while the reduced risk for alcohol drinking was most pronounced for people who never smoked. The authors characterize the joint association of smoking and alcohol with thyroid cancer as submultiplicative (p for interaction = .004).[166]

Yeo and colleagues (2022)[167] conducted a retrospective cohort study using data from the Korean National Health Insurance database (N = 9,699,104 participants). After a mean follow-up period of more than 8 years, there were ~90,000 cases of thyroid cancer (0.9%) among participants. These authors identified an inverse association between cigarette smoking and the risk of thyroid cancer, as well as an inverse association between alcohol consumption and thyroid cancer. The risk of thyroid cancer was significantly lower among participants who currently smoked (aHR = 0.74, 95% CI = 0.72–0.76), compared with participants who never smoked, using a model controlling for multiple covariates, including alcohol consumption. Adjusted hazard ratios for thyroid cancer among those who never smoked were 0.91 (95% CI, 0.89–0.92) among those with mild drinking, 0.80 (95% CI, 0.76–0.84) among those with moderate drinking, and 0.80 (95% CI, 0.74–0.86) among those with heavy drinking. The lowest risk of thyroid cancer was observed in participants reporting the highest levels of both drinking and smoking; among people reporting current smoking and drinking >30 g of alcohol per day the adjusted hazard ratio was 0.62 (95% CI, 0.59–0.66). Analyses of the risk of thyroid cancer for combined use were statistically significant (p for interaction < .001).

The submultiplicative effects of cigarette and alcohol co-use observed by Kitahara and colleagues as well as Yeo and colleagues report could be related to lower thyroid hormone and thyroid stimulating hormone levels, as well as lower prevalence of thyroid autoantibodies. This further suggests that there may be related, or common, biological pathways involved in observed reductions in thyroid cancer risk related to tobacco and alcohol use separately. However, a 2023 study using data from the NIH-AARP Diet and Health Study reported that associations between thyroid cancer and cigarette smoking and alcohol drinking were restricted to thyroid cancers at low risk for recurrence or mortality ("low-risk" thyroid cancers); no associations were observed for high-risk thyroid cancers.[168] Thus, these inverse associations may be due in whole or part to

detection bias (i.e., individuals who currently smoke or drink more heavily are less likely to undergo thyroid cancer surveillance or screening).[168] Furthermore, Kitahara and colleagues report a lower risk of thyroid cancer for people who report cigarette smoking but no alcohol use than for people who currently smoke and report one or more drinks per day.[166] This is consistent with the possibility of common biological pathways for tobacco and alcohol but their effects on such pathways may differ.

Cohort Studies of Cancer at Multiple Sites

A prospective cohort analysis by Pirie and colleagues (2013)[169] examined combined effects of alcohol and cigarette smoking on mortality in the Million Women Study cohort of 1.3 million women in the United Kingdom recruited between 1996 and 2001 and followed until January 1, 2011. The study authors conducted sensitivity analyses among women who did not drink or who consumed fewer than 3 units of alcohol (30 ml of ethanol or 1.67 U.S. standard drinks) per week. Compared to risk ratios for smoking-related mortality among the entire sample, sensitivity analyses found modest reductions in adjusted risk ratios from 4.38 in the entire sample to 3.76 (95% CI, 2.65–5.31) in the sensitivity analysis for cancer of the mouth, pharynx, larynx, nasal cavity, or sinuses; from 3.10 in the entire sample to 2.74 (95% CI, 2.27–3.30) in the sensitivity analysis for esophagus cancer; from 1.52 in the entire sample to 1.43 (95% CI, 1.11–1.85) in the sensitivity analysis for liver cancer; and from 1.13 in the entire sample to 1.06 (95% CI, 0.95–1.18) in the sensitivity analysis for breast cancer.[169]

These results do not examine interactions between tobacco and alcohol use and cancer risk directly, highlighting the need for formal tests of interaction. Viner and colleagues (2019)[139] examined individual and combined effects of cigarette smoking and alcohol use on cancer risk among participants (N = 26,607) in Alberta's Tomorrow Project. Results suggested additive effects of tobacco and alcohol on risk of all cancers, colon, and prostate cancer.

All together, these studies highlight the need for further pooled analyses of epidemiological studies addressing the combined effects of tobacco and alcohol on cancer risk at multiple sites along with further mechanistic research. Additionally, efforts are needed to examine age specific patterns of tobacco and alcohol use and co-use, particularly in relation to female breast and liver cancer where developmental windows of susceptibility or behavior change related to precancerous effects may be relevant.

Measurement and Analysis

This section discusses considerations for measuring exposure to tobacco and alcohol, including biomarkers of tobacco and alcohol use. The results of the epidemiological studies reviewed in this chapter and in chapters 2 and 3 emphasize the importance of measurement. Careful consideration of intended analyses, sources of bias, and fitness for purpose are needed for measure selection.[170] As discussed in chapter 2, surveillance or surveillance epidemiology focuses on the ongoing collection of data for planning, implementation, and evaluation of public health programs and action.[171] In contrast, measurement of behavior for etiological studies focuses on estimating potential causal relationships between aspects of exposure and outcomes as well as controlling for potential confounding in the assessment of such relationships.[172] Thus, longitudinal cohort studies could well include measures of tobacco and alcohol use

patterns, including those at different stages of the life course and correlates of tobacco and alcohol use, that may not be of immediate interest to surveillance epidemiology unless or until they are demonstrated to be critical determinants of future disease burden.

The amount, frequency, and duration of tobacco and alcohol use, as well as cessation of use, can affect cancer risk; for this reason, epidemiologic analyses should consider frequency of use, intensity of use, frequency of heavy use days, and variability of use over the life course. The most commonly used measures of alcohol consumption include quantity/frequency (QF) and graduated frequency (GF) approaches. The QF approach addresses the number of days drinking during some specified period (e.g., past year) and the average number of drinks on such days. The GF approach specifies categories of standard drinks consumed (e.g., 1–2 drinks) and facilitates comparisons across groups at the expense of some detail.[173] Table 4.5 lists examples of tobacco and alcohol use measures that are relevant or potentially relevant to assessing cancer risk.

Table 4.5 Selected Tobacco and Alcohol Use Measures Useful for Epidemiological Studies

Measure type	Tobacco	Alcohol
Current use	• Current use (not at all, some days, or every day) or use on at least 1 day in the past 30 days	• Past-month use or use in the last 30 days • Past-year use
Use frequency and intensity, including QF or GF approaches	• Cigarettes per day or other tobacco and nicotine products per day • Daily/nondaily use (cigarettes or other tobacco products) • Days per month used (cigarettes or other tobacco products) • Number of days of [tobacco product] use in the past 30 days • Number of cigarettes per day or other products on days when the respondent smokes • Ever daily smoking among those who smoke nondaily	• Frequency of alcohol consumption • Quantity of standard drinks[a,b] per occasion • Volume consumed in the past month and past year (overall and by beverage type) • Frequency heavy episodic drinking or binge drinking (≥4 drinks for women or ≥5 drinks for men on an occasion) • Frequency of high-intensity drinking: 8+,10+, 12+, or 15+ drinks per day or occasion • Maximum drinks consumed in 1 day in the past month and past year

Table 4.5 (continued)

Use history and lifetime use patterns	• Ever use (having ever smoked even a puff of a cigarette and having ever smoked 100 or more cigarettes in one's lifetime) • Age of initiation • Cigarettes per day during periods of regular use • Number of quit attempts resulting in abstinence for at least 24 hours ("Serious" quit attempts) • Age at longest successful quit attempt • Duration of smoking abstinence for longest quit attempt • Pack years (based on years smoked and average number of cigarettes per day) • Use of other tobacco products, including duration, frequency, and intensity • Time to first cigarette or first tobacco use	• Lifetime abstinence (cumulative periods of abstinence in one's lifetime) • Age of initiation • Age at which began regular drinking (for example, at least once a month for 6 months) • Former drinking • Age of cessation • Lifetime accumulated number of drinks • Frequency of lifetime heavy episodic drinking or binge drinking and during specific time periods/decades • If quit, reason for quitting

Note. GF = graduated frequency; QF = quantity/frequency.
[a]One U.S. standard drink contains 0.6 fluid ounces or 14 grams of ethanol. A standard drink is equivalent to 12 fluid ounces of beer at 5% alcohol by volume, 5 fluid ounces of wine at 12% alcohol by volume, and 1.5 fluid ounces of distilled spirits at 40% alcohol by volume.
[b]Moderate drinking is defined as up to 2 standard drinks/day for men and up to 1 standard drink/day for women.[174]

Measurement of Frequency of Use, Intensity of Use, and Simultaneous Use

Research as of 2026 indicates a need for further efforts related to several aspects of tobacco and alcohol use including measures of drinking intensity, especially high intensity drinking, and lifetime patterns of tobacco and alcohol co-use, along with more detailed data on the characteristics and temporality of co-use. For example, near simultaneous co-use might have different exposure consequences than co-use on different occasions during the same day, week, or month, as well as high intensity drinking during different developmental stages of the life course (e.g., adolescence, pre- or post-menopause).

Measures of high intensity drinking days/occasions, intended to capture higher levels of impairment relevant to acute consequences of drinking, may also be relevant to cancer and other chronic disease risks. For example, a meta-analysis of case-control studies found that alcohol use intensity (drinks per day) was a greater risk factor for head and neck cancers than duration of alcohol use,[79] emphasizing the importance of measuring heavy episodic drinking, high intensity drinking, and maximum drinks in a day for cancer risk assessment. Many standardized surveys and other health studies do not consider changes in the number of cigarettes per day over time. However, diary and ecological momentary assessment studies suggest the number of cigarettes smoked per day varies from day to day.[175] Longitudinal studies also suggest that at least some individuals change their smoking patterns and the amount that they smoke over time, which may affect cancer risk.[175-177] A growing literature on patterns of smoking examines those who smoke intermittently and fewer cigarettes, different cessation characteristics, and other aspects of smoking trajectories.[178-183] For example, in a study of 24,613 men ages 50–69 who smoked and participated in a alpha-tocopherol and beta-carotene cancer prevention trial, reducing smoking intensity at an average 5 cigarettes per day per intervention year reduced lung cancer risk compared with smoking 20 cigarettes a day, but cessation had the largest reduction in risk.[184] More efforts are also needed to incorporate such patterns into etiological analyses and modeling

of population-level tobacco use and its consequences. For alcohol, a 2021 Australian prospective cohort study (*N* = 226,162) found a marginally increased risk of female breast cancer when alcohol intake was concentrated over time (i.e., the same amount of alcohol was consumed in 1–3 days versus 4–7 days).[185]

Measures of simultaneous use of tobacco and alcohol could also be helpful for understanding cancer risk, particularly for upper aerodigestive tract cancers, for which a synergistic effect of these two substances is well documented. Broadly speaking, simultaneous use is defined as use at the same time; during simultaneous use, the effects of both substances could overlap and interact in important ways. However, the time periods involved, and the specific behaviors associated with "simultaneity" have not received much attention. Simultaneous use questions have rarely been included in epidemiologic studies, perhaps because researchers assume that alcohol drinking and tobacco smoking typically occur at the same time or in close time proximity. A study of 217 college students found that many people who smoke nondaily only smoke when drinking and that smoking is more appealing to them when drinking with greater re-enforcement of smoking and less attention to negative consequences.[186] One study of people who smoke nondaily (*N* = 183), grouped by those who reported long-term occasional smoking, early occasional smoking, and former daily smoking, found that these groups did not differ in alcohol co-use patterns.[187] Less than one-third of smoking sessions involved drinking, and 42% of drinking sessions involved smoking, with greater use amounts of both during co-use sessions. This indicates that attention is needed to co-use occurring on different time scales from simultaneous use to co-use over longer time scales such as in the same day, week, month, or year.

Measurement of Lifetime Exposure and Use Patterns

Life course measures of tobacco and alcohol co-use are also important for understanding cancer risk. Lifetime measures of tobacco use include the age of initiation, use frequency, use quantity per day/occasion in relevant periods of life (e.g., age decades or period of highest consumption), and aspects of cessation (e.g., age at cessation or longest successful cessation). These lifetime measures can be used to create estimates of accumulated use, such as pack years of cigarette smoking, calculated by multiplying the typical number of cigarette packs smoked in a day by the number of years the person has smoked. Identification of never drinking is also important for determining appropriate control groups. However, self-report of lifetime abstention from alcohol is not perfect and misclassification based on self-report can be significant.[188,189] Additionally, people who report lifetime abstention from alcohol are known to have specific demographic and health related characteristics, which can result in bias if lifetime abstainers are used a reference group.[190]

Lifetime measures of accumulated alcohol intake are available in some prospective cohorts[191]; however, such data have not been used very widely in analyses of associations between tobacco and alcohol co-use and cancer risk. Some of these studies involve repeated measures over time, potentially improving validity, but others collect alcohol consumption in prior decades retrospectively.[192] The latter format could result in recall bias and greater measurement error for more distally retrospective decades. More research is needed on the validity and reliability of such recalls and how to improve them, including by using electronic health record systems to document use.

Often, current drinking during the month or year of assessment is used to classify long-term drinking patterns, under the assumption that adult drinking patterns are stable over time.[193,194] The past-year timeframe is typically preferred for measuring current alcohol use in prospective studies of alcohol-related outcomes, as this timeframe is thought to describe average drinking over seasons and other short-term variations and repeated assessments may not be feasible. Studies have shown that changing patterns commonly occur seasonally[195] and on a year-to-year basis,[194] with aging,[196-198] due to life events,[199] and as a result of alcohol policy changes.[200] More longitudinal studies with repeated assessment of alcohol use are needed.

By contrast, smoking behavior has been found to be relatively stable over time among those who smoke daily, but the literature regarding those who smoke less than daily is mixed regarding stability of smoking behavior.[201,202] People who report nondaily smoking are an important group as they have the highest prevalence of alcohol use, including high-risk drinking behavior, compared with people who do not smoke (see chapters 2 and 3). In a study of 579 California young adults who smoked nondaily, alcohol use was associated with lower odds of abstinence.[203] Moreover, it is important to note that adolescence to young adulthood is a period of experimentation and escalation of tobacco use, so the patterns of use can be unstable.[203-206] These patterns may also be changing as the types of tobacco products available come into or out of the marketplace and with changes in regulation of where smoking is permitted and/or acceptable. Importantly, few studies have included lifetime measures for both tobacco and alcohol.

Measurement of Product Characteristics

Even more granular measures of tobacco and alcohol co-use may also be relevant to the influence of co-use on carcinogenesis. As discussed in chapter 5, all types of alcohol beverages contain ethanol, the major factor related to alcohol-related carcinogenesis, and the amount of ethanol consumed largely determines cancer risk. As discussed previously in this chapter, ABV can vary widely across alcohol products.[207,208] Measures that identify the volume of beer, wine, and spirits consumed, as well as subtypes of higher percentage ABV beverages—such as malt liquor, strong beer, fortified wine, strong cocktails, shots, and straight spirits—could be informative. Under-reporting of total alcohol consumption could be greater for those at the highest levels of consumption of spirits; observational studies are more difficult because many people with higher levels of consumption consume several types of alcoholic beverages.

Despite the key role of ethanol in carcinogenesis, considerable research has examined the type of alcoholic beverage consumed and cancer risk, particularly for certain cancers. For example, the combined effects of tobacco and alcohol on risk could be sensitive to the type of alcohol consumed and to the precise timing of use, as these may influence salivary acetaldehyde levels and the oral microbiome.[108] Similarly, a study of esophageal cancer and alcohol consumption from France suggests additional risk related to consumption of apple brandy.[209] However, all alcohol products cause increased risk of cancer and there does not appear to be a consistent difference in cancer risk based on the type of alcoholic beverage consumed (see also chapter 5).[210-213]

Methods and Measurement of Tobacco and Alcohol Consumption Across the Life Course

Epidemiological studies as well as health surveillance systems are deeply challenged by the need to consider respondent burden. This is a particular problem for studies that assess complex behaviors such as drinking and smoking. Greater efforts could be made to improve survey instruments for collecting lifetime consumption data, such as the use of graphical interfaces for online or smartphone-enabled data collection. Additionally, higher compensation for study participants or greater use of targeted cohorts focusing on specific exposures could reduce respondent burden by addressing only a few exposures in detail and including assessment of only the most critical covariates.

Study designs using detailed daily diaries that include timing of use, such as ecological momentary assessment studies which provide for real time assessment of the behaviors, could be used to measure simultaneous use. In addition, surveys can include follow-up questions on the use frequency of one substance (e.g., alcohol) about how often that substance was used together with the other substance (e.g., cigarettes) that the respondent had previously reported currently using. These follow-up questions have become more common in studies of alcohol and cannabis use and could be adapted for tobacco and alcohol.[214]

Despite considerable efforts to develop reliable and valid measures of lifetime tobacco and alcohol use, there are inherent limitations to self-reported behavior, particularly if the behaviors occur less regularly, change over time, or must be recalled from the more distant past. All these factors are relevant to both tobacco and alcohol use, especially as intermittent and multiple tobacco product use has become more common, and levels of tobacco and alcohol use can change over time.

There have also been substantial efforts to identify biomarkers of longer-term exposure, including biochemical adducts,[215] DNA methylation,[216-218] mosaic Y-chromosome loss,[219] and somatic mutation signatures.[220] These integrative biomarkers reflect the molecular and physiological effects of exposure and thus can be considered to be both biomarkers of acute and long-term exposure and also intermediate markers of carcinogenesis and other disease processes. Such biomarkers are agnostic in that they do not rely on self-report and have been shown to be useful for assessing both current and former tobacco use. However, whether biomarkers can be widely deployed to overcome the limitations of self-reported assessment remains to be determined and is an active research area.

Biomarkers of Exposure to Tobacco

Cotinine, a by-product of nicotine metabolism which can be measured in blood, urine, or saliva, is the most commonly used biomarker of tobacco use. The half-life of cotinine averages 16 hours and thresholds have been established to detect active smoking (3–10 ng/ml for plasma) as well as for secondhand smoke exposure.[221,222] Another commonly used biomarker of short-term exposure to tobacco smoke is the concentration of carbon monoxide (CO) in exhaled breath (or carboxyhemoglobin in blood). The half-life of CO is influenced by various factors but is typically in the range of about 4 hours. A cutpoint of 5–6 ppm in exhaled CO is typically recommended to distinguish a person who smokes from someone who is abstinent from smoking.[221] However, there is a limitation of using exhaled CO as a biomarker—it is not specific to tobacco smoking and may reflect exposure to other combustion products, such as cannabis

smoke, and common air pollutants, such as auto exhaust. Nicotine, a highly specific biomarker of tobacco use, can be measured directly in blood plasma; because of its short half-life (~2 hours) it is useful only for assessing short-term exposure.[221]

The tobacco-specific biomarker, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), a primary metabolite of the tobacco-specific nitrosamine NNK, is a biomarker of potential harm from tobacco. NNAL remains in the urine far longer than cotinine (NNAL half-life: 26–45 days; cotinine half-life: 16–18 hours) and is indicative of tobacco exposure from cigarettes, other tobacco products (e.g., smokeless tobacco, heated tobacco products) and secondhand smoke exposure.[223-225] Other biomarkers of potential harm that assess exposure to specific carcinogens or carcinogenic processes, such as inflammation or oxidative stress, are also widely used in environmental epidemiology, tobacco regulatory science, and other fields.[224,226] However, unlike NNAL, these biomarkers are not specific for tobacco. The minor tobacco alkaloids anabasine and anatabine are derived from tobacco leaf and can be used to determine exposure to products containing tobacco (e.g., cigarettes, cigars, smokeless tobacco, and others) in people using nicotine replacement therapy or using nicotine-containing ENDS to distinguish between exposure to products that do and do not contain tobacco.[221]

Biomarkers of Exposure to Alcohol

Biomarkers currently in clinical and research use are sensitive to different levels of alcohol use and use over different time windows, ranging from the presence of ethanol itself in blood via blood alcohol concentration tests or exhalations to various biomarkers related to alcohol, such as phosphatidyl ethanol (PEth) and fatty acid ethyl ester (FAEE).[227] FAEE is not correlated with level of use but can detect alcohol use within the past 24–99 hours. PEth is sensitive to consumption of about 50 grams ethanol per day up to 3 weeks after consumption ceases. Such tests have been used in randomized controlled trials[228] and could potentially be useful in a clinical setting or to monitor adherence to cessation regimes. Shorter term measures of consumption may also be useful to validate self-reports. For example, a 2025 nested case-cohort study of Spanish men ages 55–80 and women ages 60–80 in the PREDIMED trial used urinary tartaric acid to validate self-report of wine consumption from food frequency questionnaires.[229] However, more research is needed to identify more effective biomarkers.

For epidemiological studies, long term biomarkers of total alcohol consumption that are sensitive to lower levels of consumption are also needed to reduce measurement error related to self-report and refine estimates of risk at lower levels of alcohol consumption. Measures of liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are elevated with heavy drinking, but the degree of elevation is dependent on other aspects of the liver's response to alcohol and is reduced with reduced drinking.[227,230] Biomarkers of long-term alcohol use might be found in genetic or epigenetic changes associated with exposure to alcohol and its metabolites, although more research is needed in this area.[231] The majority of research on biomarkers of alcohol use has involved short term markers such as metabolites of alcohol and alcohol related enzyme levels,[232,233] and at the group level, several of these markers can distinguish between lower and higher levels of intake. However, none of these can distinguish between people who do not drink and sporadic or low daily intake consumers and none are able

to capture total consumption over multiple years. More research is needed to identify and validate markers of lifetime alcohol consumption, especially markers that are sensitive to lower levels of consumption. Such studies could be carried out in prospective cohort studies that included multiple self-report measures of alcohol consumption over time and recently collected blood or other biological samples.

Mendelian Randomized Studies of Alcohol and Tobacco

Genetic studies have also been used to evaluate whether tobacco or alcohol consumption are causally related to cancer risk at various sites.[234-240] Such Mendelian randomization studies leverage genetic variants as tools in an attempt to causally evaluate whether exposures, such as tobacco and alcohol use, are independently associated with disease outcomes. In traditional observational epidemiologic studies, reverse causation, bias, and confounding are of substantial concern. In contrast, Mendelian randomization studies leverage Mendel's laws of inheritance, namely that genetic alleles are randomly assorted at conception, which is analogous to the randomization process of a clinical trial.[241] In Mendelian randomization studies, researchers aim to create genetic instrumental variables are created by identifying genetic variants that are specifically associated with exposures of interest[242] (e.g., tobacco use and alcohol intake). Associations of these instrumental variables and disease endpoints of interest are then evaluated. Mendelian approaches could be informative for clarifying unresolved associations, such as whether alcohol intake increases risk of lung cancer or whether smoking increases the risk of breast cancer. However, Mendelian randomization studies themselves require a number of assumptions, not all of which can be directly testable, and thus can also be affected by bias. To date, Mendelian randomization studies of alcohol consumption and lung cancer[234-238] and smoking and breast cancer have been inconclusive,[243,244] perhaps reflecting limited statistical power. Future research in this area is needed.

Measurement Needs Related to Health Disparities

High levels of tobacco and alcohol use as well as disparities in cancers related to tobacco and alcohol have been observed in some demographic groups such as American Indian or Alaska Native individuals and sexual minorities.[53,245-248] However, great care must be taken not to interpret these studies as suggesting that all or most of the people in a demographic group have high levels of consumption (see chapter 3). These observations also highlight the importance of valid and reliable measurement of tobacco and alcohol use across diverse populations. Language, values, and social factors may influence self-report of tobacco and alcohol use behaviors.[249,250] For example, in a cross-sectional study of 2,520 individuals analyzing data from the 1999–2002 National Health and Nutrition Examination Surveys, Everhart and colleagues (2009)[249] reported that misclassification of tobacco use status was associated with both sex and acculturation status in Hispanic adults in the United States with greater levels of misclassification for men with lower levels of acculturation (almost 5%) compared to women with lower levels of acculturation (~1%). For alcohol use, prior use of alcohol and other substances was strongly related to disagreement between retrospective and prospective reporting of alcohol onset measures in a sample of 1,044 adolescents in Chicago.[251] Black and Hispanic military veterans are more likely to be diagnosed with alcohol use disorder compared to White veterans, even at the same reported levels of alcohol consumption[252]; such misclassification could bias epidemiological analyses relying on medical records. In addition, willingness to respond to surveys, ability to recall past

substance use (e.g., among older adults), and survey-specific factors leading to measurement error may differ by population group. More research is needed to identify and avoid differential misclassification of tobacco and alcohol use for epidemiological studies.

Worldwide, a 2010 report by the World Health Organization (WHO), *Equity, Social Determinants, and Public Health Programmes,*[253] found that certain groups, including those of lower SES, had a higher prevalence of the use of tobacco products and experienced a higher burden of disease from that use. The report also found that, when looking at a macro-level at the relationship between alcohol and disease that, although populations with higher SES had higher percentages of people who drink, those with lower SES had a higher percentage of people who report heavy episodic drinking.[253] Together these observations highlight the need for valid and reliable measurement of both consumption and health outcomes in different demographic groups.

Alcohol Harms and Socioeconomic Status

Several studies have examined the harm of alcohol in relation to SES. For example, Smith and colleagues (2024)[254] examined associations of education and alcohol use with alcohol attributable hospitalization or death in a large (N = 199,125) cohort study in Canada. Levels of heavy alcohol consumption and alcohol volume were similar for people with lower and higher levels of education—but levels of hospitalization or death were disproportionately higher for people with lower SES and higher levels of consumption. This observation that people with lower SES exhibit greater harms from alcohol than those at higher SES with the same consumption levels has been called the alcohol harm paradox, and a growing number of studies have reported this pattern for several measures of SES and various health outcomes related to alcohol.[255] For alcohol and cancer, very few studies have examined interactions between SES and alcohol; more analyses are needed.[256]

The synergistic effects of tobacco and alcohol in subpopulations were not addressed in the 2010 WHO report or in most studies of alcohol harms in relation to SES. These findings confirm the importance and need to accurately measure precise amounts of tobacco and alcohol consumed, as well as types of tobacco and alcohol products consumed, frequency, timing of consumption, and duration at a more granular level and over time to fully understand patterns of use of each product and to better understand how they relate to disease risk and how they interact with SES and its correlates.

Chapter Summary

Cigarette smoking has been causally linked to the development of cancer at multiple sites, including the oral cavity, pharynx, larynx, esophagus, lung (trachea and bronchus), stomach, liver, pancreas, kidney and ureter, bladder, cervix, colorectum, as well as acute myeloid leukemia. Currently, evidence also suggests an association between cigarette smoking and female breast cancer, however, consensus on this relationship has not been reached. The carcinogenicity of combusted tobacco products (e.g., cigarettes, cigars, and pipes) is attributed to the presence of more than 7,000 compounds, including more than 80 carcinogens, in tobacco smoke. The prevalence of cigarette smoking has been decreasing for decades; in 2024, 9.9% of

U.S. adults reported current use of cigarettes. However, youth and adult tobacco use now reflects the diversity of the tobacco product marketplace and polytobacco use has become increasingly common, especially among youth.

Alcohol consumption is the third most important modifiable risk factor for cancer in the United States. Conclusive evidence shows that alcohol consumption is causally linked to an increased risk of cancers of the oral cavity, pharynx, larynx, esophagus, colorectum, and liver, as well as female breast cancer; studies of a potential causal role for alcohol for other cancer sites is sparse or inconsistent. Alcohol consumption is also associated with a lower risk of kidney cancer and non-Hodgkin lymphoma. However, the potential benefit of this lower risk is vastly outweighed by alcohol's causal role in other cancers and other serious diseases. Ethanol, contained in all alcohol products, is the leading determinant of their cancer risk, although some differences in risk have been reported by specific alcohol products. The risk of alcohol-related cancers is substantially increased in people with aldehyde dehydrogenase deficiency, a genetic condition common in some populations, including people of Asian ancestry. As described in more detail in chapters 2 and 3, alcohol consumption differs significantly between men and women, and by other demographic factors such as race, age, and socioeconomic status.

Numerous studies have examined the potential for synergy between tobacco and alcohol and cancer risk (defined here as when the total effect is greater than the sum of the individual effect). Conclusive evidence for synergy between cigarette smoking and alcohol use has been shown for cancers of the aerodigestive tract (i.e., cancers of the oral cavity, pharynx, larynx, and esophagus). Few studies have addressed the potential for synergy between other tobacco products (e.g., smokeless tobacco, e-cigarettes) and alcohol use on cancer risk. Similarly, few studies have examined the potential for synergy between tobacco and alcohol use by demographic factors (age, sex, race, and/or ethnicity) and relevant health factors. For example, studies have shown that infection with human papilloma virus (HPV) weakens the synergy between tobacco and alcohol use and risk of oropharyngeal cancer. In addition, studies have examined the potential for synergy between tobacco and alcohol use for other cancer sites. For example, some studies suggest a possible synergistic interaction between tobacco and alcohol for cancer of the lung, liver, and female breast. Measurement is a critical component of studies to address the epidemiology of tobacco- and alcohol-associated cancer risk. Widely used measures of tobacco and alcohol use typically address frequency, intensity, duration, and cessation of use. Additional areas of special interest to studies of cancer risk include measuring simultaneous use of tobacco and alcohol, understanding lifetime substance consumption, and assessing how patterns of use may change over the lifespan. Given the wide range of alcoholic beverages and their variable alcohol content, measuring the volume of alcohol consumed is also important. Additionally, as the tobacco product marketplace continues to diversify, measures will be needed to assess the broad range of tobacco products.

Most surveys and epidemiologic studies rely on self-reported measures. However, self-reported measures have well known challenges, including the potential for research participants to be unwilling or unable to accurately report tobacco and/or alcohol use, which are stigmatized behaviors. Misclassification is a particular concern for case-control studies where people

diagnosed with cancer may recall their use more or less accurately than participants without cancer, but misclassification can also affect prospective cohort studies. Several biomarkers of tobacco and alcohol use are available to supplement or replace self-report; each of these biomarkers has limitations, and the field would benefit from additional tools to assess use. Finally, measures need to be valid and reliable across diverse populations, given the need to understand and address disparities in tobacco and alcohol use and cancers caused by tobacco and alcohol use.

Research Needs

Research needs concerning the relationship between tobacco and alcohol co-use and cancer risk involve a combination of measurement and analytical issues. For etiological studies of alcohol alone and tobacco and alcohol co-use, alcohol use is most commonly measured using quantity/frequency and graduated frequency approaches to estimate volume of alcohol intake. Fewer studies have examined patterns and changing patterns of alcohol intake from initiation throughout the life course, which is particularly important for cancers that may have developmental windows of susceptibility, such as female breast cancer.[157] Measures of tobacco and alcohol use over the life course increase respondent burden, a key cause of loss to follow-up or nonresponse in cohort studies[257] as well as noncompletion of health surveys.[258,259] For this reason, work is needed to further develop and refine a package of measures encompassing patterns of use and nonuse that minimize respondent burden and address both tobacco and alcohol. A few studies suggest that instruments such as the Lifetime Drink History Survey can capture data concerning alcohol use initiation and subsequent patterns of drinking over the life course.[260-262] However, more work is needed to refine such instruments and to understand their potential for bias and measurement error associated with different patterns of lifetime behavior as well as detailed aspects of co-use, such as simultaneous smoking and drinking.

Continued efforts to combine results from different cohorts and to harmonize estimates of tobacco and alcohol use for metanalytic or pooled analyses of associations between co-use and cancer risk are needed. Past analyses have often addressed tobacco and alcohol co-use and aerodigestive tract cancers because of the larger relative risks and population-attributable fractions for these cancers associated independently and synergistically with these exposures. Where relative risks and population-attributable fractions are lower, power to identify an interaction between tobacco and alcohol co-use and cancer can be limited because of the exceptionally large sample sizes needed for such analyses. Very large initiatives are needed, such as the Our Future Health study that aims to recruit 5 million participants in the United Kingdom,[263] and pooling projects such as a 2025 study that examined the association of alcohol and pancreatic cancer pooled across 30 cohorts.[84]

There are significant gaps in the literature concerning specific populations disproportionately affected by tobacco and alcohol co-use and relationship to cancer risk. Further research on the epidemiology of tobacco and alcohol co-use is needed in key populations including sexual minorities, Hispanic populations, American Indian and Alaska Native populations, and other demographic groups. Creative new approaches are needed for this work as sampling and other considerations related to small populations create significant study design challenges.[264]

More research is needed to address cancer risks associated with lower levels of alcohol consumption and intermittent (i.e., nondaily) cigarette smoking. The risks of nondaily smoking and lower levels of alcohol consumption are lower than the risks associated with daily cigarette smoking or heavier alcohol consumption, but they can still be substantial.[265] Such studies could benefit from new analytical approaches as well as continued efforts at pooling studies to increase power.

Studies are needed to determine whether the use of e-cigarettes interacts with alcohol consumption to increase risk. Studies of e-cigarettes and alcohol in relation to cancer risk were not identified, and only a few studies have examined co-use of smokeless tobacco and alcohol in relation to cancer risk. Because of the large number of young people and adults co-using e-cigarettes and alcohol, and the wide variety of patterns of e-cigarette use, this is an important area for further research.

Studies are needed to better understand the potential cancer risk of co-use of smokeless tobacco and alcohol. Studies of co-use of smokeless tobacco and alcohol may benefit from being conducted among populations and/or countries where use of both smokeless tobacco and alcohol is common.

More experimental and epidemiological studies are needed to understand the effects of tobacco cessation and alcohol cessation or reduction, separately or together, on cancer risk. These could also inform the understanding of mechanisms linking increased risk associated with use. Such studies could also examine biomarkers of risk given the long latency of tobacco and alcohol caused cancer.

In addition, more epidemiological studies that examine associations between co-use of specific alcohol/tobacco product types are needed. This is especially important as new product types are introduced to the market and where aspects of their formulation or patterns of use might influence risk.

Concerning measurement, more efforts are needed to improve biomarkers, especially of long-term use, and to harmonize measures across cohort studies. Further research on biomarkers of exposure over longer time periods could strengthen epidemiological studies of tobacco and alcohol co-use. Behavioral and etiological studies are needed in subpopulations that experience health disparities or are most vulnerable to co-use of tobacco and alcohol. Identification of additional biomarkers of exposure, especially markers related to long term alcohol consumption, could strengthen etiological studies and evaluation of interventions. Biomarkers of tobacco and alcohol exposure are an important adjunct to self-report. For tobacco use, the availability of serum markers of current or recent tobacco exposure has strengthened exposure assessment and inference. Short-term (current blood alcohol content) and medium-term (consumption over the past weeks) biomarkers of alcohol consumption are available,[227] but markers of lifetime consumption such as epigenetic changes, especially those relevant for lower levels of drinking, could potentially improve risk estimates.

Conclusions

1. **Tobacco and alcohol use independently cause multiple types of cancer, and even low levels of use increase the risk for specific cancers.** Smoking one or fewer cigarettes per day is known to increase the risk of numerous cancers. For alcohol, the risk of female breast cancer increases at less than one drink per day, and head and neck cancer risk increases with one drink per day and possibly at lower levels of consumption.
2. **Strong evidence exists for a synergistic increase in risk of squamous cancers of the head, neck, and esophagus caused by tobacco and alcohol co-use.** Numerous studies conducted in several countries over many years find that co-use of tobacco and alcohol, especially when use levels are high, results in synergistic increase in risk of head and neck and esophageal cancers. Limited evidence is available to determine whether there is a synergistic increase in cancer risk due to tobacco and alcohol co-use for several other cancer sites. For example, there have been mixed results for studies examining synergistic effects of tobacco and alcohol exposures on female breast cancer risk.
3. **Increased risk of head and neck cancers caused by alcohol use in people with aldehyde dehydrogenase 2 (ALDH2) deficiency, an autosomal recessive disorder that reduces the rate of metabolism of acetaldehyde, provides strong evidence for the causal relationship and underlying biological mechanism linking alcohol to head and neck cancers.** ALDH2 deficiency is especially common in people of East Asian ancestry, resulting in the alcohol flushing syndrome. Continued high prevalence of alcohol use in East Asian populations, especially among men, leads to a strong opportunity for intervention at the clinical and population level.
4. **Improved measurement would strengthen studies of the effect of tobacco and alcohol co-use on cancer risk**. Improving and implementing standardized measures of lifetime tobacco and alcohol exposure (including measures of frequency and duration of use, initiation, cessation, and product types used) are needed to strengthen the evidence base. Developing longer-term biomarkers of exposure and effect for tobacco and alcohol is also a significant gap in the currently available research tools; such biomarkers will advance understanding of co-use risks.
5. **Tobacco cessation reduces the risk of numerous cancers, and alcohol reduction or cessation reduces the risk of cancers of the aerodigestive tract.** As of 2026, few studies have addressed the effects of quitting or reducing both tobacco and alcohol use on cancer risk. Overall, the synergistic increases in risk of head, neck, and esophageal cancers and the potential for synergistic increases in risk for other cancers provide strong support for further developing and implementing interventions to address the co-use of tobacco and alcohol.

Appendix 4A Literature Search Strategy

The literature search included three phases: (1) identify cancer types causally associated with tobacco, (2) identify cancer types causally associated with alcohol, and (3) identify synergy between tobacco and alcohol in the causal pathways of the identified cancer types in phases one and two. To identify studies since 2010 (since the publication of IARC monographs in 2010 and 2012),[1,2] searches were conducted in PubMed in 2023 to identify studies of the synergy between alcohol, tobacco, and the following cancer sites: oral cavity, pharynx, esophagus, stomach, colorectal, liver, pancreas, larynx, lung, bronchus, skin melanoma, female breast, cervix uteri, corpus uterus, ovary, prostate, testis, urinary bladder, kidney, renal pelvis, brain, thyroid, Hodgkin lymphoma, non-Hodgkin lymphomas, myeloma, and leukemias. These sites were chosen based on specific cancer sites examined in the IARC reports.[1,2] Search strings used terms for each cancer site in combination with the following search terms: [tobacco or nicotine or cigarette] and [alcohol or "alcohol use" or drink*] and [synergy OR synergistic OR "interaction" OR "joint effect" OR "joint effects" OR "multiplicative effect" OR "amplified effect" OR "synergistic"] and [cancer OR neoplasm*]. Reference lists of relevant articles were also reviewed to identify important studies published before 2010. Relevant articles (including notable studies published prior to 2010) are described in a narrative format in the chapter and in Table 4.4 and Appendix 4B. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

Appendix 4B Select Studies Examining Synergy Between Tobacco and Alcohol Use and Cancer

Cancer type	Study	Study design	Sample	Results
Lung	Brenner et al. 2019[138]	Pooled analysis	Data from International Lung Cancer Consortium and Synergy Consortium Study. 22 participating studies: North America (11), Europe (8), Asia (3), and European Prospective Investigation into Cancer; 19,149 cases, 362,340 controls	• Interaction analysis between smoking and drinking suggested lung cancer risk was associated with higher levels of smoking, with an interaction observed for those in the highest smoking and alcohol consumption groups: effect estimate deviated from that expected on the additive scale (aOR = 11.23, 95% CI = 10.29–12.26, ≥20 grams/day alcohol, ≥10 pack years). • Test for multiplicative interaction between alcohol consumption and smoking intensity was also significant ($p < .001$). • Positive interaction between high pack years of smoking and elevated levels of drinking.
Lung	Viner et al. 2019[139]	Cohort study	Alberta, Canada; 2001–2017; 6,607 participants (2,370 cancer cases, of which 210 were lung cancer)	• Lung cancer risks were significantly elevated compared to non-smokers for men (aHR = 10.16, 95% CI = 4.29–24.04) and women (aHR = 20.05, 95% CI = 10.92–36.70) who smoked 20 pack years or more, but there was no significant synergistic interaction between tobacco and alcohol use on lung cancer risk ($p = .81$)

Appendix 4B (continued)

Cancer type	Study	Study design	Sample	Results
Liver	Aglago et al. 2025[149]	Nested case-control	10 European countries; recruited 1992–2000; 450,112 participants (255 liver cancer cases; 108 used in analysis)	• Positive relationship between smoking and HCC risk: Log-transformed cotinine, one standard deviation increments: aOR = 1.35, 95% CI = 1.11–1.63. Log-transformed nicotine, one standard deviation increments: aOR = 2.13, 95% CI = 1.28–3.56. Self-reported current versus never smoking: aHR = 2.46, 95% CI = 1.77–3.43. Self-reported former smoking versus never smoking: aHR = 1.31, 95% CI = 0.93–1.83. • Positive relationship between alcohol and HCC risk: Log-transformed 2-hydroxy-3-methylbutyric acid, one standard deviation increments: aOR = 3.04, 95 CI = 1.31–7.04. Compared with self-reported light drinking: self-reported never drinking aHR = 1.35, 95% CI = 0.59–3.10; moderate/never heavy drinking aHR = 0.94; 95% CI = 0.56–1.59; former drinking aHR = 3.20, 95% CI = 1.70–6.03; periodic heavy drinking (based on lifetime intake of >40 grams/day in women, 60 grams/day in men, reported at 1 or more assessment points) aHR = 1.98, 95% CI = 1.11–3.54; always heavy drinking (based on lifetime intake of >40 grams/day in women, >60 grams/day in men, reported at all assessment points)(M. Jenab, Ph.D., personal communication, September 2, 2025) aHR = 5.51, 95% CI = 2.39–12.7; unknown aHR = 1.79, 95% CI = 0.80–4.03. • Synergy indices for self-reported smoking and alcohol use behaviors (synergy index = 1.27, 95% CI = 0.98–1.66), and smoking and alcohol biomarkers (synergy index for 2-hydroxy-3-methylbutyric acid and cotinine = 1.59, 95% CI = 0.58–4.33; synergy index for 2-hydroxy-3-methylbutyric acid and nicotine = 1.36, 95% CI = 0.60–3.10), were not significant.
Liver	Bui et al. 2024[146]	Case-control	South Korea; 2009–2019; 15,352 cancer cases (92,112 controls)	• Among men, there was an additive effect for current smoking and current alcohol consumption on liver cancer risk, relative excessive risk due to interaction (RERI = 0.28 [95% CI, 0.15–0.40]), and a multiplicative interaction (multiplicative aOR = 1.20 [95% CI, 1.07–1.34]). When examining by smoking and alcohol use intensity, there was an additive effect for light-to-moderate drinking (0.1–0.28 grams/day) and heavy smoking (>20 pack years), on liver cancer risk, RERI = 0.34 (95% CI, 0.16–0.51) and a multiplicative interaction (synergy index = 2.75 [95% CI = 1.85–3.66]; multiplicative aOR = 1.28 [95% CI = 1.11–1.49]). • Among women, there was no significant interaction between smoking and alcohol consumption on liver cancer risk.

Appendix 4B (continued)

Cancer type	Study	Study design	Sample	Results
Liver	Kuper et al. 2000[145]	Case-control	Athens, Greece; 1995–1998; 333 cancer cases (360 controls)	• Positive, dose–response relationship between smoking and liver cancer risk (aOR for <2 packs/day = 1.6 [95% CI, 0.8–2.9]; aOR for ≥2 packs/day = 2.5 [95% CI, 1.1–5.5]; trend p = .03). • Alcohol consumption of >40 glasses per week had a positive but nonsignificant association with liver cancer risk (aOR = 1.9, 95% CI = 0.9–2.9). • Heavy smoking (≥2 packs/day) and heavy drinking (≥40 glasses/week) had a multiplicative association with liver cancer risk (aOR = 9.6 for both exposures [95% CI, 3.4–27.5]).
Liver	Pirie et al. 2013[169]	Cohort study	United Kingdom; 1996–2011; 1.2 million women	• Among women who smoked versus those who never smoked, sensitivity analyses to examine those who did not drink alcohol or consumed <3 units of alcohol per week: Liver cancer mortality adjusted RR = 1.43 (95% CI 1.11–1.85). • Alcohol consumption attenuated the association between tobacco use and liver cancer, but association remained significant.
Liver	Marrero et al. 2005[143]	Case-control	United States; 2002–2003; 70 cancer cases (70 cirrhosis controls and 70 controls with no liver disease)	• Hepatocellular cases (HCC) compared to cirrhosis controls: Cigarette smoking of ≥20 pack years was associated with increased risk of HCC (aOR = 4.9, 95% CI = 2.2–10.6) compared to never smoking. • HCC cases compared to cirrhosis controls: Alcohol consumption of ≥1,500 grams per year was associated with increased risk of HCC (aOR = 5.7, 95% CI = 2.4–13.7) compared to never alcohol use. • HCC cases compared to cirrhosis controls: Exposure to both smoking and alcohol was associated with increased risk of HCC (aOR = 7.2, 95% CI = 2.2–14.1) compared to smoking alone (aOR = 2.5, 95% CI = 1.7–15.0) or alcohol alone (aOR = 1.4, 95% CI = 1.1–4.0), with a synergistic index of 3.3 (95% CI, 1.8–5.7). • HCC cases compared to cirrhosis controls: The presence of 3 risk factors (alcohol, smoking, and obesity), compared to only 2 risk factors, was associated with increased risk of HCC (aOR = 7.4, 95% CI = 2.1–14.6), with a synergistic index of 1.6 (95% CI, 1.1–4.3).

Appendix 4B (continued)

Cancer type	Study	Study design	Sample	Results
Breast (female)	Gaudet et al. 2013[162]	Cohort study	United States; 1992–2007; 73,388 women (3,721 invasive breast cancer cases)	• Alcohol consumption did not confound associations with smoking status, although neither current nor former smoking were associated with risk among those who reported never drinking (*p* for interaction = .11). • Association between active smoking and breast cancer incidence was not attenuated by the inclusion of alcohol intake in the multivariate model. • Association was statistically significant only in those who reported current and formers drinking in analyses that stratified on alcohol consumption, although the CIs for the association in those who reported never drinking were wide. • No synergistic effect reported.
Breast (female)	Gaudet et al. 2017[155]	Pooled analysis	14 cohort studies; 934,681 (36,030 invasive breast cancer cases)	• People who currently drank 2 or more drinks per day and who formerly smoked (aHR = 1.33, 95% CI = 1.19–1.49) or currently smoked (aHR = 1.32, 95% CI = 1.16–1.49) were at slightly greater risk than expected under a multiplicative model (expected aHRs = 1.22 and 1.22, respectively). • People who currently drank ≥3 drinks per day and smoked >10 years prior to first birth (aHR = 1.85, 95% CI = 1.55–2.21) were at noticeably greater risk than expected under the multiplicative model (expected aHR = 1.43). • Results support a synergistic relationship between smoking initiation and adult alcohol drinking and increased breast cancer risk.
Breast (female)	Gram et al. 2005[163]	Cohort study	Norwegian/Swedish Cohort Study; 1991–2000; 102,098 women	• Study found that women who initiate smoking prior to menarche or a first birth and continue to smoke for at least 20 years have a small increase in breast cancer risk relative to never smokers. • People who did not drink experienced a similar increase in risk as people who drank. • No synergistic effect reported.
Breast (female)	Hamajima et al. 2002[164]	Collaborative reanalysis	Global; Collaborative Group on Hormonal Factors in Breast Cancer; 58,515 cases with invasive breast cancer; 95,067 controls	• Relationship between smoking and breast cancer was confounded by alcohol: when comparing 22,255 women with breast cancer and 40,832 controls who reported drinking no alcohol, smoking was not associated with breast cancer (versus never smoking, adjusted RR for ever-smoking = 1.03, 95% CI = 0.98–1.07, and for current smoking adjusted RR = 0.99, 95% CI = 0.92–1.05). • No synergistic effect reported.
Breast (female)	Jones et al. 2017[158]	Longitudinal cohort study	United Kingdom; 2003–2013; 102,927 women (1,815 breast cancer cases)	• No significant interaction between smoking and drinking on breast cancer risk (*p* = .41)

Appendix 4B (continued)

Cancer type	Study	Study design	Sample	Results
Breast (female)	Knight et al. 2017[159]	Case-control study	United States, Canada, Denmark—Women's Environmental, Cancer, and Radiation Epidemiology Study; 2001–2004, 2009–2012; 1,521 cases (contralateral breast cancer); 2,212 controls (unilateral breast cancer)	• Interaction between current drinking and smoking was statistically significant (p = .008), whereas the interaction between any drinking and smoking history was not (p = .76). • The consumption of cigarettes and alcohol at the time of first diagnosis had the strongest association with increased contralateral breast cancer risk. • Women who drink and smoke following diagnosis continue to be at increased risk.
Breast (female): *BRCA*, *BRCA2* mutations	Li et al. 2020[160]	Prospective and retrospective cohort analyses	21 centers in Western countries; 9,845 women (9,232 for retrospective and 3,886 for prospective)	• In the retrospective analysis, ever alcohol and ever smoking (i.e., at least 1 glass per month for 1 year and at least 1 pack of cigarettes per month for 1 year; aHR = 1.43, 95% CI = 1.13–1.81; p = .003) was associated with increased synergistic breast cancer risk for *BRCA2* mutation carriers.
Breast (female)	Pirie et al. 2013[169]	Cohort study	United Kingdom; 1996–2011; 1.2 million women	• Among women who smoked versus those who never smoked, sensitivity analyses to examine those who did not drink alcohol or consumed <3 units of alcohol per week: Breast cancer mortality RR: 1.06 (95% CI, 0.95–1.18) among women who consumed little to no alcohol. • Alcohol consumption attenuated many associations between cigarette smoking and breast cancer (no longer significant).
Breast (female)	Viner et al. 2019[139]	Cohort study	Alberta, Canada; 2001–2017; 26,607 participants (2,370 cancer cases, of which 483 were breast cancer)	• A "U-shaped" multiplicative interaction was observed for breast cancer (p = .05) when cigarettes and alcohol were used in combination—moderate alcohol consumption and minimal cigarette smoking were associated with lower cancer incidence. • This interaction rapidly changes in a multiplicative fashion when alcohol consumption and pack years increase.

Appendix 4B (continued)

Cancer type	Study	Study design	Sample	Results
Breast (female)	Zeinomar et al. 2019[161]	Secondary data analysis of cohort study	United States, Canada, Australia; 10.4 years; 17,435; 1,009 incident breast cancer cases	• Significant 3-way interaction between familial risk profile, alcohol consumption, and cigarette smoking (interaction p = .01). • Among women who were regular drinkers, there was a significant multiplicative interaction (p = .005) between familial risk profile and smoking status (current versus never) on breast cancer risk (aHR = 1.39, 95% CI = 1.13–1.71), largely driven by women with a very high familial risk profile. • 30% increased overall breast cancer risk for women at the 95th percentile of familial risk profile (5-year BOADICEA > 6.55%) who reported currently smoking at baseline and regularly drinking (aHR = 1.30, 95% CI = 0.99–1.71).
Thyroid	Kitahara et al. 2012[166]	Pooled analysis, 5 prospective studies	United States; 746,097 people at baseline; 89,527 incident thyroid cases	• Compared to never smoking and nondrinking, current smoking and drinking one or more drinks per week was associated with 36% reduced risk (joint aHR = 0.64, 95% CI = 0.47–0.86). • The joint aHR was slightly weaker than the aHR for current smoking among people who did not drink alcohol (aHR = 0.46, 95% CI = 0.29–0.73) and similar to that observed for people who reported never smoking and reported ≥1 drink per week (aHR = 0.70, 95% CI = 0.56–0.87). • The relationship for the joint association of smoking and drinking appears sub-multiplicative (p for interaction = .004).
Thyroid	Yeo et al. 2022[167]	Cohort study	Korea; 2009; 9,699,104 people	• Statistically significant interaction found between smoking and alcohol consumption on thyroid cancer risk (p interaction < .001), with differences in risk by current smoking (never/former/current) and levels of alcohol consumption (none/mild/moderate/heavy), range of aHRs (versus never smoking and nondrinking) = 0.62–0.92. • Findings suggested a submultiplicative interaction slightly lower than expected from the independent effects from tobacco or alcohol use.
Prostate	Viner et al. 2019[139]	Cohort study	Alberta, Canada; 2001–2017; 26,607 participants (2,370 cancer cases, of which 401 were prostate cancer)	• 3-way interaction model showed an additive, not a synergistic, effect between alcohol as a continuous variable (grams/day) and pack years consumed for prostate cancer. • Differences were significant for men (aHR = 10.16, 95% CI = 4.29–24.04) and women (aHR = 20.05, 95% CI = 10.92–36.70) among those who smoked 20 pack years or more.

Note. aHR = adjusted hazard ratio; aOR = adjusted odds ratio; CI = confidence interval; HCC = hepatocellular carcinoma; RERI = relative excess risk due to interaction; RR = risk ratio.

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Chapter 5
Physiological, Cellular, Molecular, and Genetic Basis of the Combined Effects of Tobacco and Alcohol Use on Carcinogenesis

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Chapter 5
Physiological, Cellular, Molecular, and Genetic Basis of the Combined Effects of Tobacco and Alcohol Use on Carcinogenesis

Introduction

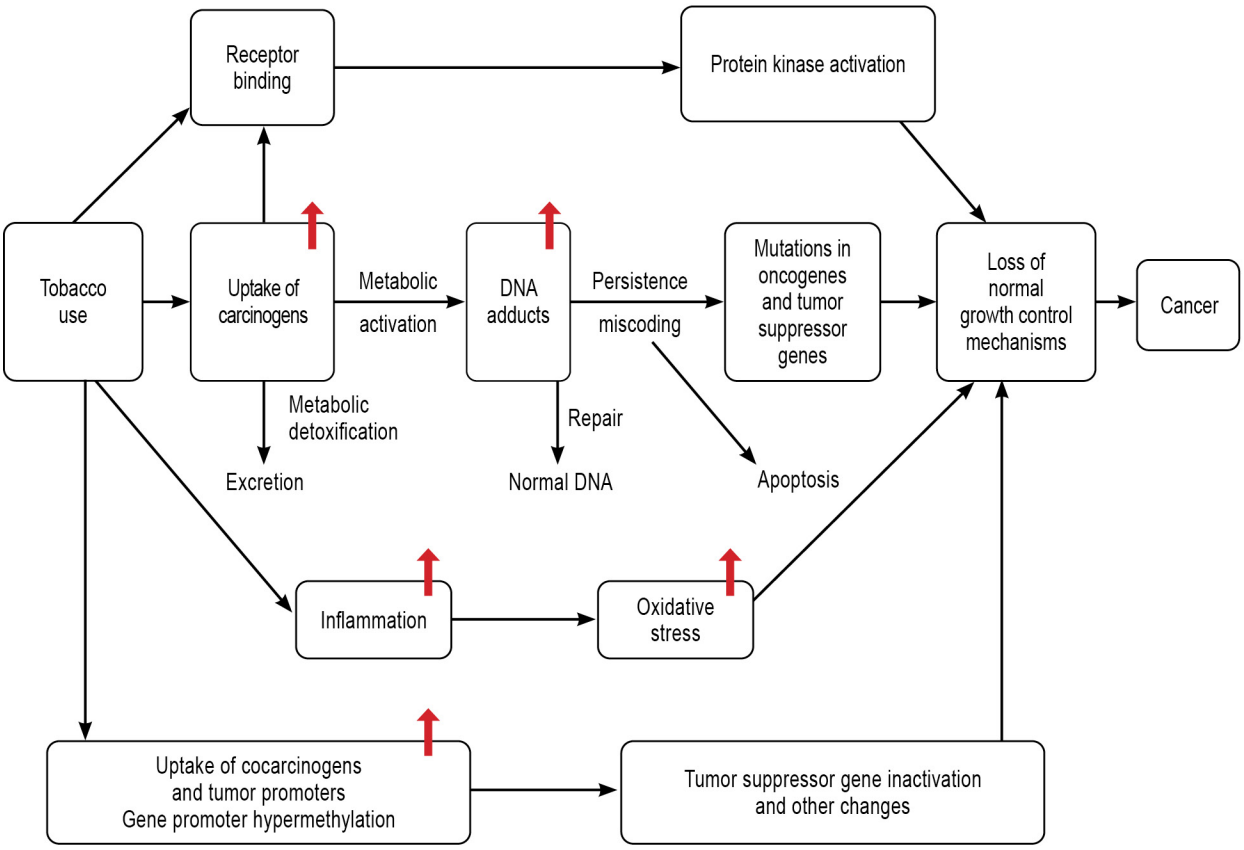
This chapter reviews research on the biological effects of tobacco and alcohol, used independently and together, and describes the underlying mechanisms that contribute to their carcinogenesis, including synergistic associations observed for some cancer sites. The chapter also highlights that much remains to be learned about these interactions, their combined roles in the initiation and/or promotion of cancer, and how these processes may be influenced by cessation of tobacco use and cessation or reduction in alcohol use.

As described in chapter 4, cigarette smoking causes cancers of the oral cavity, pharynx, larynx, esophagus, lung (including trachea and bronchus), stomach, liver, pancreas, kidney and ureter, bladder, cervix, colorectum, and blood (acute myeloid leukemia).[1] Suggestive evidence is also noted for other cancer types, including female breast,[1-3] although the totality of the evidence remains inconclusive for female breast cancer.[1,3,4] As discussed in chapter 4, tobacco products other than cigarettes also cause cancer; for example, cigars cause cancer of the oral cavity, larynx, esophagus, and lung,[5] and smokeless tobacco causes cancer of the oral cavity, esophagus, and pancreas.[2,6] In the United States, cigarette smoking is responsible for an estimated 170,000 cancer deaths annually, accounting for 28.5% of all cancer-related deaths; in 2019, an estimated 19.3% of new cancer cases (~350,000) were attributed to cigarette smoking.[7,8] The mechanisms underlying tobacco-related carcinogenesis have been studied extensively and have been summarized in numerous publications, including the U.S. Surgeon General's reports on smoking and tobacco use,[1,9,10] National Cancer Institute reports and monographs related to use of cigars and smokeless tobacco,[5,11] and several International Agency for Research on Cancer (IARC) monographs related to tobacco smoking and smokeless tobacco use.[2,6,12-14]

The use of tobacco products results in exposure to numerous carcinogens both for the individual who uses tobacco and for other people via exposure to secondhand smoke. Carcinogen exposure is especially high from combusted tobacco products; smokeless tobacco use also involves exposure to carcinogens, with overlapping but distinct carcinogen exposures from these product types.[2,6,15,16] Upon exposure, carcinogens from tobacco products react with and damage DNA, which can result in mutations occurring in critical genes leading to dysregulation of normal cellular growth and ultimately in cancer.[2,9,17] DNA damage may also occur indirectly through the action of reactive oxygen species or the generation of lipid peroxidation products resulting from inflammation. However, mechanisms related to DNA damage are only one mode of action. Other nongenotoxic mechanisms related to the induction of inflammatory responses, epigenetic changes, and receptor-specific binding also make important contributions to the carcinogenic process. A general overview of these mechanisms is provided in Figure 5.1; a vertical red arrow

indicates mechanisms described for tobacco-related carcinogenesis that may be enhanced or affected by alcohol consumption.

Figure 5.1 Overview of Tobacco-Related Carcinogenic Mechanisms and Where the Influence of Alcohol Consumption Could Play a Role



Note. Mechanisms of tobacco-related carcinogenesis and processes where interaction with alcohol can occur (red arrows) result in an enhancement of the effects. Alcohol may increase the uptake of carcinogens or cocarcinogens by increasing the solubility of these compounds or by increasing the metabolism and activation of procarcinogens by inducing the expression of cytochrome P450 enzymes. Alcohol may contribute to increased inflammation, oxidative stress, and DNA damage directly through ethanol's major metabolite, acetaldehyde, or by increasing the activation of genotoxic compounds and DNA-damaging reactive electrophiles resulting from inflammation and oxidative stress. *Source*: Adapted from USDHHS 2014: Figure 6.4.[1]

As will be discussed later in this chapter ("Potential Interactions of Electronic Cigarettes and Alcohol"), the potential carcinogenicity of electronic cigarettes (e-cigarettes) and other emerging tobacco products remains unclear. However, trends in e-cigarette use underscore the need for research in this area. Among adults in the United States, e-cigarette use increased from 4.5% in 2019[18] to 7.0% in 2024.[19] Although e-cigarette use among U.S. middle and high school students decreased from a high of 20.0% in 2019[20] to 5.9% in 2024,[21] e-cigarettes remain the most commonly used tobacco product among adolescents in the United States.[21]

Alcohol consumption is also a known cause of cancer. In the United States in 2019, an estimated 5.4% of incident cancers (96,730 cases; 42,400 among men, 54,330 among women) were caused by alcohol consumption.[7] Alcohol consumption was estimated to cause 4.1% of cancer deaths in the United States in 2019.[7] Alcohol consumption has been classified as a Group 1 human carcinogen by IARC since 1988.[22] This classification is based on a large body of epidemiological and mechanistic research. Increased risk in association with alcohol consumption has long been observed for cancers of the liver, oral cavity, pharynx, larynx, and esophagus. On the basis of studies reviewed in the 2010 IARC monograph, *Alcohol Consumption and Ethyl Carbamate*, alcohol is also thought to be a cause of female breast and colorectal cancers.[23] In addition, the World Cancer Research Fund/American Institute for Cancer Research has concluded that alcohol is a probable cause of stomach cancer,[24] although IARC has not updated its review of alcohol and stomach cancer. Possible associations between alcohol use and increased risk for cancer of the pancreas have also been observed.[23,25] Major mechanisms for alcohol-induced cancer are both direct and indirect and include the following: (1) genotoxic effects of acetaldehyde, the primary metabolite of ethanol; (2) production of reactive oxygen or nitrogen species; (3) ethanol's ability to act as a solvent for multiple carcinogens, resulting in their greater absorption; and (4) influence of alcohol on folate and estrogen metabolism. Although much is known about the biological mechanisms through which alcohol induces cancer, these mechanisms are not yet fully understood and may vary depending on the target organ considered.

The fact that both tobacco and alcohol use increase the risk for several types of cancer, and that co-use of tobacco and alcohol is common, has motivated efforts to examine interactions between their use and cancer risk. The evidence for a synergistic effect of combined exposures to tobacco and alcohol is well established for cancers of the aerodigestive tract (oral cavity, pharynx, larynx, and esophagus).[23] Evidence from observational studies also suggests that co-use of tobacco and alcohol may result in synergistic effects on risk for colorectal, liver, lung, and female breast cancer.[26-30] However, for these sites, it is not yet established if tobacco and alcohol interact to influence risk in those who use or have exposure to both substances. This evidence is discussed further in chapter 4.

Exposure to Tobacco- and Alcohol-Related Carcinogens

Co-use of tobacco and alcohol can result in biological interactions that influence initiation and/or promotion of carcinogenesis. Specifically, these interactions could result in sustained proliferation of abnormal cells, maintenance of tumor growth, and/or alterations in cell death processes.[2,23] Tobacco-related carcinogenic mechanisms that may be influenced by alcohol consumption include increased exposure to carcinogens, particularly acetaldehyde; inflammation and oxidative stress; epigenetic changes; and tumor promotion (Table 5.1). Several metabolic and genetic factors may underlie these interactions and could result in different outcomes depending on the cancer sites considered, as well as the histological subtypes at different cancer sites. This chapter provides an overview of the carcinogenic mechanisms of tobacco and alcohol exposures derived from both mechanistic evidence and germline susceptibility. The review of evidence emphasizes aspects of interaction, where available, and discusses how alcohol may enhance or interfere with the main mechanisms described for tobacco carcinogenesis and vice versa.

Table 5.1 List of Tobacco-Related Carcinogenic Mechanisms Influenced by Alcohol and Relevance to Specific Cancer Sites

Candidate mechanism	Mechanisms of alcohol interaction	Relevant cancer site
Increased exposure to carcinogens	Increased exposure to acetaldehyde	Oral cavity and pharynx Esophagus Colorectum Lung[a]
	Increased carcinogen solubility/mucosal permeability	Oral cavity and pharynx Esophagus
	Effects on activation/detoxification of carcinogens	Liver Esophagus Lung[a]
Inflammation and oxidative stress	Additional production of reactive oxygen species and lipid peroxidation products and binding of receptors	Liver Esophagus Pancreas[a] Lung[a]
Epigenetic changes	Oncogene promotion and tumor suppressor silencing	Pancreas[a] Colorectum
Tumor promotion	Variation in expression of oncogenes or tumor suppressor genes	Esophagus Liver Lung[a]

[a]There is some evidence for an interaction between tobacco and alcohol use on carcinogenic mechanisms.
Source: IARC 2010,[23] IARC 2012,[2] Li & Jiao 2003,[31] Li et al. 2019,[32] Lachenmeier et al. 2017,[33] Malkinson 2005,[34] Seitz & Stickel 2007,[35] USDHHS 2010,[9] Wight & Ogden 1998,[36] Yang et al. 2019.[37]

The physiological studies of tobacco and alcohol co-use summarized in this chapter often focus on people who consume high levels of alcohol or on people with alcohol use disorder (AUD). Consequently, the physiology of tobacco use combined with moderate alcohol consumption is not as well understood. For example, fewer studies have investigated whether there are thresholds for identified mechanistic effects of tobacco and alcohol use. On the other hand, a growing body of large-scale genetic studies including a wide range of tobacco and alcohol use behaviors are starting to emerge (reviewed in chapter 6). Evidence concerning alcohol cessation or reductions in use and the effects of these behaviors on reversing carcinogenesis is modest for head and neck cancers and sparse for other cancers.[38] Therefore, this chapter attempts to generate hypotheses in this domain and highlight needed areas of investigation.

Finally, interactions between tobacco and alcohol at multiple levels, involving genetic, cellular, physiological, and behavioral processes, combine to affect use behaviors and cancer risk.[38] Chapters 4, 6, and 7 further discuss these interactions. This growing body of research supports addressing the co-use of tobacco and alcohol as a target for cancer prevention and control.

Exposure to Tobacco-Related Carcinogens

Tobacco smoke contains more than 7,000 chemicals, and at least 80 of them are recognized carcinogens.[39] Among these carcinogens, some classes of compounds are tobacco-specific, such as tobacco-specific nitrosamines, while others can also originate from other sources. Non-tobacco specific carcinogens include polycyclic aromatic hydrocarbons (PAHs); aromatic amines; volatile carcinogens such as formaldehyde, acetaldehyde, acrolein, 1,3-butadiene,

and benzene; and various metals. Most of these compounds are metabolized by enzymes that convert them to more water-soluble products that can be eliminated by the body. These conversions are mediated by enzymes such as cytochrome P450, glutathione transferase, UDP-glucuronosyltransferases and sulfotransferases, aldo-keto reductases, and N-acetyltransferases, and can also result in the production of reactive intermediates.[40] The consequent generation of electrophilic compounds or epoxides can result in species able to bind covalently with cellular targets and to damage DNA by forming DNA adducts. If not repaired, or if repaired incorrectly, these adducts can result in genetic changes affecting specific oncogenes or tumor suppressor genes and ultimately translate into abnormal cellular proliferation and carcinogenesis.

Exposure to Alcohol Carcinogens

Alcohol consumption refers to the drinking of wine, beer, and/or spirits, which have ethanol and water as main components in different proportions. These beverages are marked by different flavor profiles due to various congeners and volatile and nonvolatile compounds, deriving from the fermentation or distillation processes and from various added ingredients.[23] In addition, a wide variety of chemicals, impurities, and contaminants are found in alcoholic beverages, including known carcinogenic compounds such as PAHs[41]; N-nitrosamines, which have been detected in beer[42]; and mycotoxins such as ochratoxin and aflatoxin, which have been found in some traditionally processed African alcoholic beverages.[43] By far the main actor in alcohol-related carcinogenesis is thought to be ethanol, as consistent differences in cancer risk among beer, liquor, and wine have not been found.[24,44] There is wide agreement that ethanol is the major factor contributing to carcinogenesis related to alcohol consumption and that cancer risk depends on the amount of ethanol consumed regardless of the type of alcoholic beverage.[23,24,44]

Alcohol and Increased Absorption of Tobacco-Related Carcinogens

Alcohol consumption may contribute to an increase in the level of exposure to tobacco-related carcinogens by acting as a solvent and resulting in more efficient cellular absorption.[45] Additionally, alcohol may increase the permeability of the mucosa in the digestive tract, specifically the areas that come into direct contact with carcinogens resulting from exposure to both tobacco and alcohol compounds.[36,46-48] A study on the effect of alcohol on pig oral mucosal permeability, using an *in vitro* perfusion model, showed an increase in the penetration of the tobacco-specific carcinogen N'-nitrosonornicotine (NNN) upon exposure to solutions of 25%–30% ethanol, but not of 50% ethanol, possibly due to higher concentrations of alcohol acting as a fixative and reducing membrane permeability.[49] For human ventral tongue, 15% ethanol increased mucosal permeability compared to 0% and 5% ethanol for tongue mucosa obtained at autopsy.[47] Additional studies are needed to determine alcohol's effects on mucosal permeability in the human oral cavity, and how this may influence tobacco-alcohol interactions and cancer risk.[50]

More consistent evidence has been observed in studies that compared absorption of chromium-51-EDTA (51Cr-EDTA) in study participants with AUD and control participants. The 51Cr-EDTA method used examines the urinary excretion of orally administered chromium and allows a direct and accurate assessment of intestinal permeability in humans.[51-53]

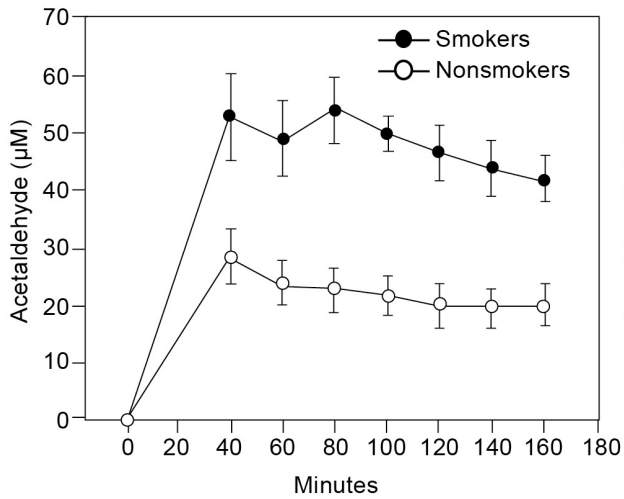
These studies show that alcohol disrupts the intestinal barrier, increasing its permeability by acting directly on the permeability of the epithelial cells and affecting the junctions between them. Alcohol and its metabolites induce oxidative stress, which damages the cells directly and weakens cell membranes while also disrupting the proteins that create the tight junctions linking cells and the proteins that stabilize the cells' cytoskeletons.[54] Such effects may be reversible. In one study, after 3 weeks of abstinence from alcohol, these effects disappeared, with levels of absorption of 51Cr-EDTA returning to those found in control participants.[53] In another study, among people with AUD and high levels of intestinal permeability, intestinal permeability decreased after 3 weeks of abstinence.[51] A growing body of literature addresses effects of alcohol consumption, especially AUD, on gut microbial composition, inflammation, and the integrity of the intestinal barrier. This literature has implications for treatment of AUD and mechanistic understanding of the health effects of alcohol.[46,48]

Role of Alcohol-Mediated Increase in Exposure to Tobacco-Related Carcinogens and Effects of Reduced Alcohol Intake

Several studies have examined the time course of changes in exposure related to the combined use of tobacco and alcohol. These studies are important because they provide a mechanistic underpinning for epidemiological observations of increased risk associated with the use and co-use of tobacco and alcohol. As discussed above and illustrated in Figure 5.2, salivary acetaldehyde increases dramatically with concurrent tobacco smoking and alcohol consumption. This relative increase could influence the risk for cancers of the upper gastrointestinal tract.[55] Salivary acetaldehyde levels are sensitive to the timing of tobacco and alcohol co-use and further characterization of the accumulation of acetaldehyde in saliva might help characterize the risk of co-use.[56]

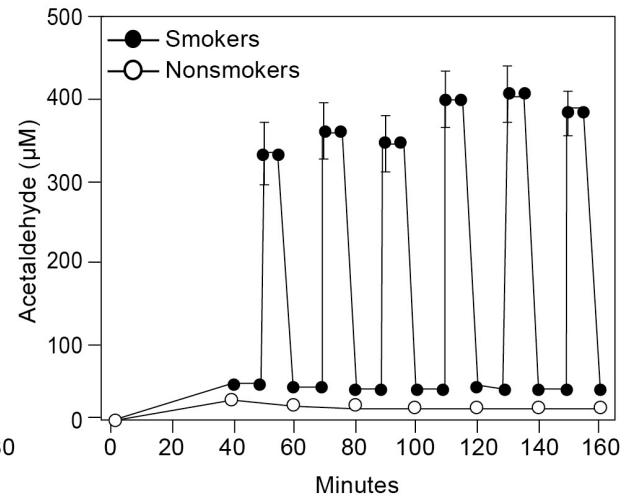
Figure 5.2 In Vivo Salivary Acetaldehyde After Ethanol Ingestion

Panel A: Acetaldehyde levels after ethanol ingestion



A: *In vivo* salivary acetaldehyde levels after ethanol ingestion in people who smoke, without concomitant smoking, (black dots) and in people who did not smoke (white dots).

Panel B: Acetaldehyde levels after ethanol ingestion and active smoking



B: To measure the salivary acetaldehyde derived from the combined and simultaneous use of ethanol and tobacco, the same procedure was repeated in people actively smoking a cigarette. After ingestion of the ethanol dose, study participants smoked 1 cigarette every 20 minutes, and saliva samples were collected every 10 minutes for 160 minutes. The graph summarizes *in vivo* salivary acetaldehyde after ethanol ingestion in people who smoke, with concomitant smoking, (black dots) and in people who did not smoke (white dots).

Note. Study participants (n = 6 individuals who did not smoke; n = 7 individuals who smoked) were administered 0.8 g of ethanol/kg body weight, and salivary acetaldehyde levels were measured at several time points after ethanol ingestion. Differences between acetaldehyde concentrations are significant at all time points from 40 minutes to 160 minutes ($p \leq .05$).
Source: Reproduced with minor formatting changes from Salaspuro and Salaspuro 2004[55] with permission from *International Journal of Cancer* (publisher: John Wiley & Sons, Inc; Copyright © 2004 Wiley-Liss, Inc.).

In an experimental study of drinking cessation, the induction of cytochrome P450 2E1 (CYP2E1) by ethanol declined within 3, 8, and 15 days after alcohol consumption ceased.[57] Similarly, a mouse model has shown that 1 week after alcohol cessation, the effects of ethanol exposure on the hepatic imbalance of cellular redox homeostasis—regulated by the ratio of nicotinamide adenine dinucleotide, reduced nicotinamide adenine dinucleotide hydrogen (NADH) and nicotinamide adenine dinucleotide, oxidized (NAD^+)—disappears.[58] Additionally, alcohol-induced gut inflammation can be moderated by the gut microbiome and dietary intake.[54] Multiple organs, including the gut, show recovery after cessation of alcohol use.[59] For example, after 3 weeks of abstinence, gut permeability largely recovers, inflammatory pathways are partially improved, and gut microbiota begin to return to more typical compositions.[51,59] Together these mechanistic studies support the idea that cessation of alcohol consumption can reduce the risks associated with elevated exposure to acetaldehyde, to reactive species resulting from alcohol-induced enzymatic activity, and to alcohol-related increased gut permeability. A 2024 review of the epidemiological literature conducted by IARC concluded that there is

sufficient evidence that reduction or cessation of alcohol use reduced the risk of oral cancer and esophageal cancer (squamous cell carcinoma); limited evidence for reduced risk of larynx, colorectal, and female breast cancer; and inadequate evidence for reduced risk of pharynx and liver cancer.[38,60] Studies including large numbers of people who stop drinking or reduce the amount they drink for multiple years are needed to determine the effects of cessation and reduction on cancer risk at sites where the evidence is currently limited or inadequate.

The Role of Acetaldehyde

Increased Levels of Exposure to Acetaldehyde

Multiple mechanisms are thought to underlie the causal relationship between alcohol and cancer; the strongest mechanistic evidence is for acetaldehyde, the primary metabolite of ethanol, which is also one of the main volatile compounds present in tobacco smoke.[23,25,61] Consumption of both tobacco and alcohol products results in increased exposure to this genotoxic compound. Aldehydes in tobacco smoke are important carcinogens, inducing mutagenic PdG (1,N^2 propano-2′-deoxyguanosine) DNA adducts and impairing DNA repair, but also inhibiting some other procarcinogens in tobacco smoke from damaging DNA.[62] Alcoholic beverages also contain acetaldehyde, and ethanol is metabolized into acetaldehyde in the body by alcohol dehydrogenase (ADH); acetaldehyde is further oxidized by aldehyde dehydrogenase (ALDH).[63] Hibberd and colleagues (2020)[63] review further details of the genetics and regulation of ADH in gastrointestinal and adipose tissues.

Acetaldehyde in alcoholic beverages is responsible for an initial increase in acetaldehyde in the saliva, but after a few minutes, conversion of ethanol into acetaldehyde dominates.[64] Acetaldehyde has been classified as a Group 1 carcinogen (indicating carcinogenic to humans) by IARC,[23] specifically in relation to head and neck and esophageal cancer through the induction of DNA damage. Additionally, the U.S. National Toxicology Program states that acetaldehyde is "reasonably anticipated to be a human carcinogen."[61]

IARC's Group 1 classification for acetaldehyde is further supported by the observation that individuals with genetic polymorphisms that reduce activity of aldehyde dehydrogenase 2 (ALDH2), for example rs671, which codes for an enzyme involved in detoxification of acetaldehyde, have an increased risk for alcohol-associated aerodigestive tract cancers.[65-69] Individuals with one copy of the polymorphism have lower ALDH2 activity; if these individuals consume alcohol they have higher risks of cancer, especially of the aerodigestive tract, than individuals who lack the polymorphism. People with two copies of the polymorphism still have less detoxification of acetaldehyde. Although these individuals tend to not drink alcohol because the experience is extremely unpleasant, if they do drink alcohol, they have very high cancer risks. As described in chapter 4, 35%–45% of people of East Asian descent (an estimated 540 million people) have an ALDH2 polymorphism reducing acetaldehyde detoxification and increasing cancer risk, making this group an important target for alcohol-related cancer prevention efforts.[66]

Role of the Oral Microbiome in Acetaldehyde Production

In the oral cavity, alcohol is metabolized mainly by the ADH enzymes present in cells of the oral mucosa and in oral microbes.[70] Metabolism of alcohol in the oral cavity is important for head and neck cancers, even though the liver is the primary site of alcohol metabolism.[71-74] The oral microbiome contributes substantially to the production of acetaldehyde in saliva, as shown by dramatically reduced acetaldehyde production after use of chlorhexidine (an antibacterial agent) mouthwash.[75] Although salivary acetaldehyde production and metabolism show high interindividual variation, there is a strong correlation between salivary ethanol and acetaldehyde levels after alcohol consumption.[75] Certain oral bacterial species, such as *Neisseria*, have very high ADH activity and acetaldehyde-producing capacity, and alcohol ingestion results in selection and increased production of these bacterial strains.[76] Bacteria of the oral mucosa present in saliva such as *Neisseria* species, *Rothia mucilaginosa*, *Streptococcus mitis*, *Streptococcus salivarius,* and *Prevotella histicola* can produce acetaldehyde from ethanol *in vitro*.[77] However, when a fixed amount of ethanol is added to whole samples of human saliva, acetaldehyde production is greater in the saliva with a lower abundance of *Neisseria* species.[78]

These observations point to the complex role of the microbiome for ethanol metabolism in the oral cavity. This role is influenced by the considerable variability of the oral microbiota composition and by the balance of different species. Species representation and composition is influenced by exposures, including tobacco exposures.[70,79-81] Salaspuro and Salaspuro (2004)[55] demonstrated that after ethanol ingestion, people who smoke have higher salivary acetaldehyde levels than people who do not smoke, and that salivary acetaldehyde production deriving from ethanol consumption is increased by active smoking. Specifically, as shown in Figure 5.2, after a dose of ethanol, the mean *in vivo* salivary acetaldehyde level in people who smoke was observed to be two times higher than in those who did not smoke. When the ethanol challenge was repeated during active smoking, the levels of salivary acetaldehyde were seven times higher in participants who smoked than in those who did not smoke. The authors suggested that these findings may be due to "microbial changes in the oral cavity of smokers, to inhibition of ALDH of the oral mucosa by tobacco smoke, and to the significant deposition of acetaldehyde from tobacco smoke in the saliva."[55] This study suggests a potential mechanism for the interactive effects of tobacco and alcohol co-use: substantially increased acetaldehyde exposure that results from oral alcohol metabolism combined with the high levels of acetaldehyde present in inhaled tobacco smoke.[55]

Acetaldehyde-Derived DNA Damage

Somatic genomic studies have also been informative regarding carcinogenic mechanisms mediated by acetaldehyde-derived DNA damage. Acetaldehyde, a reactive metabolite, can covalently bind to DNA, forming monoadducts, such as N^2-ethylidene-dG or 1,N^2-propano-deoxyguanosine, and DNA and protein crosslinks. These modifications trigger different repair pathways and differ in their mutagenic potential and effects on carcinogenesis.[82] Recurrent mutation patterns, or DNA mutational signatures, have been linked to exposure to alcohol[83] and DNA damage in stem cells.[84] Genetic polymorphisms that inhibit the function of ALDH2 appear to modulate exposure to carcinogens (including acetaldehyde) and their impact on the somatic genome of carriers.[85,86] For example, a study examined levels of acetaldehyde-derived DNA monoadducts by *ALDH2* genotype in peripheral blood samples collected from individuals who were admitted for treatment after diagnosis of AUD.[87] The level of

three acetaldehyde-derived DNA adducts was found to be substantially higher in blood samples from patients with AUD who were carriers of the *ALDH2* polymorphism than in samples from patients with AUD who were noncarriers, providing direct evidence that the *ALDH2* genotype affects the acetaldehyde-related genotoxic damage.

Additional insight is provided by studying patients with Fanconi anemia (FA), a rare genetic disorder characterized by impaired DNA repair. The FA repair pathway is the major mechanism counteracting the damage caused by endogenous and exogenous carcinogenic aldehydes (including acetaldehyde) by avoiding the resulting interstrand crosslinks from degenerating into double-stranded breaks.[84,88,89] As a consequence, patients with FA are at higher risk for developing certain types of cancers. For example, the incidence of head and neck squamous cell carcinoma in patients with FA is 500 times higher than in the general population.[90]
These observations further point to the carcinogenic role of acetaldehyde through its genotoxicity. They also support increased carcinogenicity due to a higher level of exposure with tobacco and alcohol co-use. Future studies of people with genetic variants can provide novel and important insights into causal mechanisms of carcinogenesis.

Further Mechanisms of Tobacco and Alcohol Carcinogenesis

Perturbation of Carcinogen Metabolism

Alcohol may interfere with the metabolic degradation of tobacco-related carcinogens. The most consistent evidence to support this hypothesis relates to the effects of ethanol on exposure to carcinogenic tobacco-specific nitrosamines that can be formed from tobacco products. Swann (1984)[91] demonstrated that alcohol inhibits the hepatic metabolism and clearance of N-Nitrosodimethylamine (NDMA). This inhibition occurs because ethanol competitively inhibits hepatic CYP2E1, the main hepatic enzyme responsible for the metabolism of NDMA. Consequently, higher amounts of NDMA reach extrahepatic tissues where the nitrosamine can be metabolically activated to reactive species with the potential to interact with cellular targets and disrupt their function.[91,92] Similarly, chronic alcohol consumption induces cytochrome P450 2A6 (CYP2A6), the enzyme primarily responsible for the metabolism of nicotine, leading to an increased rate of nicotine metabolism.[93] This effect in turn could lead to compensatory increases in tobacco use, thereby increasing exposure to carcinogens in tobacco smoke. One study found that CYP2A6 levels were elevated in liver biopsy specimens of individuals with histories of excessive alcohol consumption.[94] Other studies found that alcohol induced CYP2A6 in the U937 macrophage cell line exposed to alcohol[95] and in the mouse orthologue of human CYP2A6—CYP2A5—in mice exposed to alcohol.[96,97] These findings are consistent with the observation that the increased rate of nicotine metabolism in men undergoing treatment of AUD declined 4–7 weeks following cessation of alcohol use.[98]

Alcohol-Induced CYP2E1 and Activation of Carcinogens Present in Tobacco Smoke

Chronic alcohol consumption increases the rate of ethanol elimination by the induction of CYP2E1 activity.[99] This induction results in increased production of reactive oxygen species, augmenting those produced by tobacco use while also more efficiently converting procarcinogens present in tobacco smoke to active carcinogens. Ethanol intake has been shown to induce these carcinogen-activating enzyme systems in the liver, the esophagus, the lungs, and the intestine, which are all target organs for tobacco-related cancers.[100,101]

Effects of Alcohol on Retinol Metabolism and Other Metabolic Dysfunctions

Alcohol consumption can reduce hepatic retinol (vitamin A) levels, either by increasing its mobilization from the liver to extrahepatic regions or by increasing its degradation through the induction of the cytochrome P-450 2E1 (CYP2E1) metabolic systems.[102] Additionally, retinol and ethanol have similar metabolic pathways, and ethanol can act as a competitive inhibitor of vitamin A oxidation to bioactive retinoic acid involving ADHs and ALDHs.[103] Similarly, tobacco smoke interferes with retinoid metabolism and reduces its plasma retinol levels.[104] Retinol plays a fundamental role in maintaining normal cellular growth and differentiation mechanisms.[105] Furthermore, retinol has been shown to have anticarcinogenic properties that interfere with both the initiation and promotion of the carcinogenic process; therefore, reduced retinol levels can contribute to the disruption of retinol-mediated signaling, leading to uncontrolled cellular proliferation and carcinogenesis.[105]

Finally, AUD can lead to nutritional deficiencies.[102] These nutritional deficiencies include folate deficiency, a vitamin deficiency that can contribute to an inhibition of methyltransferases, enzymes that are important in gene regulation. Additionally, AUD may also result in zinc deficiency,[106] and low levels of this essential vitamin are associated with adverse health outcomes including impaired immune system function with a possible role in carcinogenesis.[107]

Inflammation and Oxidative Stress

Inflammation and induction of oxidative stress are two distinct processes that may play independent roles in carcinogenic processes.[108] In the context of tobacco- and alcohol-related mechanisms of interactions, the focus of most research has been on the ability of both substances to induce inflammation resulting in lipid peroxidation and production of reactive oxygen species.

Cigarette smoke contains substances that can induce inflammation resulting in increased cellular proliferation, activation of nuclear factor-kappa B (NF-kB), and tumor promotion.[9] The promotion of oxidative stress by the generation of reactive oxygen species and the depletion of endogenous antioxidants may account for some of the increased risk of hepatocellular carcinoma associated with smoking.[109]

Alcohol consumption has been shown to result in the induction of several hepatic metabolic changes resulting in oxidative stress.[110] The induction of CYP2E1 is a source of production of reactive oxidative species. Furthermore, depletion of the antioxidant glutathione in the setting of chronic alcohol exposure in people with AUD can also increase cellular oxidative stress.[111,112] Additionally, alcohol consumption has been associated with the upregulation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidases[113,114] and mitochondrial oxidative stress.[115] Consequently, simultaneous exposure to tobacco and alcohol is expected to concurrently promote the role of these processes in increasing the risk for developing liver cancer.[109]

People who smoke experience proinflammatory changes in their lungs,[34,116] and studies in laboratory animals demonstrate that anti-inflammatory agents can decrease tobacco carcinogen-induced lung tumorigenesis.[117] In addition, chronic obstructive pulmonary disease (COPD), especially emphysema, is an independent risk factor for lung cancer in individuals who smoke.[118] Because COPD causes inflammation in the airways, this association further implicates the role of inflammation in lung cancer.[118,119] The mechanisms associated with these results are

based in part on the fact that inflammatory processes result in lipid peroxidation and generation of reactive oxygen species which can damage DNA and result in mutations. Additionally, products from these processes can result in the induction of cyclooxygenases (COXs) which can lead to the production of prostaglandins that are thought to be important mediators in solid tumor carcinogenesis.[120]

Individuals with AUD have an increased risk of both the occurrence and severity of lung infections, including pneumonia, tuberculosis, respiratory syncytial virus, and acute respiratory distress syndrome.[121,122] These infections could lead to exacerbations of chronic inflammatory lung diseases, which increase the risk of lung cancer in people who smoke. Epidemiological evidence for associations between alcohol and lung cancer remains mixed, with some studies suggesting an increased risk of lung cancer associated with drinking alcohol[123] and others reporting inverse associations of alcohol consumption.[124,125] Further evidence concerning lung cancer and co-use of tobacco and alcohol is discussed in chapter 4.

The interaction of tobacco and alcohol in inducing inflammation and oxidative stress has also been suggested to play a role in increasing the risk of pancreatic cancer.[126] Oxidative DNA damage and lipid peroxidation-induced DNA adducts have been found in the pancreas in association with increased *KRAS* mutations in tissues from people with and without pancreatic cancer. These mutations have also been found to occur in both people who smoke and people who use alcohol.[31,127] However, further analyses have mixed results with only some studies reporting associations between alcohol consumption and pancreatic cancer,[23,128,129] mostly with higher levels of alcohol consumption.[23,130-132] Interpretation of these results is also complicated by the potential for confounding by smoking or exposure to secondhand smoke and by dietary variables related to inflammation. Nevertheless, higher levels of alcohol consumption appear likely to be associated with pancreatic cancer.[132]

Epigenetic Changes

Epigenetic changes, defined as heritable modifications to gene expression that are independent of changes to DNA sequences,[133] are associated with tobacco and nicotine exposure.[134] Such changes are most apparent in people who smoke cigarettes, and some may be reversible upon smoking cessation, raising the possibility of using patterns of methylation or other epigenetic changes as biomarkers to distinguish between current, former, and never smoking.[135] Additionally, the oxidative damage and gene promoter methylation processes induced by cigarette smoking are an integral part of cancer progression. Gene promoter methylation is a common epigenetic change, involving extensive methylation at the 5-position of cytosine (C) in CpG islands within the promoter region and often extending into exons of regulatory genes.[136,137] Generally, hypermethylation of CpG islands in promoter regions is associated with gene silencing, and hypomethylation is associated with gene activation. Both processes can be involved in a variety of functions including inactivation of tumor suppressor genes[136,137] and modulation of cellular sensitivity to genotoxic compounds, which occurs via influences on the activity of genes involved in the metabolism of xenobiotics (substances foreign to the body).[138-140] In lung cancer, more than 750 genes are inactivated by gene promoter hypermethylation, and additional genes are still being identified through genome-wide screening approaches.[141,142] Additionally, smoking leads to hypomethylation of the aryl hydrocarbon receptor repressor (*AhRR*) gene. This epigenetic modification has been suggested as an indicator

of long-term exposure to tobacco smoke based on the observation that hypomethylation correlates with current and former smoking status, cigarette consumption, cumulative smoking, smoking duration, and time since quitting smoking.[143] *AhRR* hypomethylation has also been associated with decreased lung function and increased respiratory and cardiovascular morbidity.[143] However, it remains unclear what proportion of these epigenetic changes directly influence carcinogenic processes.[136,137,142]

Epigenetic processes involving methylation may be partially dependent on one-carbon metabolism. One-carbon metabolism is a series of metabolic pathways, including methionine and folate cycles, that provide one-carbon or methyl groups for a variety of cellular functions.[144] Alcohol can interfere with one-carbon metabolism in several ways including by disruption of folate status and consequent inhibition of DNA methyltransferase,[145] thereby potentially generating aberrant DNA methylation patterns.[146] Additionally, alcohol consumption has been associated with variations in histone acetylation and histone methylation during carcinogenesis.[147] The hypermethylation of promoter genes with tumor suppressive characteristics has been observed in healthy tissue adjacent to tumors and oral premalignant lesions, suggesting that these epigenetic modifications are early events in oral carcinogenesis.[148,149] In this context, tobacco use has been associated with several epigenetic changes in oral carcinoma that have also been observed as a consequence of alcohol exposure. Specifically, hypermethylation of the *CDKN2a* (*p16*), *DAPK*, and *CDH1* genes; inactivation of *P15* by promoter hypermethylation; and alteration of methylation patterns of the repetitive element long interspersed nuclear element-1 (LINE-1) have all been observed for both exposures. These modifications are believed to play a role in processes regulating cancer progression.[147]

Effects of Smoking and Alcohol Cessation on Epigenetic Changes

Genome-wide methylation profiling studies have shown that smoking-induced epigenetic changes are dynamic and partially reversible. A study conducted by Guida and colleagues (2015)[150] showed that two distinct classes of CpG sites were affected by smoking cessation. Within decades after smoking cessation, methylation at some sites reverted to levels typical of women who have never smoked, while other sites remained differentially methylated, even more than 35 years after smoking cessation.[150] Another study identified both site-specific and regional changes in DNA methylation in women who smoked.[151] The study also found substantial reversibility of methylation changes following smoking cessation, although some specific genes remained differentially methylated up to 22 years after smoking cessation. Additional research is needed to determine the extent to which reversible changes in methylation contribute to reduced risk of cancer after smoking cessation.

Few studies have investigated the effects of alcohol cessation on alcohol-induced epigenetic modifications. A study conducted among men undergoing treatment for AUD examined levels of gene-specific methylation (*ALDH2* and methylenetetrahydrofolate reductase [*MTHFR*]) and global methylation in DNA from peripheral blood leukocytes. Compared with healthy control participants, participants with AUD had higher gene-specific methylation and lower global DNA methylation; no statistically significant change in these methylation patterns was observed 3 months after the participants with AUD quit drinking.[152] Additionally, a genome-wide

methylation analysis was conducted among men with AUD (n = 99) undergoing medically supervised withdrawal to identify methylation patterns associated with withdrawal from alcohol. This study identified a higher number of differentially methylated CpG sites in men with AUD than in control participants, a difference that decreased after 2 weeks of recovery from acute alcohol withdrawal.[153] More research is needed to determine how methylation and other epigenetic changes respond to changes in alcohol intake and whether these changes can be used as biomarkers of behavior or effect (see chapter 4).[154]

Tumor Promotion

In addition to mechanisms in which tobacco and alcohol share the ability to promote similar molecular steps associated with tumor initiation, tobacco and alcohol act independently as tumor promotors.[35,155] Tobacco use contributes to tumor promotion via influences on cell proliferation, cell cycle progression, and induction of nicotine.[155] Although the effects of alcohol have been studied less, its tumor promoting effects may involve effects on M2 macrophages[156] and histone deacetylation.[157]

Tobacco and alcohol may also interact to influence tumor growth in tobacco-initiated cancer, and alcohol may promote tumor growth in cancers initiated by exposure to tobacco smoke. This observation is based on studies performed *in vitro* and in animal models.[15,158] The tobacco-specific nitrosamines NNN and nicotine-derived nitrosamine ketone (NNK) are both categorized as Group I carcinogens (sufficient evidence for carcinogenicity in humans) by IARC,[13] and NNN and NNK are major contributors to tobacco smoke carcinogenicity.[15] A study conducted in rats found that ethanol caused an increase in the mean frequency and mean size of tumors induced by NNN or NNK in the rat esophagus, oral cavity, lung, and liver.[158] The study authors suggested that the ethanol-mediated promotion of NNN- and NNK-induced tumors may result from alcohol metabolism generating free radicals and increasing lipid peroxidation processes in the target tissue.[158] Many more mechanistic aspects of tumor promotion by co-use of tobacco and alcohol remain to be explored.

Genetic Susceptibility

Understanding how genetics influences cancer susceptibility in cancers related to tobacco and alcohol can help in understanding the mechanisms by which these environmental factors influence the carcinogenic process. The genome-wide association study (GWAS) approach has emerged as a common method for identifying genetic variants involved in cancer susceptibility, as well as related traits such as those that influence behaviors related to smoking and drinking (as described in chapter 6).[159] The GWAS approach assesses the common (>5%) genetic variation and low-frequency (1%–5%) genetic variation in the genome and thus identifies genetic variants without *a priori* evidence that a particular gene is involved.[160,161] Large GWAS have been conducted for smoking- and alcohol-related behaviors and cancer types related to tobacco and/or alcohol use, such as lung, head and neck, and bladder tumors.[162,163] As of 2026, the genes located near the genetic variants identified via agnostic GWAS include genes involved in smoking behaviors, alcohol metabolism, xenobiotic metabolism, and DNA repair,[163,164] making them very similar to the genes and pathways associated with the mechanistic

evidence discussed previously in this chapter. As such, the agnostic GWAS approach supports much of the mechanistic data presented. The following sections discuss the genetic variants identified by GWAS and other approaches and how the tobacco- and alcohol-related carcinogenic mechanisms may be influenced by the functions of these genes.

Lung Cancer

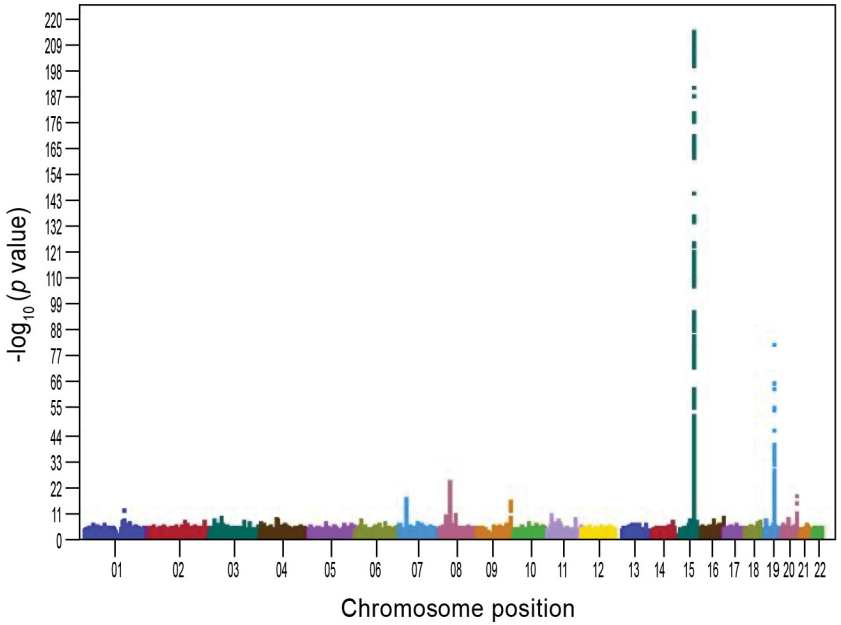
GWAS of lung cancer have identified loci involved in genetic susceptibility and identified genetic variants related to smoking behavior, DNA repair, telomerase activity, and immune response, as well as many variants where the target genes are unclear.[162] Further, loci and variants related to alcohol and lung cancer have also been identified, including some that are related to alcohol metabolism and acetaldehyde.[164]

Genetic Variants Related to Lung Cancer Risk and Smoking Behavior

One of the key insights stemming from genetic studies of lung cancer is that lung cancer genetic susceptibility tends to overlap with alleles associated with smoking behavior, as shown in Figure 5.3. First, key genetic variants identified from GWAS of lung cancer are the same as those independently identified from GWAS of smoking behaviors (e.g., in the genes *CHRNA5,* encoding the a5 nicotinic acetylcholine receptors (nAChR) subunit, and *CYP2A6,* encoding the enzyme that metabolizes nicotine); second, this genetic overlap extends to a polygenic risk model (i.e., many thousands of small effect susceptibility variants) estimated through sophisticated genetic analysis, such as genetic correlation based on linkage disequilibrium score regression.[165-172] This key finding is consistent with the idea that by influencing smoking behavior, such genetic variants modulate exposure to tobacco and, in turn, carcinogen exposure and lung cancer risk in susceptible individuals.[165,168] The finding is further supported by the observation that the association with these alleles is predominantly observed in individuals with a history of smoking,[165,168,173,174] and that there is more DNA damage, particularly mutations associated with tobacco exposure, in lung cancer tumors arising in genetically at-risk patients.[167] Gene and environment interactions involving tobacco use provide further evidence of the importance of preventing initiation of tobacco use to avoid development of lung cancer.

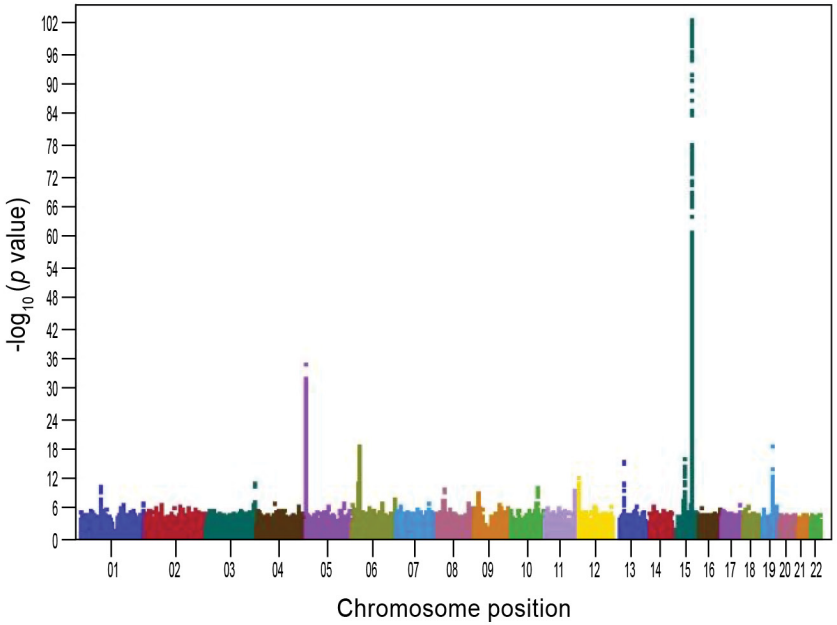
Figure 5.3 Manhattan Plots From Genome-Wide Association Studies of Cigarettes per Day and Lung Cancer in Participants of European Ancestry

Panel A: Cigarettes per Day



A: Genome-wide association study of cigarettes per day in European ancestry participants.[175]

Panel B: Lung Cancer



B: Genome-wide association study of lung cancer in European ancestry participants.[173]

Note. Each dot on the plot signifies a single nucleotide polymorphism (SNP). Genomic coordinates are displayed along the *x*-axis, and each color block represents a chromosome. The *y* axis is the negative logarithm of the association's *p* value for each SNP. The strongest associations have the smallest *p* values and their negative logarithms will be the greatest. Chromosomal region 15p25 encompasses *CHRNA5* and chromosomal region 19p encompasses *CYP2A6.*
Source: Plots created from data reported in Liu et al. 2019[175] and McKay et al. 2017.[173]

The study of how these alleles individually influence the carcinogenic process provides insight into tobacco-related behavior and carcinogenesis. For example, susceptibility alleles influence different aspects of smoking behaviors, including smoking initiation, smoking intensity, smoking cessation, or combinations of these behavioral aspects of smoking.[163,167,170,173,175] Together, this genetic variation can help account for some of the variation in tobacco-related behaviors observed in human populations. Some alleles display rather paradoxically with respect to addictive behavior. For example, variants in the *CYP2A6* gene region (which plays a major role in nicotine metabolism) are associated with higher cigarette consumption, and higher risk of lung cancer and COPD, but an increased likelihood of successful smoking cessation.[176-178] Other alleles also point to unexpected biological mechanisms. For example, the lung cancer susceptibility alleles near *CHRNA2* influence expression levels of this gene uniquely in the cerebellum, a brain area not typically linked with smoking behaviors.[173] It is worth noting that genetic variation in behaviors involving other forms of tobacco, such as the daily frequency of tobacco chewing, has also been documented.[179] These results suggest a complex set of genetic influences on behavior related to stages of nicotine dependence (see chapter 6).

Genetic Variants Related to Lung Cancer Risk That Are Unrelated to Smoking Behavior.

The GWAS approach has also identified many lung cancer susceptibility variants unrelated to smoking behavior.[162,173] These genes are implicated in several hallmarks of cancer,[180] with genetic variants identified near genes involved in DNA repair, telomere maintenance, xenobiotic metabolism, and immune response, as well as other variants where the functional target unclear.[162,173] Many of these alleles appear to be relevant to a particular histological subtype and smoking status. Examples of such loci include *TERT* (telomere reverse transcriptase, which helps maintain the end of telomeres),[181-183] *TP63* (tumor protein p63, which helps regulate cell activities including proliferation, cell maintenance, differentiation, cell adhesion, and apoptosis),[184,185] *ROS1* (receptor tyrosine kinase, which acts as a cell surface receptor for a variety of growth factors),[186] *NRG1* (neuregulin 1, an epidermal growth factor),[173] and *ATM* (a tumor suppressor gene).[187] These loci are generally related to lung adenocarcinomas and imply smoking independent mechanisms of action. Other loci, such as variants in *BRCA2*[185] and *CDKN2A* (tumor suppressor genes),[188] *CHEK1*[167] and *CHEK2* (genes for regulating cell cycle checkpoints),[185,189] *CYP1A1* (metabolism of xenobiotics),[167] and *IRF4* (regulatory transcription factor for immune cells)[190] appear particularly relevant to lung squamous cell carcinomas in people who use tobacco.

Elucidating the precise mechanisms by which these genetic variants confer susceptibility to lung cancer can help to better understand how tobacco influences the carcinogenic process and how genetic susceptibility may influence the legacy of tobacco-related damage in tissues in individuals who have quit tobacco use. Additionally, a better understanding of genetic susceptibility to tobacco-induced carcinogenesis could further inform efforts to identify how combined exposure to tobacco and alcohol influences carcinogenesis.

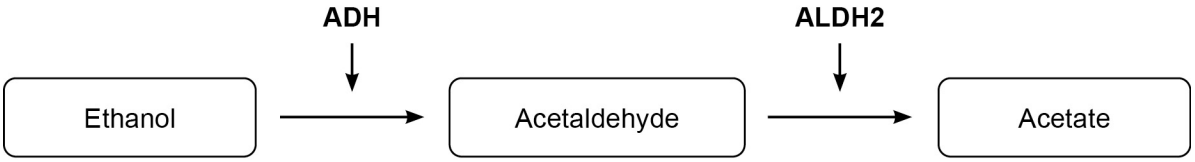
Aerodigestive Tract Cancers

Tobacco and alcohol are strong independent risk factors for many aerodigestive tract cancers, and substantial observational evidence supports a synergistic relationship between exposure to these two substances. As described in chapter 4, genetic variation in the alcohol metabolism gene *ALDH2*, specifically the ALDH2*2 polymorphism, reduces the function of the ALDH2 enzyme, leading to increased exposure to acetaldehyde in the oral cavity based on host and oral microbiome enzyme action. This increased exposure is associated with a higher risk of cancers of the head and neck and esophagus (squamous cell carcinoma).[22,66,191,192] Genetic variation in various alcohol metabolism genes may increase or decrease the rate at which acetaldehyde is metabolized.[192,193] Some of these variants lead to a relative increase in exposure to acetaldehyde in susceptible individuals, resulting in increased risk of cancers of the head and neck and esophagus.[66,191]

The most studied *ALDH2* genetic polymorphism is rs671, a missense variant that results in the loss of function of the ALDH2 protein and, as described in chapter 4, facial flushing. Individuals with *ALDH2* who drink alcohol also have increased cancer risk.[191,194,195] As discussed in this chapter's section, "Acetaldehyde-Derived DNA Damage," research on *ALDH2* polymorphisms provides strong evidence for the role of aldehydes in alcohol-related head and neck cancers.[67,194,195] Notably, the loss-of-function allele is also associated with a marked increase in the risk of developing esophageal squamous cell cancers, particularly in heterozygote carriers who consume alcohol despite experiencing facial flushing and other unpleasant effects.[66,191,196] Genomic-based studies of the somatic environment of squamous cell esophageal tumors suggest a higher prevalence of somatic mutations associated with alcohol exposure (COSMIC signature SBS16) in tumors occurring in carriers than noncarriers.[85,86] These findings are consistent with the hypothesis that the increased risk of esophageal cancer in *ALDH2* heterozygote allele carriers is mediated by acetaldehyde. Together, these observations support the need for alcohol-related cancer prevention efforts for people who are *ALDH2* variant carriers.[66]

In addition to the *ALDH2* gene, studies have identified genetic variants in *ADH* genes[197,198] related to alcohol consumption and risk of aerodigestive cancers, including evidence for interactions between these genetic variants and alcohol consumption (see Figure 5.4).[196] The *ADH1B* and *ADH1C* variants accelerate the metabolism of alcohol to acetaldehyde, elevating acetaldehyde levels in people with these variants who drink alcohol and increasing their risk of cancer.[199] Relatedly, studies in Japan and China have shown that, among people who consume alcohol, those who have less active *ADH* alleles are at greater risk for head and neck cancers,[200] but in Western countries, where different polymorphisms are more common, mixed results are observed in relation to ADH activity and cancer risk.[201] Alleles of *ADH* and *ALDH2* that are present interact to modulate the levels of acetaldehyde associated with alcohol consumption, influencing both cancer risk and the effects of alcohol flushing on consumption.[66,198,200] Although research has identified the alleles and mechanisms through which the *ADH* variants influence cancer risk, the expression of *ADH* in tissues such as those of the lower gastrointestinal tract and subcutaneous adipose tissue, regions not considered primary for alcohol absorption and metabolism, as well as cardiovascular and lung tissues, suggests that the role of these *ADH* variants is not yet fully understood.[63]

Figure 5.4 Simplified Diagram of Ethanol Metabolism



Note. Alcohol dehydrogenase (ADH) is the predominant enzyme catalyzing the conversion of ethanol to acetaldehyde, and aldehyde dehydrogenase 2 (ALDH2) metabolizes acetaldehyde into acetate. Alleles of *ADH* and *ALDH2* interact to influence the concentration of acetaldehyde in the aerodigestive tract, increasing or decreasing cancer risk.
Source: Seitz and Becker 2007.[73]

There is overlap between lung cancer susceptibility alleles and aerodigestive tract cancers, particularly with regard to genetic variants located near DNA repair genes such as *RAD52*, *MDM4*, *BRCA2*, and *CHEK2*,[202-206] suggesting the importance of the modulation of DNA repair in the context of the overlapping etiology of exposure to tobacco and alcohol. Mendelian randomization studies also support the causal relationship between tobacco and alcohol and head and neck cancers.[207] One study reported that the susceptibility variants located near the *CHRNA5/3/b4* gene cluster associated with smoking propensity were related to aerodigestive tract cancers in women but not in men, although sex differences in the effects of these variants on smoking behavior were not observed.[208] Additional research is needed to understand the biological basis for these findings.

Interactions Between Nicotine and Alcohol

Nicotine, the major alkaloid in tobacco, is responsible for the addictive properties of tobacco products.[9] By driving smoking behavior, nicotine is responsible for the exposure to tobacco-derived carcinogens in people who use tobacco products. The mechanisms by which nicotine induces addiction to tobacco products are covered in chapter 6. The Surgeon General has concluded that "there is insufficient data to conclude that nicotine causes or contributes to cancer in humans," but notes that "nicotine is bioactive for a number of carcinogenic mechanisms in experimental systems."[1]

Nicotine tolerance is higher in people with AUDs than in people without AUDs.[209,210] People who smoke and have a history of heavy alcohol consumption score higher on measurements of nicotine reinforcement than those who smoke but have no history of AUD.[211] Individuals who smoke have a higher prevalence of alcohol use than those who have never smoked (see chapter 2). Cross-tolerance (when repeated administration of one substance induces decreased response to another substance) between nicotine and ethanol consumption has been widely observed in animal models and also occurs in humans, although the evidence for such cross-tolerance in humans is somewhat indirect.[212-214] This topic is also discussed in chapters 6 and 7. Further research is needed to understand the neurophysiological and genetic basis of these patterns of cross-tolerance and associated patterns of co-use.

These associations of increased tobacco and alcohol consumption with co-use could be related to both environmental and genetic factors. However, metabolic cross-tolerance likely plays a role because nicotine and ethanol share similar CYP-mediated metabolic pathways—CYP2E1 and CYP2B1, respectively—and both substances can induce these enzymes.[215] This induction can lead to more rapid elimination of ethanol and nicotine, which can result in increased consumption. Additionally, alcohol can induce CYP2A6, and high levels of CYP2A6 are associated with nicotine exposure in animal studies.[98] nAChRs are also expressed in tissues outside of the brain,[216] so it is plausible that the genetic effects of the susceptibility alleles with nAChRs—and their response to nicotine—may influence other tissues. Metabolic cross-tolerance and resulting behavior patterns can make quitting smoking or reducing alcohol consumption more challenging (see chapter 7).[217,218]

Potential Interactions of Electronic Cigarettes and Alcohol

E-cigarettes are battery-powered devices that generate aerosol by heating a liquid (e-liquid) that typically contains nicotine, dissolved in a mixture of propylene glycol and/or vegetable glycerin, and flavorings. The health effects of e-cigarettes are influenced by many factors, including constituents of the e-liquid, physical characteristics of the device, and chemical reactions related to the heating characteristics of the device.[219] Assessing the health effects of e-cigarettes is complicated by the diversity of e-cigarette devices and e-liquids, their relatively recent entry into the tobacco marketplace, and their frequent use with other tobacco products.[220] E-cigarette aerosol contains markedly lower levels of many of the harmful constituents and carcinogens present in cigarette smoke.[221] However, heating the main ingredients and the flavorings often used in the e-liquids results in exposure to many different carbonyl compounds including aldehydes, such as acetaldehyde and formaldehyde,[221] that are genotoxic and mutagenic.[222-224] Although a detailed review of the potential short- and long-term toxicity of e-cigarettes is beyond the scope of this monograph, potential health effects have been documented for respiratory diseases[225-231] and cardiovascular disease.[232,233]

A joint statement published by the American Association for Cancer Research (AACR) and the American Society of Clinical Oncology (ASCO) in 2022 described evidence linking e-cigarette use to cancer.[234] The statement summarized research linking e-cigarette use to DNA damage, inflammation, and cell replication:

> A growing body of evidence points toward a biologically plausible role for ENDS [e-cigarette] use in contributing to human carcinogenesis, based on the presence of carcinogens in ENDS aerosols; metabolites of carcinogens in human urine samples; inflammation markers in human lung swabs and blood samples; and cell culture and mouse experiment exhibiting DNA damage and inflammation.

Given these concerns, there is an urgent need for additional epidemiologic and mechanistic studies.

At present, there is also limited information about patterns of co-use of e-cigarettes and alcohol. However, like other tobacco products, most e-cigarettes contain nicotine. Alcohol consumption is likely to influence the use of e-cigarettes, and vice versa, given the potential increased tolerance and cross-tolerance between nicotine and ethanol, among other factors.

Additionally, the evidence suggesting a possible role for e-cigarettes to contribute to cancer—and the fact that both e-cigarette and alcohol exposure induce inflammatory processes and result in exposure to genotoxic compounds such as acetaldehyde—highlights the importance of studying the combined health effects of alcohol and e-cigarette use on cancer risk. Moreover, patterns of use and co-use of tobacco, nicotine, and alcohol products continue to evolve very rapidly. As other novel tobacco products (e.g., heated tobacco products, nicotine pouches) enter the marketplace, it will be important to study the influence of alcohol on patterns of their use across the life course and the combined health effects of co-use of tobacco products and alcohol, including the etiology, prevention, and treatment of cancer.

Chapter Summary

Tobacco smoke contains more than 7,000 chemicals, including at least 80 known carcinogens. Ethanol, the substance responsible for alcohol's addictive nature, is also the primary carcinogen present in alcoholic beverages. Alcohol may influence tobacco-related carcinogenesis by increasing exposure to carcinogens through its effects on membranes and metabolism, inflammation and oxidative stress, epigenetic changes, and promotion of tumor growth. Tobacco and alcohol co-use results in overlapping and distinct exposures to carcinogens, as well as effects on metabolic processes resulting in increased use. In addition, individual vulnerability to tobacco and alcohol carcinogens and the effects of nicotine and ethanol metabolites are influenced by genetic variation at multiple sites.

Research suggests that the interactive effects of combined tobacco and alcohol use on carcinogenesis of the aerodigestive system is largely associated with increased exposure to acetaldehyde, a potent carcinogen and the primary metabolite of ethanol. Other mechanisms include the alcohol-driven increase in the absorption of tobacco-related carcinogens, chronic inflammation, and the increased production of reactive oxygen species that both risk behaviors induce. Additionally, ethanol acts to increase membrane permeability in the aerodigestive system and acts as a solvent for tobacco-associated carcinogens, thus increasing their absorption. Together these processes increase uptake of acetaldehyde associated with both tobacco and alcohol use, as well as thousands of additional chemical compounds associated with tobacco use, including many carcinogens.

The oral microbiome plays an important role in metabolizing ethanol into acetaldehyde, and salivary acetaldehyde levels increase substantially with concurrent tobacco smoking and alcohol consumption. The interactive effects of tobacco and alcohol on aerodigestive tract cancers may be attributed to substantially increased acetaldehyde exposure from oral alcohol metabolism, combined with the high levels of acetaldehyde in tobacco smoke.

Further evidence for the causal role of acetaldehyde in alcohol-induced cancer is derived from studies of people with genetic polymorphisms that reduce the activity of aldehyde dehydrogenase 2 (ALDH2). Ethanol is converted to acetaldehyde in the oral cavity, but reduced ALDH2 activity in heterozygous individuals results in greater persistence of acetaldehyde. ALDH2-deficient individuals experience facial flushing and other adverse symptoms if they consume alcohol and are also at much higher risk of esophageal squamous cell carcinoma (among people completely lacking ALDH2 activity, the flushing response is so unpleasant that alcohol consumption is very low or absent, and consequently, the risk of esophageal cancer is much lower). Similarly, studies

of patients with Fanconi anemia, a rare genetic disorder characterized by an inability to repair damage to DNA caused by endogenous and exogenous carcinogenic aldehydes (e.g., acetaldehyde) have been informative. People with Fanconi anemia are at extremely high risk for developing certain types of cancer, including head and neck squamous cell cancers. These findings highlight that studies of people with rare genetic variants can provide important insight into carcinogenic mechanisms associated with tobacco and alcohol use.

Alcohol induces CYP2E1 which acts largely in the liver to metabolize many compounds, including the conversion of ethanol to acetaldehyde and then acetate. Additionally, CYP2E1 activates some carcinogens and produces reactive oxygen species, further pathways to carcinogenesis. Some studies have also examined potential interactions between tobacco and alcohol exposure and inflammation, retinol pathways, and other metabolic perturbations. Additionally, multiple studies in rodents report that ethanol can accelerate progression of NNN and NNK-induced tumors. The use of different types of tobacco products results in exposure to an overlapping, but not identical, mix of carcinogens as compared to those associated with cigarette smoking. How these different mixes of carcinogens interact with ethanol exposure is not well understood. For example, little is known about the potential interaction of e-cigarettes with alcohol on carcinogenesis. As new tobacco products enter the marketplace, it will be important to assess these interactions.

Tobacco cessation dramatically reduces risk of tobacco-related cancers but less is known about the effects of alcohol cessation or reduction and cancer risk. A 2024 IARC review found that the evidence is sufficient to indicate that alcohol use cessation or reduction reduces the risk of cancer of the oral cavity and esophagus, but the evidence is limited or inadequate for alcohol-caused cancer at other sites. Mechanistic studies suggest that these reductions in risk are related to reduced exposure to acetaldehyde, reduction in DNA damage, and reduced intestinal permeability, however more research is needed.

Research Needs

The study of the interactions of tobacco and alcohol on carcinogenesis from a mechanistic point of view is limited, especially when compared to the amount of similar research on tobacco and alcohol individually. This is a significant research gap because co-use of tobacco and alcohol is very common. Relatively few mechanistic or genetic studies have assessed detailed physiological and metabolic variation in the combined effects of tobacco and alcohol use over time, and even fewer have examined these processes after cessation of tobacco and/or alcohol use. The relative paucity of mechanistic and genetic studies in this context highlights the need to better understand the combined effects of tobacco and alcohol on carcinogenesis. Research is needed to assess how changes in tobacco and alcohol use, separately and especially together, influence inflammation, other carcinogenic processes, and cancer risk.

More research is needed on the combined effects of tobacco and alcohol on inflammation and oxidative stress. Both tobacco and alcohol use lead to production of reactive oxygen species and inflammation in multiple tissues.[235-237] Less research has addressed the effect of heavy alcohol consumption on the response to proinflammatory agents in people who smoke and people who do not smoke, and results have been contradictory. More research on the combined effects of tobacco and alcohol exposure on oxidative stress and inflammation could clarify these mixed results.[238]

Evidence is limited or inadequate for understanding the effects of reduced alcohol consumption at multiple sites, with and without tobacco co-use. More research is needed to develop studies that have the power to observe a reduced risk, examine effects of reduction in alcohol consumption, and assess patterns of consumption over the life course, including consumption alongside tobacco use. Such studies might also provide opportunities to explore specific biologic mechanisms influencing cancer risk after different durations of cessation or reduced consumption of alcohol (and cessation or reduction alongside co-use with tobacco).[38]

More research is needed on mechanistic aspects of combined tobacco and alcohol use and carcinogenesis covering diverse tobacco products and diverse populations. For example, relatively little research has examined the potential mechanisms of cancer risk among people who co-use e-cigarettes and alcohol.

More research is also needed on interactions between tobacco and alcohol use at low and moderate alcohol consumption levels. Most studies have focused on people who engage in risky drinking or those with alcohol use disorder. Few studies have examined the physiological effects of cessation or reduction in drinking at lower levels of consumption with or without concurrent tobacco use. Epidemiological studies show that even low levels of drinking increase the risk of cancer at multiple sites including female breast and colorectal cancer[24] and can increase the risk of liver disease.[239] Results from human and animal studies also demonstrate a variety of physiological effects resulting from lower levels of alcohol consumption. However, most of the mechanistic studies on the effects of alcohol cessation include individuals with alcohol use disorder who are entering treatment programs and measure variables related to the overall health of the participants. Research involving this population can be challenging because of frequent loss to follow-up and the prevalence of multiple substance use disorders. More research is needed to explore the interactions of alcohol consumption at lower levels than for people with alcohol use disorder with diverse aspects of tobacco initiation, continued use, cessation, and relapse.

Advances in study design are needed to improve studies of co-use of tobacco and alcohol. There is a need for research using study designs that specifically address the effects of tobacco and alcohol exposures combined. These may include time-course designs, analysis of the effects after cessation of the exposures, dose-response designs to investigate effects considering lower levels of exposures, and investigation of effects in different populations. Research is also needed to specifically understand the mechanisms by which the initiation and/or promotion of cancer is influenced by smoking cessation and drinking cessation or reduction. Future research could evaluate how cessation or reduced use of tobacco and alcohol may result in metabolic changes, inflammation change, repair of DNA damage, and changes in risk of cancer.

Conclusions

1. **Tobacco and alcohol are known human carcinogens and increase the risk of cancer via both shared and independent pathways.** These pathways include exposure to dozens of carcinogens in tobacco, many associated with combustion, the presence of acetaldehyde in both tobacco smoke and as a metabolic product of ethanol, and physiological effects of ethanol on membrane permeability, hormonal and inflammatory pathways, and nutrient status.
2. **Combined carcinogenic and physiological effects of tobacco and alcohol provide a mechanistic basis for the synergistic increases in risk for aerodigestive tract cancers observed for co-use of tobacco and alcohol.** These mechanistic factors include exposure to acetaldehyde from both tobacco and alcohol; the composition of the oral microbiome, which results in increased acetaldehyde production; and increased permeability of aerodigestive tract membranes resulting in elevated uptake of tobacco carcinogens produced by tobacco combustion. Genetic variants of alcohol dehydrogenase (ADH) and aldehyde dehydrogenase 2 (ALDH2) interact to influence cancer risk. These enzymatic variants influence acetaldehyde levels in the aerodigestive tract by influencing metabolism of ethanol to acetaldehyde (ADH) and acetaldehyde to acetate (ALDH2).
3. **Individuals with the ALDH2 variant (rs671) have increased risk of cancer from alcohol consumption.** Worldwide, an estimated 540 million people (primarily people of East Asian descent) are heterozygous or homozygous for mutations that reduce ALDH2 function, resulting in elevated levels of acetaldehyde, alcohol flushing responses, and increased risk of cancer. Efforts to address alcohol use in these populations are important for cancer control.
4. **Tobacco cessation reduces the risk of many types of cancer, and alcohol reduction or cessation reduces the risk of head and neck cancer.** There is a dearth of studies on whether alcohol reduction or cessation reduces the risk of other types of cancer. The mechanisms of carcinogenesis of tobacco and alcohol co-use are best understood for acetaldehyde-related pathways, but less well understood for hormonal- and inflammation-related pathways. Nevertheless, this mechanistic evidence strongly supports efforts to address tobacco and alcohol use and co-use to improve cancer control.

Appendix 5A Literature Search Strategy

The literature cited and summarized in this chapter was identified from searches conducted in 2023 using Google Scholar and PubMed, with a focus on peer-reviewed publications. Various combinations of the following keywords were used for the searches: tobacco, cigarette smoking, alcohol, alcohol consumption, ethanol, mechanisms, interactions, genetic susceptibility, genome-wide association studies, germline, addiction, metabolism, and carcinogenesis. The publications resulting from these searches were reviewed for relevant citations. Publications that cited relevant articles were also evaluated for relevance. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

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Chapter 6
Shared Physiological and Genetic Factors Contributing to Tobacco and Alcohol Co-Use

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Chapter 6
Shared Physiological and Genetic Factors Contributing to Tobacco and Alcohol Co-Use

Introduction

Significant progress has been made toward identifying biological factors contributing to tobacco and alcohol use through large-scale human genetic studies of dependence and substance use disorders.[1,2] Substance use disorders are complex diseases in which thousands of genetic variants contribute to an individual's overall risk. Indeed, thousands of genetic loci have been identified as contributing to tobacco and alcohol use. Studies have found that some of these genetic variants are associated either with tobacco or alcohol use, and some are associated with the use of both substances.[1-7]

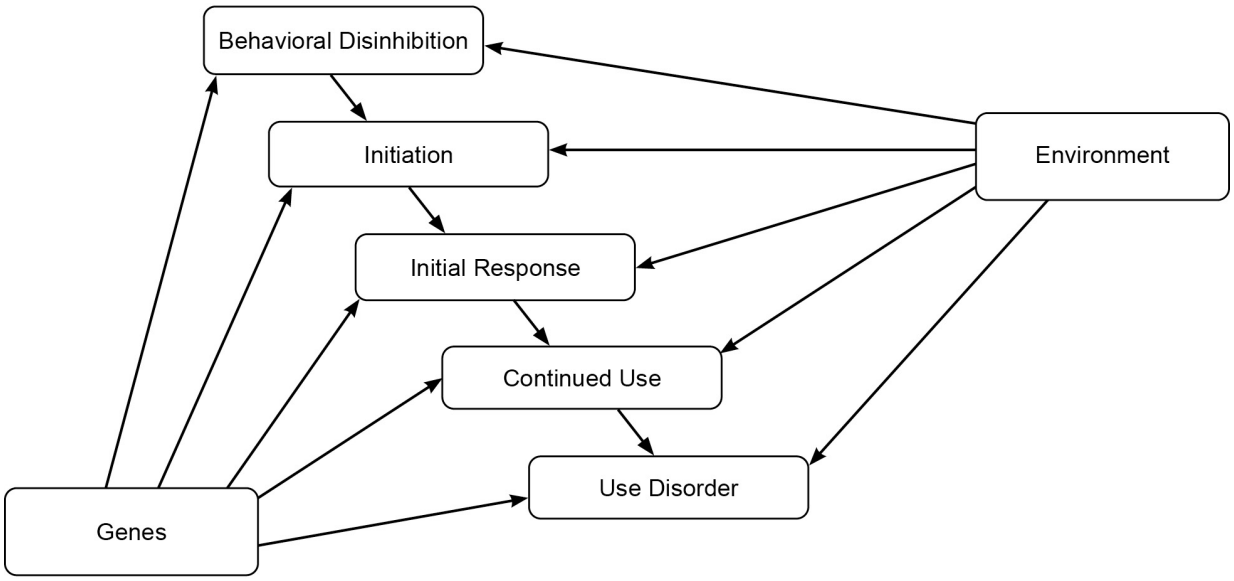
Throughout the 1990s and early 2000s, the prevailing methodology in genetic studies utilized family-based approaches when conducting whole genome studies or focused on candidate genes based on known physiological or biochemical pathways.[8-10] A summary of those approaches applied to smoking behaviors was reviewed in the 2010 Surgeon General's report, *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease*, and National Cancer Institute (NCI) Monograph 20, *Phenotypes and Endophenotypes: Foundations for Genetic Studies of Nicotine Use and Dependence*.[8,10] The development of low-cost technologies for examining hundreds of thousands of variants across the genome has expanded researchers' ability to conduct large-scale agnostic association studies.[11-14] Agnostic association studies consider all regions of the genome without prior assumptions about the relevant area of the genome or type of genetic element of interest.[15] These genome-wide association studies (GWAS) leverage very large sample sizes to achieve the statistical power needed to detect small-effect genetic variants and have been applied to both tobacco and alcohol.[1,2,16]

This chapter discusses the physiological, cellular, molecular, and genetic factors contributing to tobacco and alcohol use. Overall, genetic studies have demonstrated that tobacco and alcohol use disorders, as well as other substance use disorders and psychiatric disorders, have both shared and unique genetic risk factors. The chapter describes overlapping genetics and corresponding neurophysiology contributing to the co-use of tobacco and alcohol, summarizing the biological pathways affected by nicotine and ethanol and describing findings from neuroimaging studies and genetics studies, particularly results from large GWAS. When discussing biological mechanisms, this chapter focuses on nicotine and ethanol. Nicotine and ethanol are the main drivers of the addictive processes for tobacco and alcohol, respectively. However, one important contrast between tobacco and alcohol is that ethanol is almost entirely responsible for alcohol-related carcinogenesis, while tobacco smoke is a complex mixture of thousands of compounds, including many which are toxic and carcinogenic.[17-19] There is insufficient evidence to conclude that nicotine plays a direct role in carcinogenesis, although it does interact with some relevant pathways.[20]

The initiation of tobacco and alcohol use and the progression from initiation and use to tobacco or alcohol use disorders is a process with multiple stages, as shown in Figure 6.1. Both environmental and genetic factors contribute to the development of dependence and disease, and different genetic factors contribute to tobacco and alcohol use patterns across different phases of use and development of use disorders.[19,21] Further, biological and behavioral studies of tobacco and alcohol use address a wide range of phenotypes at different stages of the developmental trajectory of use. The combined influence of these factors on tobacco use and dependence are also highlighted in NCI Monograph 20.[8] Notably, individuals may quit using tobacco or alcohol or make unsuccessful attempts at cessation at any point and thus move forward or backward along this trajectory. Therefore, this diagram is a simplification in that it is unidirectional and does not include additional feedback loops.

NCI Monograph 20[8] emphasizes the importance of identifying endophenotypes (i.e., phenotypes related to the underlying mechanisms of disease), including those that are related to the adoption or maintenance of nicotine dependence. Further research identifying the phenotypes and endophenotypes underlying use and co-use of both tobacco and alcohol could refine the conceptual model presented in Figure 6.1 to incorporate factors in addition to behavioral disinhibition that influence initiation and continued use of tobacco and alcohol, including various patterns of use. Efforts are being made to characterize endophenotypes related to alcohol use,[22] and some research has directly addressed endophenotypes of co-use and polysubstance use.[23] Large longitudinal neuroimaging studies such as the Adolescent Brain Cognitive Development (ABCD) and Healthy Brain and Child Development (HBCD) studies[24,25] could facilitate identification of new phenotypes and endophenotypes related to co-use.

Figure 6.1 Developmental Trajectory of Tobacco and Alcohol Use Disorders Is a Process With Multiple Stages



Source: Adapted from Bierut 2011.[26]

Tobacco use and alcohol use disorder may develop concurrently with first or early use, or use disorders may manifest more gradually or not at all.[19] Alcohol use often occurs without characteristic features of dependence or use disorder; however, even alcohol use within the 2020–2025 U.S. Dietary Guidelines (≤2 drinks/day for men and ≤1 drink/day for women)[27] is associated with increased cancer risk.[28] Attention to alcohol use at all levels of consumption is important from the perspective of cancer prevention.[27] For example, a low level of alcohol consumption (1 drink per day) is associated with an increased risk for breast cancer among women (see chapter 4). Moreover, as discussed in chapter 4, there is a dose-response relationship between alcohol consumption and many cancers, and increased synergistic risk of head and neck cancer and squamous cell esophageal cancer associated with tobacco and alcohol use. Alcohol use disorders often involve higher levels of consumption and thus greater cancer risk.

As discussed in chapter 2, more than half of those who report current (past-year or past-month) tobacco use also report using alcohol. It should also be noted that, among U.S. adults in 2022, daily or near-daily cannabis use in the past month (5.3%) was approximately as prevalent as daily or near-daily alcohol use in the past month (4.4%).[29] Although discussion of co-use of cannabis with alcohol and/or tobacco is beyond the scope of this monograph, research to understand the interplay among tobacco, alcohol, and cannabis is increasingly important, given trends in cannabis use.

Most of the research as of 2026 has been conducted in adults, but a handful of longitudinal studies investigating precursors and the onset of alcohol use in childhood and adolescence are underway. The largest (N = ~11,500) and most comprehensive of these is the ongoing ABCD study,[25,30] which investigates neurodevelopment among youth ages 10–20 years.[31,32] Another key study is the HBCD study, a cohort designed to examine *in utero* effects as well as subsequent environmental exposures in infancy and beyond on ontogeny of brain development and ultimately substance use.[24]

In most past studies, sex has not been directly examined to identify whether distinct genetic variants play differential roles in men and women. Where appropriate, this chapter incorporates discussion about whether sex differences have been examined or identified. Similarly, the field of human genetics has only recently begun a strong effort to recruit a diverse sample of participants at a large scale. For example, the *All of Us* Research Program, with recruitment launched by the National Institutes of Health (NIH) in 2018, is making such an effort.[33] Where available, data for previously underrepresented populations are presented in this chapter, describing similarities and differences between results from the initial European ancestry-based studies. Finally, one of the major challenges in the field is moving from the identification of genetic variants to understanding their biological function; this is especially difficult because most of the variants do not fall in genes with known or predicted impacts on molecular function. Research has expanded to begin to incorporate data from other "omics" resources, such as studying gene expression in the brain, epigenetic features, proteomics, and metabolomics.

Nicotine, Ethanol, and Biological Pathways in the Brain

Tobacco and alcohol are frequently used together, and high comorbidity exists between nicotine dependence and alcohol use disorder (see chapter 2).[34-36] Furthermore, both substances target many of the same neuronal pathways in the brain, sometimes in a manner that accentuates the effect of the other substance and in other cases reduces it.

General Overview of Neurophysiology

As research reveals more about the brain, characteristics of its functions and complexity become even more challenging to describe and understand. For example, the brain used to be described primarily in terms of different functions of certain morphologically defined regions, such as the striatum, hippocampus, or prefrontal cortex. With advances in the technologies and computational analysis approaches used, it has become clear that each brain region is part of a network, comprised of complex connections of subregions, and that even within very small subregions, different cell types (e.g., neurons, interneurons, astrocyte, microglia) are found that confer unique neurophysiological signals.[37,38] Such signals may be different between immediately adjacent cells.[39] Likewise, many psychoactive substances affect dopamine release and binding in key brain regions, and there has been a significant focus on understanding how substances disrupt the dopaminergic pathway and thereby lead to dependence.[40] In addition to dopamine, substance use also influences the release of norepinephrine, serotonin, glutamate, and acetylcholine. Furthermore, differences between individuals due to both genetic variation and environment, including past experiences or precursors, can confer different baseline levels of neurotransmitters and/or differential responses to substances according to functional changes of brain receptors.

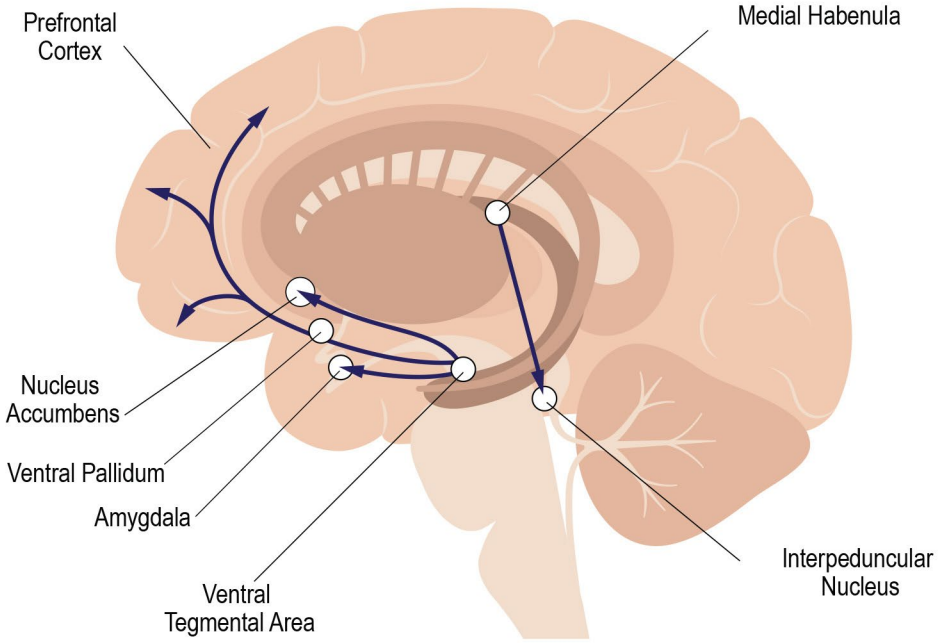
The neurophysiological mechanisms underlying substance use disorders have been described and studied in the context of three functional domains or mechanisms: (1) incentive salience, (2) negative emotional states, and (3) executive function; each of these is mediated by a distinct neurobiological circuit: (1) basal ganglia, (2) extended amygdala, and (3) prefrontal cortex, respectively.[41] This triadic neurobehavioral model was primarily developed to explain the maintenance of use and return to use after abstinence in adults who have already progressed to substance use disorder. In addition, evidence also supports its relevance in explaining the onset and escalation of substance use earlier in life,[42] demonstrating its importance across different stages of substance use and addiction. Furthermore, the triadic neurobehavioral model appears to apply to most or all substance use disorders.[43]

Addictive substances—including nicotine and ethanol—affect these three major neurobiological circuits implicated in dependence and addiction. The neurobiological circuit implicated in incentive salience includes the nucleus accumbens, dorsal striatum, globus pallidus, thalamus, and ventral tegmental area (Figure 6.2).[41] This system is a major pathway for dopamine, involving modulation of reward, motivation, and desire, and contains the cell bodies of neurons that, when stimulated, cause dopaminergic release in multiple other structures, including the nucleus accumbens.[19] Nicotine, ethanol, and other addictive substances increase dopamine concentrations in the nucleus accumbens, leading to an urge or motivational drive to continue consuming these substances. In contrast, the extended amygdala recruits the brain's stress

systems, which drive feelings of negative affect during withdrawal states.[41] The prefrontal cortex, which is involved in higher-order executive functioning, is dysregulated among individuals with substance use disorders and contributes to disadvantageous decision-making, impaired self-awareness, and dysregulated self-control and self-regulation.[19,41,44,45] Intense feelings of craving and anticipation are further associated with the prefrontal cortex and intervals between use.[44]

Importantly, addictive substances affect some of the same neurobiological circuits in overlapping but not identical ways, and are often used together or in a short time window. The effects of co-use may be additive, disinhibitory, or have other types of interactions. For example, additive effects of co-use of nicotine and ethanol have been demonstrated in rats.[46] The long-term effects on brain function from concurrent co-use of two or more addictive substances can be stronger than the effects of using either substance separately.[47] Accordingly, polysubstance use or co-use of multiple addictive substances has been considered a severity indicator in substance use disorders and is linked to poorer health outcomes and higher rates of return to use after treatment.[47-50] Finally, in addition to shared effects of addictive substances on the brain, there are also substance-specific effects. For example, a pathway between the medial habenula and interpeduncular nucleus (IPN) contains numerous neuronal nicotinic receptors. These receptors have been widely studied for their role in the effects of nicotine and ethanol on behavior and the development of tobacco and alcohol use disorders,[10,51,52] and were highlighted in the 2010 Surgeon General's report.[10] Figure 6.2 illustrates the key brain regions affected by nicotine and ethanol use.

Figure 6.2 Key Brain Regions Affected by Nicotine and Ethanol Use



Source: Adapted from Koob & Volkow 2016.[41]

Dysregulation of Dopaminergic Signaling With Nicotine and Ethanol Use

All addiction processes affect the function of the brain. This includes substance use disorders and other mental health conditions that involve compulsive behaviors, such as gambling disorders, compulsive sexual behavior, eating disorders, and internet gaming addictions.[53] At its core, any kind of dependence or addiction involves cyclical changes in the production, release, and binding of dopamine in the brain.[41] Increases in addictive behaviors occur as people seek out the specific substance or experience that leads to a transient increase in dopamine release, and an increasing amount of the experience or substance may be needed to elicit this response, a process referred to as "tolerance."[19] The fact that cross-tolerance (when repeated administration of one substance induces a decreased response to another substance) is associated with nicotine and ethanol co-use as well as use of other substances together with tobacco and/or alcohol[54,55] has important implications for interventions addressing initiation and cessation in the context of co-use (see chapters 5 and 7). Furthermore, chronic use of addictive substances shifts baseline dopamine levels downward in animal models and some human studies, such that addiction is sometimes described as a hypodopaminergic state.[56] This results in increased craving and use of drugs of choice and reduced salience of other nondrug stimuli. Morel and colleagues (2019)[57] provide a comprehensive overview of the important role of dopaminergic neurons in modulating nicotine and ethanol reinforcement.

Most physiological studies of addictive behaviors have been conducted using rodent models.[58] Rodents and humans share similar brain structures, neuronal pathways, and genetic homology across the genome.[41,59] However, it is important to recognize the limitations of animal models, as certain aspects of these models may not translate to humans. For example, most rodent studies employ a single inbred strain of mouse or rat for research,[60] and these genetically identical animals are assessed for behavioral measures believed to capture different aspects of the developmental trajectory depicted in Figure 6.1. In some studies, animals self-administer the substance, but methods for administration can vary according to access (limited, unlimited, choice between substance and water) and route of administration (oral or injection). When examining measures for tolerance or withdrawal, nicotine or ethanol are often injected by the investigator to ensure a consistent dose. Reviews of rodent behavioral paradigms provide additional detail.[61-63] Mouse and rat models have different strengths and weaknesses that have evolved over time. As genetic tools developed in the mouse have become standard in the rat, many investigators have transitioned to using the rat for studies of the neuroscience of psychiatric disorders because the processes fundamental to addictive behavior in humans are more similar to those in the rat than in the mouse.[64]

Many human studies indicate that adolescent exposure to alcohol and/or tobacco leads to an increased risk of developing tobacco and alcohol use disorder in adulthood, as well as other substance use disorders.[65-67] However, because co-use of tobacco and alcohol products in adolescence is common, it is difficult to disentangle in longitudinal human studies which substance accounts for what proportion of the subsequent development of a substance use disorder.[65] Chronic consumption of alcohol in rats may increase the rate of biotransformation of nicotine and its metabolites, and this may differ by sex.[68] Nicotine and ethanol also appear to have interactive influences on neuronal activity in rodents.[46,69] These interactions may underly the observation that nicotine and ethanol have not only antagonistic but also synergistic effects in the brain.[52]

Neuronal nicotinic acetylcholine receptors (nAChRs) play a key role in mediating the interactions of nicotine and ethanol.[6,36,70] nAChRs are widely expressed throughout the brain and are present on many of the key neuronal projections from the ventral tegmental area depicted in Figure 6.2. Nicotine directly binds to nAChRs in the brain, while alcohol modulates these receptors both directly, by interacting with them, and indirectly, by influencing other signaling pathways that affect nAChR function.[71,72] Nicotine stimulates the cholinergic system and the nAChRs specifically in the ventral tegmental area.[36,73] This stimulates the release of dopamine into the nucleus accumbens and augments activity in the prefrontal cortex. Therefore, nicotine can act to prime dopamine neurons in the ventral tegmental area such that alcohol becomes more reinforcing. Animal studies have also shown that consumption of ethanol alone leads to increases in dopamine release in the nucleus accumbens via activation of nAChRs in the ventral tegmental area, which contributes to the reinforcing properties of alcohol.[71,73,74] Thus, co-use of nicotine and ethanol produces greater reinforcement through synergistic dopamine effects, which then causes continued self-administration of both substances.[52,73]

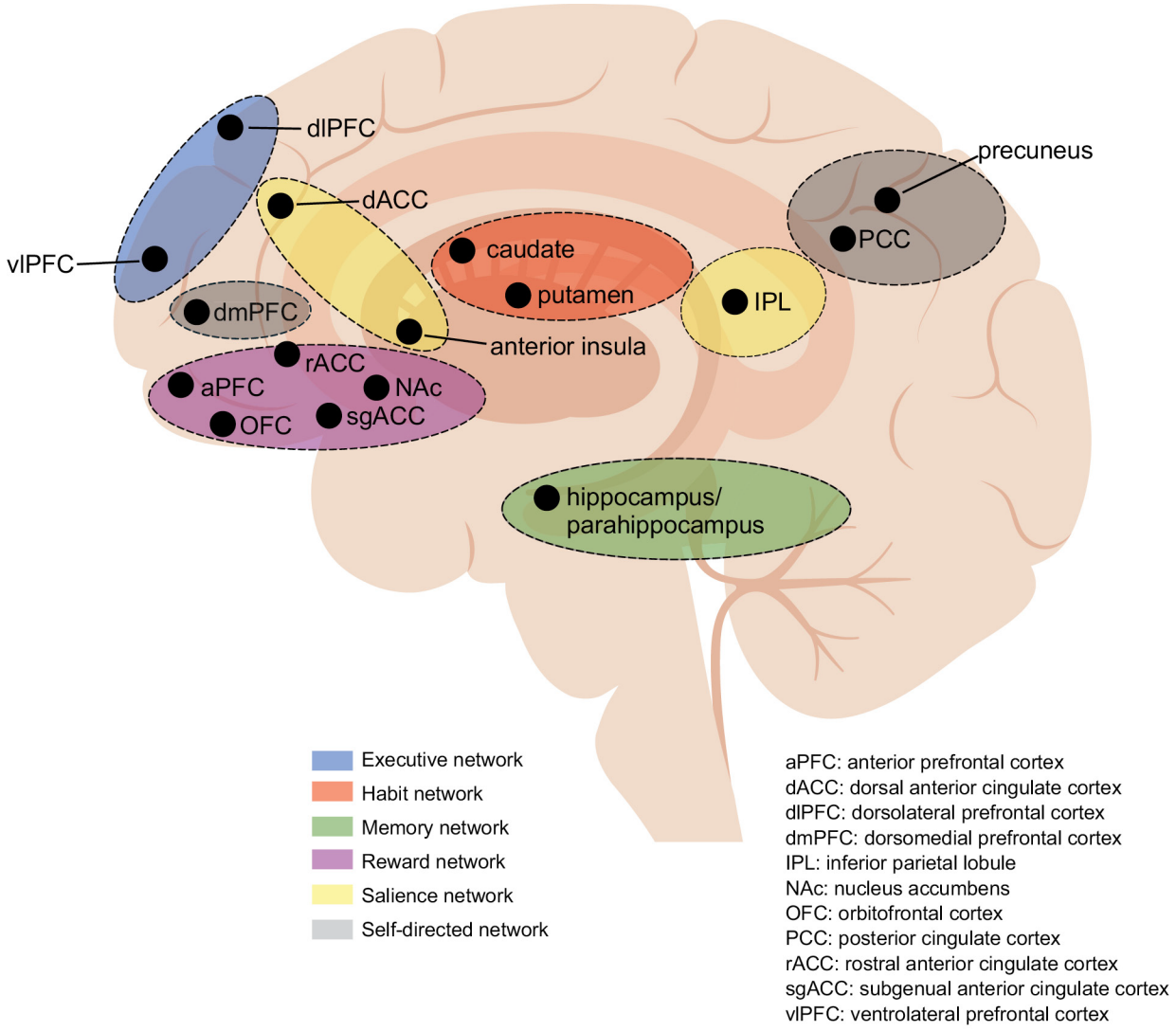
The interaction effects of nicotine and ethanol highlight that responses to these substances can be enhanced with co-use and will change over time with continued use. Continued use and co-use may lead to the development of tolerance, especially if use increases. nAChRs are also highly expressed in the medial habenula and IPN (Figure 6.2), two regions believed to be involved in abstinence and withdrawal, but this circuit is not as well-characterized as the mesolimbic pathway.[75,76]

Neuroimaging Studies

A longstanding question in the neurobiology of addiction has been whether early baseline differences in brain structure and function contribute to the development of substance use disorders and/or whether substance use leads to lasting changes in brain structure, brain function, or both. As of 2026, however, it has not been feasible to unpack substance-use-related differences in brain structure or function, because most participants in past neuroimaging studies have been adults.[77] With time, answers to this important question may come from 2, large-scale longitudinal neuroimaging studies ongoing as of 2026: the HBCD study, which began enrolling pregnant participants in 2023,[78,79] and the ABCD study which began enrolling 9–10 year olds in 2016.[31,32] Additionally, although preclinical neuroimaging studies in animal models have given insight into the direct consequences of chronic drug use,[80] the complex interactions between genetic vulnerability factors and chronic substance use in humans cannot be sufficiently modeled in animals. Neuroimaging studies in humans have attempted to incorporate underlying genetic risk factors into studies of brain structure and function; until recently these have been limited mainly to candidate genes and small sample sizes.[81] These studies share similar design constraints as the original candidate gene studies. Nevertheless, neuroimaging techniques have been used to study the three major neurocircuits implicated in substance use disorder: the (1) basal ganglia, (2) extended amygdala, and (3) prefrontal cortex.[41] The most comprehensive review (published in 2018) of the neuroimaging literature in adults with substance use disorders has extended this prior theoretical framework by identifying 6 large-scale brain networks consistently implicated in addiction (Figure 6.3).[82]

- The reward network includes subcortical and cortical brain regions activated during the appraisal of subjective value (anterior prefrontal cortex, rostral anterior cingulate, nucleus accumbens, orbitofrontal cortex, and subgenual anterior cingulate).
- The striatal habit network underlies learning of automatized behavior (caudate and putamen).
- The salience network is involved in (re)directing attentional resources toward salient stimuli (dorsal anterior cingulate, anterior insula, and inferior parietal lobule).
- The executive network supports the selection of possible behavioral responses (dorsolateral prefrontal cortex and ventrolateral prefrontal cortex).
- The self-directed network is activated during self-directed/referential cognitive processes (precuneus, posterior cingulate cortex, and dorsomedial prefrontal cortex).
- The memory network is involved in flexible, multi-cue learning and memory (hippocampus and parahippocampus).

Figure 6.3 Large-Scale Brain Networks Implicated in Addiction Through Neuroimaging



Source: Adapted from Zilverstand et al. 2018.[82]

Importantly, Zilverstand and colleagues (2018)[82] noted that multiple studies have revealed the importance of emotional and cognitive factors beyond the feelings of anticipation, intoxication, and withdrawal described in the classic triadic model of addiction. Overall, the evidence from human neuroimaging studies supports the role of impaired salience attribution, negative emotional states, and dysregulated executive function underlying substance use disorders, but also points to the importance of changed appraisal of subjective value, altered self-directed processing, and changes in flexible learning and memory.[82] These higher-order cognitive processes could not be studied in the animal models from which the classic triadic model of addiction was mostly derived; hence, their importance was only revealed through the human neuroimaging literature. Furthermore, Zilverstand and colleagues' review of the neuroimaging literature highlighted that, for individuals with substance use disorders, both the classically studied and the newly identified brain networks show increased recruitment during

substance-related processing. However, they are not activated to the same degree during non-substance-related processing, when individuals with substance use disorders engage in non-drug-related processing.[82] Finally, the neuroimaging literature points to nuanced sex differences in the neurobiological mechanisms underlying substance use disorders,[83] though this remains an area of need for further research.

It is important to recognize that the networks described in the human neuroimaging literature are slightly different than the pathways described in the literature based on animal models.[84] This occurs because neuroimaging studies use a whole-brain approach to measure neuronal activation in humans *in vivo*, rather than the localized measurements in animals following experimental procedures. Although the same or similar brain regions are examined, they are therefore sometimes described at different levels of detail, depending on the tools used to measure substance effects, resulting in somewhat different descriptions of the role of specific regions. There may also be differences between species in relevant pathways and networks.

Several studies have used neuroimaging to examine changes in the brain associated with nicotine or ethanol, but very few studies have considered both substances together.[82] In 2014, Liu and colleagues[85] used functional magnetic resonance imaging (fMRI) to examine brain regions activated in response to cues for smoking and alcohol use. The study used a framework of three networks (reward, salience, and cognitive control). Their data supported an interconnection between networks that activated together in response to both smoking and alcohol cues. Specifically, they found that the posterior cingulate cortex and right posterior insula were associated with alcohol behavioral traits (loss of behavioral control) and responded to both smoking and alcohol cues. These two regions were classified as part of the salience network in this study,[85] whereas Zilverstand and colleagues placed the posterior singular cortex in the self-directed network and the insula in the salience network.[82] However, there were also regions that showed differences in response to alcohol taste cues versus tobacco visual use cues.

Multiple studies have also examined volumes of various brain regions associated with cigarette smoking and alcohol use.[86-88] A 2020 mega-analysis (meta-analysis of pooled data from multiple existing datasets) revealed different results for individuals with alcohol dependence and smoking compared with nondependent controls.[87] Smaller volumes in the hippocampus, thalamus, putamen, and amygdala were detected among individuals with alcohol dependence. However, compared with nonsmoking control participants, larger volumes in multiple regions, including the hippocampus and right nucleus accumbens, were associated with smoking. It was unclear whether these differences were the result of substance exposure and continued use, or whether individuals with early differences in brain structure were at increased risk for the development of dependence and substance use disorders.

Two developments have opened the neuroimaging studies to more powerful analyses: (1) large scale datasets with tens of thousands of subjects and (2) Mendelian randomization, that can use genetic variation to help determine whether an exposure is causally related to an endpoint (see chapter 5). A 2022 Mendelian randomization study addressed this predisposition or consequence question by using subcortical brain volume data from approximately 37,000 participants in combination with tobacco and alcohol phenotypic and genotypic data from the UK Biobank study.[89] Alcohol dependence was inversely associated with volume of the amygdala and hippocampus; similarly, cigarettes per day was inversely associated with

hippocampal volume. The study authors did not perform a combined analysis to examine comorbid effects. The results strongly suggest that differences in brain volume are the consequence of use, rather than a predisposing factor. As genetically informative neuroimaging datasets continue to emerge, further knowledge will be gained of the predisposing features in the brain that increase tobacco and alcohol use and the consequences of substance use.

More research is moving beyond the addiction cycle to try to understand different trajectories leading to addiction and the functional domains associated with substance use. For example, detailed phenotyping of a sample of 454 adults in the United States, 186 with current alcohol use disorder, identified 3 neurofunctional domains—incentive salience, negative emotionality, and executive function—that distinguished between participants with and without alcohol use disorder.[90] Drossel and colleagues (2023)[91] characterized neurobehavioral subtypes based on extensive questionnaires for 173 people with past substance use disorders and 420 controls. Among people with a past substance use disorder, latent profile analysis identified three subtypes involving higher approach-related behavior, lower executive function, or higher negative emotionality. Participants in these three groups had distinct patterns of functional connectivity between brain networks, as measured with fMRI, that were associated with return to substance use. These results suggest substantial heterogeneity in these three major mechanisms related to addiction. Additionally, these results point towards the existence of stable trait-like factors related to dependence and addiction.

Shared Genetic Factors That Contribute to Tobacco and Alcohol Behaviors

This section is divided into three parts: (1) background information about methodology for the study of genetics of complex traits, (2) a description of the challenges of combining data from studies that used different measures of tobacco and alcohol related phenotypes, and (3) a summary of findings from large GWAS. As discussed in the introduction of this chapter, tobacco and alcohol use involve a complex developmental trajectory and diverse phenotypes beyond simply “use” or “substance use disorder.” This section emphasizes that there are multiple behaviors associated with tobacco and alcohol use and better understanding and measurement of these behaviors in genetic studies could strengthen understanding of these complex traits. In many studies, researchers have focused on tobacco or alcohol behaviors individually, although in most cases the participants were assessed for multiple substances, and many used both tobacco and alcohol (see Appendix 6A). Technological advancements and the development of innovative analytical approaches have bolstered the ability to perform integrative analyses that ask more nuanced questions about shared genetic features relevant to both tobacco and alcohol across the lifespan. In humans, most studies of the genetics of tobacco use have examined cigarette smoking; however, genetic studies in animal models have also examined the effects of nicotine specifically.[8] With changes in the tobacco marketplace and the increasing use of e-cigarettes and other novel tobacco products (see chapter 2), current and future genetic studies may consider addressing tobacco use other than cigarette smoking.

How Genetic Studies Are Conducted

As mentioned above, considerable evidence from genetic studies has demonstrated that tobacco and alcohol use disorders, as well as other substance use disorders and psychiatric disorders, have both shared and unique genetic risk factors.[1-7] Geneticists use twin and family study designs to estimate the proportion of variance in a trait that is due to genetic factors and the proportion that is due to environmental factors.[92-94] Because monozygotic (identical) twins share 100% of their genes, while dizygotic (fraternal) twins share on average only 50%, heritability can be estimated by comparing the similarity of an observed trait between monozygotic and dizygotic twins. Heritability is a statistical concept that estimates how well differences in genetic factors within a population account for differences in a particular trait (phenotype). Heritability can range from zero to one, where values close to zero indicate that the trait is due mostly to environmental factors in the population studied. Higher heritability estimates suggest a larger proportion of variation in the trait within that population is due to genetic factors, which can include both additive and multiplicative effects between genes.[92-94] The remaining phenotypic variance due to environmental factors can be parsed into those shared between siblings reared together and nonshared environmental factors unique to each person.

Heritability is dependent on the population in which it is studied, and heritability can change over time with different environments as well as with changes in the genetic makeup of a population, for example through genetic admixture.[92-94] Twin and family studies are usually underpowered to detect sex-dependent differences in heritability but can provide evidence of potential differences.[95]

It is also important to distinguish heritability from other similar concepts.[92-94]

- Heritability does not indicate the proportion of phenotypic variability determined by genetics for any particular person. For example, a heritability estimate of 50% for the trait "number of cigarettes smoked per day," indicates that approximately half of the variability in that population for this trait is due to genetic differences in that population at that time.
- Heritability estimates do not provide any information about the specific genetic or environmental factors contributing to a trait. Heritable traits are not the same as familial traits. Familial traits are traits shared within a family. They may or may not include a genetic component.
- Heritability estimates are not intended to indicate that a trait cannot be changed. Genes are not destiny. For instance, although the risk of developing certain chronic conditions like type 2 diabetes has a genetic component, behavioral factors such as diet and physical activity play a significant role in disease development. A person with a genetic predisposition to diabetes can significantly reduce their risk through healthy behaviors, demonstrating that genetic risk can, in many cases, be mitigated by environmental factors.

Heritability is used by geneticists as an important first step to confirm that a trait is determined in part by genetic factors, and it is also used to determine how much shared genetic variance exists between traits.

Decades of research demonstrate that, across populations, an approximately equal split between genetic and environmental factors contributes to risk for smoking and alcohol use.[10,96] A 2003 meta-analysis of twin studies from the United States, Australia, and Finland provided heritability estimates and shared and non-shared environmental risk factors for smoking initiation and cigarette smoking for men and women.[97] For both sexes and both phenotypes, heritability estimates centered around 50%,[97] as is the case for most substance use behaviors.[98] A similar meta-analysis of alcohol use disorders from 12 twin and 5 adoption studies revealed a heritability estimate of 49% for alcohol use disorder, with no differences due to sex.[96] Furthermore, two studies modeling rate of progression to alcohol use disorder suggested that environmental factors played a larger role in initiation of alcohol use, while genetic factors contributed more toward continued use and increased use.[99,100] Some of the underlying environmental variables were associated with family circumstances that may have genetic influences, as well. Cohort studies addressing the ontogeny of substance use could help clarify the relative importance of genetic and environmental factors for initiation and use.[79,101] Notably, environmental factors, such as social influences of parental or peer smoking behavior and attitudes, also appear to play a larger role in smoking initiation than genetic factors.[102,103]

Twin studies examining shared genetics of substance use disorder generally and for specific substances, including tobacco and alcohol, suggest a complex mix of genetic and environmental influences that may differ for different substances.[104] Delineating the relative contributions of each is a substantial methodological challenge, especially as many past studies suggest that other factors, such as disinhibition, are shared across many substance use disorders and other comorbid psychiatric diseases. This is discussed in detail in NCI Monograph 20.[8]

As reviewed by Goldman and colleagues (2005),[105] initial studies investigating the genetic contributions to alcohol and tobacco use were conducted either as family-based linkage analyses or as case-control association studies of unrelated individuals.[10,106] Linkage studies examine genetic markers spread across the entire genome in families to identify regions of the genome shared by family members affected by a trait of interest, called quantitative trait loci. This approach had notable successes, for example identifying the genetic basis of single-gene disorders, such as cystic fibrosis and Huntington's disease.[107] Genetic loci for some substance use behaviors were identified through this approach, including identification and confirmation of alcohol dehydrogenase (*ADH*) and aldehyde dehydrogenase (*ALDH2*) genes as loci affecting alcohol use behaviors (see chapter 5). However, in general, linkage studies failed to identify strong loci linked to tobacco and other substances.[105] Rather, behavioral traits are largely influenced by the small effects of thousands of genetic factors in combination with environmental influences.[105]

An alternate approach was common until the early 2000s, whereby case-control studies compared unrelated individuals with a specific characteristic, trait of interest, or disease (cases) with unrelated individuals without that characteristic (controls).[10] Investigators would then use these studies to compare the frequency of hypothesized candidate genetic variants in the cases and controls. For studies of substance use disorders, these "candidate gene" studies often focus

on genes expressed in neuronal pathways known to be involved in dependence and addiction, such as serotonin receptor and dopamine transporter, among others.[108] However, these studies had numerous limitations, including being underpowered to detect small effect sizes,[109] not always controlling for population stratification (a situation in which cases and controls are derived from genetically distinct underlying populations with different frequencies of specific genetic variants),[110] and often not thoroughly considering the impact of multiple testing on statistical significance.[108,111-115] Reflecting these limitations, results from these initial studies largely failed to replicate in later GWAS studies that appropriately considered potential population stratification and multiple comparisons.[108,111-114]

In the early 2000s, high-throughput genotyping technologies emerged that facilitated assessment of many thousands of variants across the genome, leading to the advent of GWAS.[116-118] This moved the field away from family-based linkage studies toward case-control association studies with sufficiently large sample sizes to detect small effects of single nucleotide polymorphisms (SNPs), the most commonly assessed genetic variant. Although GWAS provide benefits over linkage studies and have now yielded thousands of genetic variants associated with tobacco and alcohol use,[1,2] many GWAS to-date have had other limitations, including limited phenotypic assessments, challenges with controlling for population stratification, and samples collected primarily from participants of European ancestry.[110,116,118] Efforts toward more detailed phenotypic assessments and increased collection of samples with individuals from diverse ancestry have begun to address these limitations.[116] In addition, initial studies (linkage, candidate gene, and GWAS) typically focused on either smoking or alcohol phenotypes individually.[119] As additional studies have examined co-use, the field has begun to learn more about shared molecular genetic factors.

Challenges in Conducting Phenotype Assessments

There are several challenges associated with assessing substance use phenotypes for genetic, physiologic, and behavior studies. Many large-scale studies of tobacco and alcohol use are meta-analyses of samples collected from existing studies in which the phenotypic measures often do not align perfectly (see Table 6.1 and Appendix 6A). For example, the assessment protocols used often vary across the studies included in a meta-analysis, so a significant amount of time must be devoted to harmonizing the phenotype before results can be pooled. Resources with recommended measures for cohort and genetic studies (e.g., the PhenX Toolkit) are available and useful for incorporating in various types of studies. Further, previous research is limited by lack of ancestral diversity that limits the generalizability of genetic findings and their relationship to phenotypic diversity and the ability to implement precision medicine across all populations.[120]

In addition, as discussed previously, findings from earlier genetic studies have not been replicated in larger studies. GWAS efforts are often subject to data sharing policies that require scientific data to be available for use by the larger research community.[121,122] Such data sharing requirements are vital to ensure reproducibility and rigor of analyses performed using the data. Despite progress in standardizing data, expanding biobanks, and promoting data sharing, challenges such as inconsistent measures and lack of diverse representation still affect the overall impact and applicability of genetic research.

Measurement challenges also contribute to important limitations in large-scale genetic studies of substance use. In general, measures for smoking have involved variables describing initiation and lifetime patterns of use, such as age of initiation, number of cigarettes smoked per day, and smoking cessation (see chapter 2). Each of these variables may be defined in different ways, depending on choices made in the underlying study and evolving evidence for the validity and reliability of different instruments. Large-scale studies typically do not include diagnostic measures of tobacco use disorder.[13] Furthermore, specific diagnostic criteria for substance use disorder from the Diagnostic and Statistical Manual of Mental Disorders (DSM) continue to evolve.[123,124] Other measures of tobacco use are also used and many studies have measured nicotine dependence with the Fagerstrom Test for Nicotine Dependence (FTND).[125,126]

For alcohol behaviors, many of the initial large-scale studies were limited to queries measuring the number of alcohol-containing drinks consumed per week.[127] Some studies have begun to disentangle similar and different genetic results for consumption measures compared to measures of an alcohol use disorder, such as the Alcohol Use Disorders Identification Test (AUDIT),[128] developed by the World Health Organization.[129] A 2023 cross-ancestry meta-analysis that measured alcohol consumption and alcohol use disorder in the same participants and focused on those who reported current drinking found that the genetic correlation between alcohol use disorder and alcohol consumption is quite high in both European (0.86–0.92) and African (0.98–1.00) ancestry groups.[130] This reflects a very large proportion of variance that is likely due to pleiotropy (one or more genes influencing two traits) and confirms that there are multiple loci relevant both to alcohol consumption and alcohol use disorder. However, Kember and colleagues[130] also identified a set of variants related to alcohol use disorder, independent of level of alcohol consumption; such variants are of great interest in better understanding the transition to alcohol use disorder and its harmful consequences.

In studies focused on alcohol and other substances, DSM diagnoses of substance use disorder have been examined.[128] In addition, the inclusion of phenotypes that are not defined as disorders, such as presence or absence of alcohol consumption, increases the sample size available for GWAS analysis, thereby increasing power to detect genes associated with alcohol-related behavior.[131] Most initial studies of the genetics of tobacco and alcohol use examined results individually for smoking and alcohol consumption, followed by a simple comparison of overlapping genes.[132] It has also been common for investigators to analyze data for other substances and/or psychiatric disorders in combination with tobacco and alcohol.[119] In 2018, a new method for multi-trait analysis of GWAS (MTAG) was developed to enable joint analysis of multiple traits, which boosts statistical power for detecting associations with each trait.[133] MTAG can be applied to GWAS summary statistics without access to individual-level data for multiple traits, and the summary statistics do not need to come from independent discovery samples. For each SNP, trait-specific effects are estimated in a computationally efficient manner.[133] Examples of application of this tool for tobacco and alcohol phenotypes are presented later in the chapter.

Another challenge associated with assessment of tobacco and alcohol use is that overall study design can affect results obtained. Study design includes decisions about participant inclusion criteria such as age, sex, ancestry, measures of socioeconomic status, and others that may or may

not be clearly described. For tobacco and alcohol use, it can also be important to define thresholds of exposure. Initiation and experimentation as behaviors themselves may be more closely related to environmental variables such as access to these substances or social context, but the family setting and individual risk-taking also involve traits influenced by genetics and genotype by environment interactions.[134] Not everyone who tries tobacco or alcohol progresses to regular smoking or drinking. To address the idea that a person with very limited experience with a substance may not be considered as having initiated use, some studies define a threshold of use, such as having smoked at least 100 cigarettes for an adult participant to be considered a person who has ever smoked.[135,136] Changes in availability and use of specific tobacco products are another important point to consider when studying smoking behaviors. Although cigarette smoking initiation has decreased substantially, the introduction of e-cigarettes in the mid-2000s has led to substantial uptake among adolescents. Since 2014, e-cigarettes have been the most used tobacco product among adolescents, and adolescent e-cigarette use remains a major public health problem.[137,138] As of 2026, there is no standard for a minimum threshold of e-cigarettes or alcohol exposure for classifying participants as having ever used e-cigarettes or alcohol. Such a standard will be helpful, because having taken a puff of an e-cigarette or a single sip of alcohol does not adequately classify an individual as having initiated use. Furthermore, there is a lack of research on whether a combined threshold might be useful when examining both tobacco and alcohol use and the genetic correlates of nondaily tobacco and alcohol use.

Main Results From Genetic Studies

This section presents findings from genetic studies that emerged during the early 2000s through 2025. At the individual gene level, early studies identified associations with genes involved in the metabolism of nicotine and ethanol and genes associated with known targets of nicotine, nAChRs, in the brain. Subsequent large-scale studies expanded the number of variants and genes associated with smoking and alcohol use to the thousands, so interpreting the results has become much more challenging. Researchers developed methods to determine whether genes fall within specific physiological pathways, cell types, or other shared biological features to better understand the mechanistic pathways associated with specific genes. Although initial analytical approaches were primarily focused on a single phenotype, the development of multi-trait analysis has proven effective at increasing the number of variants associated with tobacco and alcohol use. As samples including individuals from more diverse ancestries have continued to grow, it has become clear that similar genetic pathways contribute to risk for tobacco and alcohol use disorders across populations.

Drug Metabolism and Neuronal Response Genes

Initial genetic results for both nicotine and ethanol have highlighted the impact of individual differences in drug metabolism on use and use disorders. The first genes identified that influence alcohol use, albeit somewhat indirectly, were the *ADH* and *ALDH2* genes,[139] which encode the proteins that are the main enzymes that metabolize ethanol and its primary metabolite, acetaldehyde (see chapters 4 and 5). Similarly, variants within genes coding for proteins that metabolize nicotine (cytochrome P450 and *CYP* genes) have been reproducibly associated with smoking behaviors.[140,141] Another highly replicated association with smoking involves nAChRs (*CHRN* genes).[142] This region includes the top genome-wide significant associated genes for

smoking behaviors[26,136] and also lung cancer (see chapter 5)[26,136] and has effectively become a positive control for human genetic studies related to nicotine phenotypes. These initial studies were described in both the 2010 Surgeon General's report and NCI Monograph 20.[8,10] nAChRs are the main site of action for nicotine in the brain, indicating that these genetic findings align well with the neurophysiology of nicotine's action.

Further characterization of the *CHRNA5/CHRNA3/CHRNB4* gene region revealed multiple independent genetic signals within this cluster, perhaps implicating functional variants in nearby genes as well.[143] However, the primary genetic variant, a single nucleotide polymorphism, rs16969968, corresponds to an amino acid change in the *CHRNA5,* a gene coding region that has been demonstrated to confer differential functional responses in the nAChRs.[144] Other genetic signals in the region are also associated with differential gene expression.[145] Evidence for an association between *CHRN* genes and alcohol behaviors specifically is equivocal as the large-scale human GWAS have not replicated findings from candidate gene studies, which have found associations between various subunit genes and alcohol behaviors.[146-149] Regardless, findings of the association have informed the use of varenicline, a high-affinity partial agonist at α4β2 nAChRs, as one of the current treatments for smoking.[150]

Some studies have reported associations between variation at SNP rs16969968 in the *CHRNA5* gene and a variety of differences in brain structure using neuroimaging approaches.[151] Many of these appear as baseline differences, independent of whether someone smokes, and there has been some evidence for differential effects of smoking dependent on genotype, along with evidence for differential cue reactivity by sex.[151] However, these studies had several design limitations (small sample size and differences in age, location, cohort, possible population stratification, or other environmental confounders), so it will be important to replicate the studies in larger samples before drawing firm conclusions. Genetic associations in humans between different *CHRN* genes and alcohol behaviors have been difficult to disentangle from the associations with smoking behaviors. A possible explanation for these difficulties is the high levels of co-use of these substances (see chapters 2 and 3).[6] New analyses or studies of people who use cigarettes or alcohol exclusively could help address this challenge.

Genome-Wide Association Studies

Appendix 6A presents a summary of selected large GWAS for tobacco, alcohol, tobacco and alcohol combined, tobacco and alcohol with other substances, and imaging studies published since 2018, with information about the studies' samples and main results. The search strategy for identifying relevant large GWAS is summarized in Appendix 6B. Four publications focusing specifically on tobacco, alcohol, and combined effects results are summarized in this section and in Table 6.1. The first publication discussed in this section presented results from the most powerful study conducted as of 2026, GWAS and Sequencing Consortium of Alcohol and Nicotine use 2 (GSCAN2), which included approximately 3.4 million individuals from 60 cohorts representing people with biological ancestors from African, American, East Asian, and European populations, with around 21% of the total sample derived from non-European samples.[2] This publication reported genetic loci associated with individual smoking and alcohol

phenotypes,[2] whereas the second publication conducted a multi-trait analysis for a combined addiction risk factor including four substances: alcohol, tobacco, opioids, and cannabis.[4]
Two other publications applied MTAG to conduct joint analyses of genetically correlated traits. One of these studies focused on smoking and alcohol use phenotypes only,[11] while the other included smoking, alcohol, opioids, and cannabis.[14] All four studies have begun to converge on loci shared across substances.

Table 6.1 Key Genome-Wide Association Studies of Tobacco and Alcohol

Study	Phenotype(s)	Sample[a]	Selected results
Cheng et al. 2023[11]	Alcohol and smoking joint analysis	Million Veteran Program • *N* = 318,694 European American subjects from Million Veteran Program database	• Joint analysis of alcohol use measures and smoking measures using MTAG ▪ 21 loci for smoking initiation and 17 for smoking cessation when combined with alcohol use disorder (compared to 16 and 8, for single-trait smoking initiation or cessation, respectively) ▪ 10 shared loci for alcohol use disorder and smoking status, including *SIX3*, *NCAM1*, and near *DRD2* • MTAG of smoking behaviors with alcohol consumption did not enhance discovery
Hatoum et al. 2023[4]	Alcohol Cancer Tobacco Opioid Cannabis General addiction risk factor	• *n* = 1,025,550 European American subjects and *n* = 92,630 African American subjects	• Disaggregated general and substance-specific loci; substance-specific loci include metabolic and receptor genes ▪ 19 addiction risk factor SNPs ▪ 9 alcohol-specific ▪ 32 tobacco-specific ▪ 5 cannabis-specific ▪ 1 opioid-specific
Saunders et al. 2022[2]	Alcohol Smoking	• GSCAN2: *N* = 3.4 million individuals from 4 major clines of global ancestry (~21% non-European)	• Many of the 3,823 associated variants from 2,143 loci showed consistent effect sizes across ancestry dimensions ▪ Polygenic scores developed in one ancestry performed poorly in others

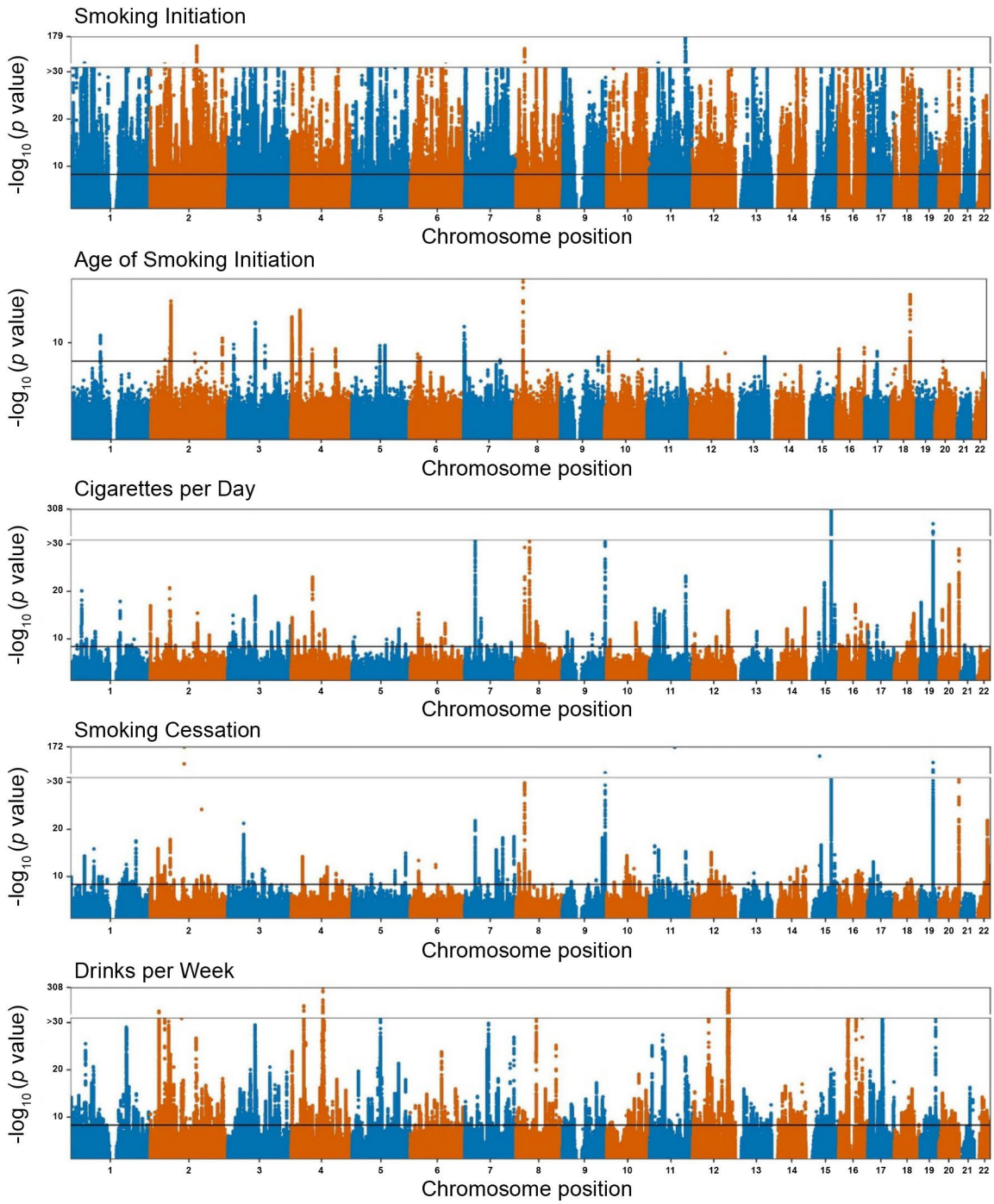
Table 6.1 (continued)

Study	Phenotype(s)	Sample[a]	Selected results
Xu et al. 2023[14]	Opioid Cannabis Alcohol Smoking	• Yale-Penn sample (*n* = 5,692 European ancestry, *n* = 4,918 African ancestry); Penn Medicine Biobank sample (*n* = 29,054 European ancestry, *n* = 10,265 African ancestry)	• Multi-trait analysis of opioid use disorder, cannabis use disorder, alcohol use disorder, and smoking initiation in European ancestry individuals; examined opioid use disorder, alcohol use disorder, and smoking trajectory in African ancestry individuals • MTAG boosted number of loci identified for all traits and identified genes not previously linked to any substance, and increased power of polygenic scores • European ancestry results: 36 loci for opioid use disorder, 60 loci for cannabis use disorder, 52 loci for alcohol use disorder, 144 loci for smoking initiation ▪ African ancestry results: 2 loci for opioid use disorder, 3 loci for alcohol use disorder, 1 locus for smoking trajectory

Note. GSCAN2 = GWAS and Sequencing Consortium of Alcohol and Nicotine use 2; MTAG = multi-trait analysis of GWAS; SNP = single nucleotide polymorphism.
[a]Population descriptors (e.g., European American) are quoted from the cited studies.

Large Genome-Wide Association Study for Tobacco and Alcohol. In GSCAN2, a large GWAS study, investigators examined four cigarette smoking phenotypes: initiation (whether an individual ever smoked regularly), the age of onset of regular use (age individual began smoking regularly), amount smoked (cigarettes per day among people who currently or formerly smoked), and cessation (current versus former smoking).[2] "Regular" use was based on survey questions addressing regular use explicitly or questions about number of cigarettes smoked over the life course or smoking every day for at least 1 month.[2] One alcohol use phenotype, drinks per week, was available across most studies. The results of this study are illustrated in Figure 6.4 using Manhattan plots, which allow graphical representations of the results of GWAS.[152] In these plots, the x-axis displays the genomic location of the SNPs identified and the y-axis the negative base 10 log of the *p* value obtained for each SNP. Points above the solid black line represent variants that have a significant association with the phenotype of interest adjusting for the genome-wide comparison number. Some of the y-axes are discontinuous because of very low *p* values for some SNPs (e.g., 1.9×10^{-308} for some drinks per week SNPs).[2] Compared to the original GWAS and Sequencing Consortium of Alcohol and Nicotine use (GSCAN) publication,[1] the multiancestry analysis greatly expanded the number of loci found, identifying a total of 2,143 loci across all phenotypes, with 3,823 independent variants.[2] Follow-up analysis of high-priority genes nearby top loci revealed that these genes were highly enriched across brain and nerve tissues, specifically within regions of the cortex, hippocampus, amygdala, hypothalamus, and midbrain. Furthermore, the high-priority genes were enriched in gene pathways related to neurogenesis, neuronal development, neuronal differentiation, and synaptic function, connecting them to the developmental and longitudinal aspects of tobacco and alcohol use disorders.[2] Five loci were associated with all 4 smoking phenotypes and the alcohol phenotype, localizing to large regions on chromosomes 2, 8, and 16, all of which contain multiple genes (G. Saunders, Ph.D., personal communication, September 12, 2023). These areas are excellent targets for focused attention on the comorbidity between tobacco and alcohol use.

Figure 6.4 Multiancestry Meta-Analysis Manhattan Plots for Smoking Behaviors and Alcohol Consumption



Note. Each dot on the plot signifies a single nucleotide polymorphism (SNP). Genomic coordinates are displayed along the x-axis, and each color block represents a chromosome. The y-axis is the negative logarithm of the association *p* value for each SNP. The strongest associations have the smallest *p* values (e.g., 10^{-15}), and their negative logarithms will be the greatest (e.g., 15). The black line across the plot marks 5×10^{-9} and SNPs above this line surpass the genome-wide significance level. Some y-axis scales are discontinuous to illustrate variants with very small *p* values.
Source: Saunders et al. 2022.[2] Reproduced, with minor formatting changes, with permission from *Nature.*

Multi-Trait Genome-Wide Association Studies. The Psychiatric Genomics Consortium (PGC) was established to unify researchers studying the genetic basis of psychiatric disorders and consists of more than 800 investigators from 36 countries with combined datasets exceeding 400,000 participants.[153] The Substance Use Disorder Working Group of PGC is focused on examining the genetics of substance use disorders and comorbid phenotypes.[154] Hatoum and colleagues (2023)[4] conducted a multivariate GWAS meta-analysis of a "general addiction risk factor," alongside analysis of individual substances including "problematic alcohol use," "problematic tobacco use," opioid use disorder, and cannabis use disorder. The term "problematic substance use" was introduced when one or more of the measures in the meta-analysis was derived from a scale rather than a diagnostic measure (A. Hatoum, Ph.D., personal communication, March 19, 2024). Hatoum and colleagues defined "problematic alcohol use" as including both alcohol use disorder (as a case-control GWAS) and the Alcohol Use Disorders Identification Test-Problem Items (AUDIT-P) subscale, a continuous measure that focuses on the problematic consequences of drinking. "Problematic tobacco use" was quantified based on the FTND scale of dependence.[125] The FTND has a genetic correlation of 0.95 to 0.97 with cigarettes per day, so these 2 items were parceled together using MTAG (A. Hatoum, Ph.D., personal communication, March 19, 2024). The total sample size included 1,025,500 individuals of European ancestry and 92,630 individuals of African ancestry.[4] In the European ancestry sample, the study identified 19 independent SNPs associated with the "general addiction risk factor" and highlighted top associations with the *PDE4B* and *DRD2* genes, both of which are involved in the dopaminergic reward pathway. Importantly, the association with *DRD2* was discovered through chromatin remodeling analyses, which indicates the variants are likely to be involved in regulation of the gene by turning it on or off under different circumstances (e.g., during development, time of day, in response to an environmental stimulus). This is a common theme among GWAS results, where many of the top SNPs do not fall within protein-coding regions of genes, but rather in regions where function is more difficult to predict.[155] In these cases, the SNP could be in a region contributing to regulation of gene expression or it could be associated (in linkage disequilibrium) with a different, causally related SNP.

Hatoum and colleagues (2023)[4] observed that substance-specific loci fell into three categories: substance receptors, substance-specific metabolism, and general neurotransmitter mechanisms. The first two categories confirmed previous results for both tobacco (e.g., *CHRNA5*) and alcohol (e.g., *ADH1B*). The largest category of variants was associated with general neurotransmitter mechanisms. Hatoum and colleagues also presented tables listing the top SNPs from each chromosome; in many cases, they are associated with both "problematic tobacco use" and "problematic alcohol use."

Finally, Hatoum and colleagues[4] showed that the polygenic score generated based on their results and applied in an independent sample of mostly substance-naive children ages 9–10 years (from the ABCD study)[25] was correlated with other co-occurring earlier childhood behaviors and environments, including measures of externalizing behavior and fun-seeking, sleep duration, parental externalizing and substance use behaviors, and socioeconomic disadvantage.[4] While a number of these associations are statistically significant, the standardized effect estimates were small, ranging from about 0.06 to 0.10.

This genetic connection to early risk factors highlights that predecessors of substance use are in part genetically driven by the same genetic variants associated with later tobacco and alcohol use. As with providing genetic risk information about other diseases or medical conditions, providing genetic risk scores for tobacco and alcohol use must be done with great care because this information can be stigmatizing or result in inappropriate victim blaming. This is especially challenging when treatments are only modestly effective and when social and environmental factors influencing tobacco and alcohol use are often difficult to address. The emerging body of research addressing how people respond to biological or genetic models of behavior and how such framing relates to stigmatization may be informative.[156] As of 2026, evidence is mixed as to whether information about genetic risk influences behavior change.[157] Furthermore, there appear to be geographic and cultural differences in how people respond to information about family and genetic variation in risk.[156] Continued research is needed to optimize communication about genetics and environmental aspects of risk.

Two other publications have applied MTAG to the analysis of genetically correlated traits. This approach was applied to data from the Million Veteran Program (N = 318,694) to examine 2 smoking phenotypes (smoking initiation and smoking cessation) and 2 alcohol phenotypes (alcohol consumption, as defined by the Alcohol Use Disorders Identification Test-Concise [AUDIT-C], and alcohol use disorder).[11] For both smoking initiation and smoking cessation, the MTAG strategy using alcohol use disorder GWAS summary statistics identified more genome-wide significant loci than the initial single-trait analysis.[11] In contrast, no additional variants were discovered for smoking measures using the alcohol consumption summary statistics. There were 10 loci showing colocalization between alcohol use disorder and smoking traits, including a functional analysis demonstrating pleiotropic effects between 2 genes and both smoking behaviors and gene expression. A second publication using MTAG with a smaller sample (n = 5,692 European ancestry; n = 4,918 African ancestry) examined 4 substance use traits among participants of European ancestry (opioid use disorder, cannabis use disorder, alcohol use disorder, and smoking initiation) and 3 substance use traits among participants of African ancestry (opioid use disorder, alcohol use disorder, and smoking trajectory).[14] This study also had power to detect additional associations via the multi-trait approach. Of note, eight novel and significant genome-wide loci for smoking initiation were detected in the European ancestry sample and one novel locus for smoking trajectory was detected in the African ancestry sample. Additionally, 10 novel genome-wide significant loci were identified in relationship to alcohol use disorder in the European ancestry sample. Overall, only 5 of 19 novel loci identified in the analysis of the European sample were significantly associated with 2 or more substances.

Many studies have converged on the same genes and/or physiological pathways as shared components of underlying risk for tobacco and alcohol use across diverse populations. Such findings hold substantial promise for the development of new classes of treatments and personalized medicine. However, these genetic risk factors may differ among populations with less genetic similarity. One strategy to examine this has been the use of multiancestry transcriptome-wide association studies, whereby expression data are integrated with GWAS data to identify correlations between associated SNPs and patterns of gene expression.[158] This

approach was applied to tobacco use phenotypes to identify additional genes and perform *in silico* drug repurposing analysis that identified possible alternative treatments for nicotine dependence.[159] These results highlight the importance of recruitment of large samples from diverse populations across the world as further efforts are made to develop personalized medicine approaches for tobacco and alcohol use and co-use.

The Potential for Personalized Medicine for Prevention and Cessation

As researchers continue to identify genetic loci associated with tobacco and alcohol use, some specific genetic markers and their related biological markers have been studied in the context of treatment trials to assess whether individuals with specific variants show differential likelihood of successfully quitting. Most of these studies have been conducted for treatment for either tobacco or alcohol use, but a few have encompassed treatment for both substances. NCI Monograph 20, published in 2009, presented initial forays into pharmacogenetic studies for cessation of tobacco use, providing potential avenues to design personalized treatment for smoking cessation[8] (personalized approaches to tobacco cessation are further discussed in chapter 7).

Surveys have shown that individuals want information about their personal genetic risk.[160] Providing genetic risk information accompanied by education/coaching about behavior change[161] could potentially strengthen efforts to reduce tobacco and alcohol use. However, such approaches have yet to be applied to tobacco and alcohol co-use; as of 2026, the evidence is mixed that such information improves success of behavioral interventions for either tobacco or alcohol use.[157,160-162]

Personalized Treatment for Tobacco Use

The field of pharmacogenomics seeks to understand how genetic variation influences the pharmacokinetic (absorption, distribution, metabolism, and excretion) and pharmacodynamic (interaction with receptors, transporters, and downstream targets) properties of a substance. These characteristics may affect treatment response and the potential for adverse events.[163] The nicotine metabolite ratio (NMR) measures the ratio of 3'-hydroxycotinine (3HC) to cotinine; higher ratios indicate faster nicotine clearance and lower levels of nicotine in the blood.[164] The NMR is a validated measure of nicotine metabolism that can be ascertained using saliva, plasma, or urine.[165]

The NMR is as a proxy for variation in the *CYP2A6* gene, because the CYP2A6 protein is largely responsible for the metabolism of nicotine.[166,167] Clinical studies typically separate participants into slow metabolizers (usually those in the first quartile of NMR) and fast metabolizers (all others, or those in the highest quartile of NMR) to determine whether different treatments (e.g., nicotine replacement therapy [NRT], bupropion, varenicline, placebo with counseling) are more or less effective in one group than the other. Some data support using NRT for slow metabolizers and non-nicotine treatments for normal metabolizers.[165,168,169] Together these and other studies support the possibility that NMR could be a predictive biomarker contributing to improved selection of treatments for smoking cessation.[167]

Studies have also examined *CYP* gene variants and CYP activity in people who smoke from different racial and/or ethnic groups. CYP2A activity levels differ across several groups but do not fully account for differences in smoking intensity and exposure to carcinogens.[170-172] Further studies of race and/or ethnic variation in metabolism of carcinogens and nicotine are needed to help account for differences in lung cancer incidence between demographic groups that appear independent of smoking intensity.[173] *CYP2A6* is thought to be an important target for the optimization of smoking treatments,[174] but its potential has not yet been realized.[175]

Variation in nAChR subunit genes has also been associated with differences in ability to quit smoking and may vary according to treatment.[167,168,176-180] A 2022 systematic review included studies of individuals of European ancestry and evaluated multiple variants across the *CHRN* genes.[180] The review found that variants in the *CHRNA5-A3-B4* gene cluster are risk alleles for cessation failure.[180] Results of pharmacotherapy studies suggested that NRT and bupropion might support abstinence among people with the high-risk alleles. However, the review did not assess combined NRT and varenicline, and the odds ratios for cessation were not significant for any therapy tested.[180] As of 2026, data for other nicotinic receptor subunits genes are lacking. In summary, this research represents the first foray into precision treatment to promote smoking cessation.

Personalized Treatment for Alcohol Use Disorders

Lohoff compiled a comprehensive review in 2020[163] describing the status of pharmacogenomic studies of alcohol use disorder. This included an evaluation of U.S. Food and Drug Administration (FDA)-approved medications for alcohol use disorder (disulfiram, acamprosate, and naltrexone) along with medications used off-label for alcohol use disorder (topiramate, gabapentin, baclofen, ondansetron, varenicline). For each of the FDA-approved medications, specific genetic variants have been studied in clinical trials based on genes known or believed to be related to the primary target for the medication. Variants in the *DBH*, *OPRM1*, and *ALDH2* genes were examined in a few studies; in some cases, specific alleles were associated with improved treatment response with disulfiram compared to the alternate allele, but these were small studies that have not been replicated.[163]

Naltrexone primarily targets the μ-opioid receptor, and a specific variant in *OPRM1* gene, rs1799971, corresponding to an amino acid substitution (asparagine to aspartate: Asn40Asp) has been widely studied in multiple clinical trials.[163] A meta-analysis evaluated seven randomized control trials (RCTs) that examined the effect of Asn40Asp on naltrexone treatment for heavy drinking or alcohol use disorder. This meta-analysis identified a moderating effect of Asn40Asp on drinks per day, but this effect was not significant when taking into account multiple comparisons; the authors concluded that the available evidence remains unclear about whether this genetic variant predicts naltrexone treatment response.[181]

Acamprosate is believed to target N-methyl-D-aspartic acid (NMDA) receptors and indirectly affect γ-aminobutyric acid type A ($GABA_A$) neurotransmission.[182] Polymorphisms in *GATA4*, *DRD2*, *GABRA6*, *GABRB2*, and *GRIN2B* have been studied in relation to treatment with acamprosate, and overall evidence suggests that individuals who have an initial desire to remain abstinent and receive acamprosate treatment may have reduced risk of relapse to drinking.[183] The efficacy of treatment may be partially moderated by variation in these genes, but results are limited.[183]

Topiramate increases GABA transmission, leading to inhibition of glutamate, and is used as an anticonvulsant medication; this medication requires close monitoring for side effects.[184] A meta-analysis of seven RCTs found topiramate to be significantly better than placebo at facilitating decreased drinking, but the effect was moderate.[185] Furthermore, emerging evidence suggests that individuals with the C-allele of rs2832407 in the *GRIK1* gene may respond better to topiramate treatment.[184]

For the other medications, few or no pharmacogenomic studies have been conducted. As with treatment for tobacco use, additional studies are needed before large-scale clinical efforts to implement personalized treatments for alcohol use disorder should be undertaken. For example, research is just beginning on the potential of somatostatin (SST), vasoactive intestinal peptide (VIP), glucagon-like peptide-1 (GLP-1), nociceptin (NOP), and neuropeptide S (NPS) as targets for alcohol use disorder pharmacotherapies.[186] Interest has also emerged in treating alcohol use disorder with psychedelic drugs based on some promising results,[187-189] but data to consider personalized approaches are lacking.

Personalized Treatment for Comorbid Tobacco and Alcohol Use Disorders

Varenicline, a pharmacotherapy for smoking cessation, is a partial agonist at the α4β2 nAChRs, but a full agonist at α7 nAChRs, and likely affects other nAChR subtypes.[190-192] Several studies have also examined use of varenicline for alcohol use disorder.[163] Two trials of varenicline for alcohol use disorder suggested varenicline could be effective in reducing heavy drinking days or alcohol consumption in people who smoke at least 10 cigarettes per week[193] and in both people who do and do not smoke.[194] Evidence also indicates that varenicline can facilitate smoking cessation among people who drink heavily and also smoke.[195] However, while varenicline use for alcohol use disorder has shown some positive effects,[196] it is not FDA approved for this indication (see chapter 7). Most recently, a 2023 meta-analysis of 22 RCTs of varenicline for treatment of alcohol use disorder reports reduction in some but not all measures of alcohol use and use behavior.[197]

No studies were identified that examined genetic factors (e.g., variation in the *CHRN* genes) that may moderate treatment responses to varenicline use for co-use of tobacco and alcohol. Previous studies focused on either tobacco or alcohol use may have collected data from participants that used both substances; however, these studies are likely to be underpowered to draw meaningful conclusions about the complex genetic interactions relating to co-use behaviors.

In sum, there is evidence that varenicline may be a useful treatment option for people who both smoke and have alcohol use disorder. However, effect sizes are modest and understanding of variation in effectiveness is limited. As more well-powered clinical studies examining various therapies for cessation of tobacco and alcohol are conducted, opportunities for treating co-use and for exploring personalized treatment approaches will likely increase. See chapter 7 for further discussion of pharmacotherapies for tobacco and alcohol use.

Box 6.1. The Potential Role of Glucagon-Like Peptide-1 Agonists in Treating Substance Use Disorders

GLP-1 receptor agonists were originally developed as treatments for type 2 diabetes and are now commonly used to treat obesity.[198] These medications mimic the incretin hormones that stimulate the release of insulin in response to eating; they suppress appetite and decrease body weight by activating gut-brain signaling mechanisms involving several brain regions.[199] FDA approval of GLP-1 medications to treat type 2 diabetes and obesity has resulted in their wide use, and patients have anecdotally reported unanticipated decreases in their use of alcohol and other addictive substances.[200] In addition, studies conducted in animals support a potential role for these medications in treating alcohol use disorder.[201,202] These findings have resulted in a small but growing number of trials examining the effects of these medications on substance use. Martinelli and colleagues (2024)[203] identified 5 RCTs examining tobacco (n = 2), alcohol (n = 2), and cocaine use (n = 1) in a total of 630 participants receiving either of 2 GLP-1 agonists (exenatide or dulaglutide). Of these five studies, one observed increased abstinence from smoking in the medication group and one observed reduced alcohol consumption in the medication group (compared with patients receiving placebo). Additionally, a study comparing patients taking semaglutide for type 2 diabetes to patients taking other antidiabetes medications, found that those taking semaglutide had a lower risk of utilizing health care for tobacco use disorder, suggesting that the medication may have reduced their tobacco use.[204] Other studies have found that semaglutide taken once per week reduced craving and some measures of consumption in people with alcohol use disorder,[205] and once weekly exenatide increases tobacco cessation success and weight loss.[206] Interestingly, a further study of exenatide for smoking cessation reported that it was only associated with cessation for a specific genotype (*CHRNA5* rs16969968 GG),[207] a gene known to be associated with response to treatment for smoking cessation.[177] Additional RCTs are needed to clarify the potential role of GLP-1 agonists in treating substance use disorders, although their widespread use will also result in substantial data from observational studies.

Using Large-Scale Genetic Studies to Predict Cessation

As the field of genetics has moved from candidate gene studies to large-scale GWAS, there has also been a movement from studies of single genes predicting cessation to using information from many genes to predict successful cessation. A goal of this approach is to use results of large-scale GWAS to generate polygenic scores accounting for clinically significant variation in cessation success and other aspects of tobacco and alcohol use. Polygenic scores are an estimate of genetic risk based on variation in SNPs calculated with results from GWAS and other data.[208]

Most polygenic scores have been derived in populations of European ancestry and therefore have greater predictive accuracy among genetically similar populations.[209] Research is underway to improve the predictive ability of polygenic scores across different populations.[210] For example, Lennon and colleagues (2024)[211] have proposed a detailed polygenic scores pipeline designed to improve the development and validation of polygenic scores and prevent exacerbation of health disparities associated with polygenic scores derived primarily from European ancestry populations.

The use of polygenic scores represents the next frontier in translating knowledge of genetic risk into improved health.[160] However, research is needed to determine whether this approach can contribute to addressing tobacco and alcohol use. Utility of polygenic scores for public health goals depends on both the capacity to identify subjects differing in risk and the availability of specific interventions that could help people with different polygenic scores for initiation, continued use, and cessation. A 2022 study to identify polygenic scores for alcohol use disorder and alcohol use disorder severity found that the odds ratio for developing alcohol use disorder associated with the highest decile of the polygenic score (OR = 1.96, 95% CI = 1.54–2.51) was similar to those for having a first-degree family history[212] and that both family history and polygenic score provided unique information about risk. Whether this kind of risk information can be used to improve cessation of tobacco and/or alcohol use is not yet established.

Chapter Summary

Understanding how genetic variation influences the risk of substance use, including the use of tobacco and alcohol, can contribute to the prevention and treatment of substance use disorders. Genetic studies have focused on identifying variants related to nicotine, the primary addictive component of tobacco products, and ethanol, the primary addictive component of alcohol products. Complex behaviors, such as tobacco use, alcohol use, and tobacco-alcohol co-use, develop in stages over time, as a result of the interaction of thousands of genetic factors with diverse environmental factors. As of 2026, most genetic research on substance use has been conducted in adults. However, new longitudinal studies—the Adolescent Brain Cognitive Development (ABCD) study and the Healthy Brain and Child Development (HBCD) study—are underway to help understand the onset of substance use in adolescents and younger children. The topic of genetic contributions to tobacco use was reviewed in NCI Monograph 20, *Phenotypes and Endophenotypes: Foundations for Genetic Studies of Nicotine Use and Dependence*, published in 2009. Since that report's publication, advances in technology—most notably the development of high-throughput genotyping technologies enabling genome-wide association studies (GWAS)—have contributed to significant advances in the field.

Research into how the brain responds to nicotine and ethanol and to the use of tobacco products and different types of alcohol-containing beverages reveals a complex interplay of many brain regions that contribute to altered neurobehavioral processes involved in substance use and development of substance use disorders. The triadic neurobehavioral model describes the neurophysiological mechanisms underlying substance use disorders in terms of three functional domains (incentive salience, negative emotional states, and executive function) mediated by

distinct neurobiological circuits (basal ganglia, extended amygdala, and prefrontal cortex, respectively). All addictive substances, including nicotine and ethanol, affect these three major neurobiological circuits; additionally, substance-specific neurobiological effects have also been identified.

Addictive behaviors, including tobacco and alcohol use, involve changes in the production, release, and binding of dopamine in the brain. Over time, tolerance (when a greater amount of the substance is needed to elicit the same response) develops. Cross-tolerance (when repeated administration of one substance induces a decreased response to another substance) is associated with co-use of tobacco and alcohol and has important implications for prevention and treatment interventions.

General processes of addiction influence response to multiple substances with co-use leading to additive effects on reward systems and development of substance use disorders. This conclusion is supported by neurophysiological findings from both human and rodent studies. These shared processes at the genetic and neurophysiological levels help explain why co-use of tobacco and alcohol is especially common in people who use tobacco and who drink on average at higher levels, such as more than one drink a day for women and two drinks per day for men.

Neuroimaging studies support the importance of the classic triadic neurobehavioral model of addiction—but also extend it. Altered salience attribution, negative emotional states, and dysregulated prefrontal function are at the core of substance use disorders. However, higher order processes, such as changed appraisal of subjective value, altered self-directed processing, and changes in flexible learning and memory in individuals with substance use disorders also play a critical role in substance use and substance use disorders. The brain networks identified from the human neuroimaging studies of tobacco and alcohol use differ slightly from those identified in animal (rat, mouse) models; this is likely attributed to differences in experimental procedures and imaging tools, as well as interspecies differences.

Studies of people who use tobacco and alcohol as well as other addictive substances suggest there may be several different neurophysiological precursors that can lead to substance use and/or substance use disorders. Ongoing longitudinal studies that include sophisticated brain imaging and extensive phenotyping concerning psychological traits should help further elucidate these potential precursor phenotypes and may help inform the development of pharmacotherapies to prevent the onset of tobacco and alcohol use and co-use.

Genetic studies further illuminate the relationship between genes and substance use. Studies of monozygotic (identical) and dizygotic (fraternal) twins have been used to estimate heritability, a statistical concept that estimates how well differences in genetic factors within a population account for differences in a particular trait. Heritability ranges from zero to one, with values close to zero indicating that the trait is mostly due to environmental factors, and higher values indicating the trait is largely due to genetic factors. Heritability is specific to the population studied and can change as the environment or genetic makeup of a population changes. Geneticists use heritability to ascertain that a trait is determined, at least in part, by genetic factors, and to determine how much shared genetic variance exists between traits. Extensive

research has now demonstrated that, across populations, the risk for smoking and alcohol use is split roughly equally between genetic and environmental factors. Research suggests that environmental factors (e.g., substance use by peers and family, price, advertising) play a larger role in initiation of tobacco and alcohol use than genetic factors. Conversely, evidence suggests that genetic factors play a larger role in continued and increased substance use than environmental factors.

Prior to the availability of GWAS, genetic studies employed family-based linkage analyses or case-control association studies. While important, these studies had very limited ability to identify genes underlying behavioral traits, such as substance use, which result from the interaction of thousands of genetic factors, combined with environmental influences. Although GWAS represent an enormous advance, these studies also have limitations, which may include limited phenotypic assessment, difficulty controlling for population stratification, and limited ancestry diversity of study participants. Additionally, initial studies have focused either on tobacco use or alcohol use phenotypes, and few have examined tobacco–alcohol co-use. Progress is being made, however, thanks to the use of electronic medical records, the emergence of large "biobanks," measures to promote data sharing, and other factors.

Large-scale human genetic research has identified numerous potential risk loci linked to both tobacco and alcohol use, whether individually or as co-occurring disorders. Of note, studies show that the genetic pathways that contribute to risk for tobacco and alcohol use disorders are similar across diverse populations. While many risk loci have not been mapped directly to specific genes, those that have confirm the significance of previously identified neurophysiological pathways (e.g., involving nicotinic acetylcholine receptors, *CHRN* genes). Additionally, variations in genes and proteins that affect the metabolism of nicotine (e.g., cytochrome P450, *CYP* genes) and ethanol (e.g., alcohol dehydrogenase, *ADH* genes; aldehyde dehydrogenase, *ALDH2* genes) have consistently been associated with individual responses to these substances, influencing the risk of increased use, and suggesting avenues for the development of pharmacotherapies or more effective use of existing therapies. In cases where GWAS results identify single nucleotide polymorphisms (SNPs) outside protein-coding regions of genes, the function of these is more difficult to predict; however, these SNPs could contribute to regulation of gene expression or be associated with a different, causally related SNP. Despite these advancements, developing effective treatments for substance use, including tobacco and alcohol co-use, tailored to individual genetic profiles is not yet a reality. The field of personalized medicine for substance use is still in its early stages, requiring further research to determine whether personalized strategies for tobacco cessation and alcohol cessation/reduction can be developed. Progress hinges on collaborative efforts among experts in physiology, pharmacology, genetics, and psychology, who must address the intricate interactions between genetic factors and environmental influences on tobacco and alcohol use.

In summary, significant strides have been made in understanding the brain's response to nicotine and ethanol and identifying genetic factors linked to substance use. However, translating these findings into precise, effective treatments remains a challenge. Continued interdisciplinary research and integration of genetic and environmental data are crucial for advancing personalized medicine in the realm of substance use disorders.

Research Needs

Although the field has been successful at identifying genetic variants associated with nicotine and ethanol use, very few studies have carefully considered these behaviors together. Improved understanding of genetic associations and pathways that overlap between tobacco and alcohol use behaviors will provide important insight in the context of personalized medicine. Additional well-powered studies that combine brain imaging with genetics in children and adolescents, such as the HBCD study and ABCD study, have the potential to differentiate between functional brain differences that are predisposing to substance use and those that result from substance exposure. Such studies could also be strengthened by the further characterization of phenotypes and especially endophenotypes of tobacco and alcohol co-use at specific periods of initiation and use over the life course.

Further, more research is needed to develop prevention and treatment approaches that specifically address heterogeneity in the three neurofunctional domains related to addiction. Longitudinal studies of brain development and neurobehavioral characteristics are needed to better understand how these trait-like factors are involved in addiction and substance use across the lifespan. Ongoing cohort studies, such as the HBCD and the ABCD studies, should help address these questions through a combination of extensive phenotyping, brain imaging, and biospecimen collection.

Genetic and epidemiological analyses depend on consistent and meaningful definitions of use categories. For cigarettes, a threshold of having smoked at least 100 cigarettes is widely used to classify a person as having ever been an established user (e.g., rather than having tried smoking a few times). However, there is no standard for a minimum level of e-cigarettes or alcohol exposure for classifying participants as ever users. Establishing such thresholds and consideration of combined thresholds across both tobacco and alcohol products may facilitate research in this area. In addition, consistent measurement of various consumption patterns in longitudinal epidemiologic studies, such as nondaily smoking and drinking over the life course and analysis with genetic data, would further discovery of gene-environment interactions for tobacco and alcohol co-use.

Further analysis of the ontogeny of tobacco and alcohol co-use in diverse populations is needed. Such studies should continue efforts to address populations of diverse ancestry and to examine interactions between environment and the range of genetic and development variation over the entire life course. In addition, the neuroimaging literature suggests that there are sex differences in the neurobiological mechanisms underlying substance use disorders[83]; research is needed to explore these differences.

Although *post hoc* strategies used to characterize the genes and variants emerging from GWAS have begun to identify pathways, few of the GWAS results have been pursued at the functional level. Most GWAS results do not fall within a gene with obvious function, so methods to quickly predict and test functional implications of individual variants must be expanded. One such method (Functional Mapping and Annotation of Genome-Wide Association Studies; FUMA) has been developed and is now widely used.[213,214]

Although the field widely recognizes that gene-gene interactions and gene-environment interactions contribute to tobacco and alcohol use, studies have been underpowered to detect such interactions. This area of research is poised for the development of new methods, which should also consider environmental factors that change over time, such as the tobacco product marketplace. For example, the entry of e-cigarettes into the marketplace has resulted in high levels of adolescent use of e-cigarettes. These genetic characteristics and behavioral phenotypes could also be studied in relation to additional disease outcomes related to tobacco and alcohol co-use.

There remain many research questions related to the genetics of tobacco and alcohol use phenotypes and their diverse health effects. For example, the GWAS and Sequencing Consortium of Alcohol and Nicotine use (GSCAN) data were used to estimate the genetic correlation between smoking measures and lung cancer; in general, heavier smoking and lower smoking cessation were positively associated with increased risk for lung cancer and chronic obstructive pulmonary disease.[215] Further, variation in the *CHRNA5* gene, which is one of the loci most strongly associated with heavy smoking and failed smoking cessation,[167] is also one of the strongest associations with the development of lung cancer (see chapter 5).[215] It is also clear that there are genetic associations specific to the development of lung cancer that are not smoking related.[216] However, genetic variants of the locus coding the nicotine metabolizing genes (*CYP2A6*) displayed an inverse relationship with smoking behaviors; despite being associated with an increased risk of heavier smoking, lung cancer, and chronic obstructive pulmonary disease, they were also associated with successful smoking cessation.[215] This inverse relationship highlights the need to better understand how nicotine metabolism alters the risk of heavier smoking measured by cigarettes smoked per day, lung cancer, and chronic obstructive pulmonary disease while also contributing to increased smoking cessation. Disentangling the common and specific genetic contributions to smoking and lung cancer may be useful in developing new treatments or treatment approaches. Together these results highlight the importance of investigating the genetic associations between behavior and risk to determine whether they can inform the development of improved approaches to cessation.

Similarly, there is a relationship between the genetics of alcohol consumption and aerodigestive tract cancers. Multiple risk variants in the *ADH* and *ALDH* genes have emerged from GWAS for aerodigestive tract cancers (see chapter 5).[217] There is evidence that tobacco and alcohol use leads to genetic variations in tumor suppressor genes, along with other disruptions to cell-cycle processes, which contribute to the development of oral cancers.[218] Emdin and colleagues (2021)[219] report that a polygenic score derived from 12 independent genetic variants for cirrhosis of the liver identifies individuals who are most susceptible to the hepatotoxic effects of excess alcohol consumption or obesity. Strong genetic predictors of excess risk related to behavior and obesity could potentially guide more targeted interventions. Overall, it is a significant research gap that few genetic studies as of 2026 have directly addressed phenotypes of co-use of tobacco and alcohol.

Further research is needed to optimize health communications related to family history, and to genetic and environmental influences of risk of tobacco and alcohol use, and co-use. Notably, it is unclear from the present literature whether information about family history improves responses to health communications or influences decision-making in a positive way. Further research is also needed on how stigma influences health cognitions related to individual and family history.

Conclusions

1. **Tobacco and alcohol use affect multiple complex neurophysiological processes in the brain.** Networks of brain regions that include the nucleus accumbens; amygdala; habenula; interpeduncular nucleus; hippocampus; insula; as well as cingulate, orbitofrontal, and prefrontal cortices influence how people respond to nicotine and ethanol, the primary substances in tobacco and alcohol products that lead to dependence and addiction.
2. **Large-scale human genetic studies have identified thousands of genetic variants associated with tobacco and alcohol use.** Variation in genes and in proteins involved in the metabolism of nicotine and ethanol have been associated with an individual's response to these substances, patterns of continued use, and risk for sustained use and dependence. These associations provide a mechanistic basis for some of the observed differences in substance use at the population level.
3. **Multiple risk loci have been mapped to genes known to be involved in nicotine and ethanol use and their physiological effects.** The pathways of interest include dopaminergic, GABAergic, glutamatergic, and stress response pathways. These pathways influence how genetic variations affect individual responses to nicotine and ethanol, patterns of continued use, and the potential development of substance use disorders related to other drugs.
4. **Genetic studies have identified a substantial number of genetic variants that are associated with both alcohol use and tobacco use across populations, providing evidence for shared underlying mechanisms.** A better understanding of the shared biological mechanisms from additional genetic studies will inform efforts to prevent use disorders and develop pharmacotherapies for tobacco and alcohol co-use and polyuse with other substances.
5. **Research to understand neurophysiologic and genetic factors contributing to tobacco and alcohol use and their co-use is beginning to inform precision medicine treatment approaches.** An understanding of the complex interplay between genetic and environmental factors is essential for personalized treatments for tobacco and alcohol use to be most effective. Additional research is needed to effectively use existing therapies and develop personalized pharmacotherapies for tobacco and alcohol co-use.

Appendix 6A Selected Genome-Wide Association Studies of Tobacco and Alcohol Published Since 2018

Study	Phenotype(s)	Sample[a]	Selected results
23andMe Research Team 2019[220]	Alcohol Smoking	UK Biobank and 23andMe (*N* = 939,908) Replication • 10 cohorts from 7 studies (*n* = 35,445)	• 46 of 99 general risk tolerance loci contain lead SNP for at least 1 other GWAS • Genes near SNPs are highly expressed in the brain
Deak et al. 2022[221]	Alcohol	Million Veteran Program (*n* = 218,623 European American and *n* = 29,132 African American)	• 15 genome-wide significant loci for "maximum habitual alcohol intake" • "Maximum habitual alcohol intake" is genetically correlated with measures of alcohol-related problems, but alcohol consumption measures load on a separate factor
Deak et al. 2022[222]	Alcohol Cannabis Tobacco Opioid	7 cohorts: Million Veteran Program, Psychiatric Genomics Consortium, iPSYCH, FinnGen, Partners Biobank, Vanderbilt University Medical Center (BioVU), Yale-Penn 3 • *N* = 639,063 (*n* = 84,877 African ancestry; *n* = 544,186 European ancestry)	• 18 independent, genome-wide, significant loci for opioid use disorder, including *OPRM1* • Strong genetic correlations of opioid use disorder with alcohol use disorder (r_g = 0.77) and cannabis use disorder (r_g = 0.82)
Kember et al. 2023[130]	Alcohol	Million Veteran Program longitudinal data (*N* = 409,630; *n* = 296,989 European American; *n* = 80,764 African American; *n* = 31,877 Hispanic American) • Validation in Vanderbilt University Medical Center (BioVU) (*n* = 66,903 European American; *n* = 12,384 African American) • UK Biobank (*n* = 123,561 European ancestry; *n* = 1,182 African ancestry)	• 26 loci associated with alcohol use disorder and 22 loci associated with AUDIT-C (some ancestry-specific and some novel) • Excluding subjects reporting abstinence, additional loci were found (7 for alcohol use disorder and 8 for AUDIT-C) ▪ Mediation analysis revealed set of variants with effects on alcohol use disorder that are not mediated through alcohol consumption
Kranzler et al. 2019[223]	Alcohol	• Million Veteran Program (*N* = 274,424) • Replication samples: Penn Medicine BioBank (*n* = 9,965), Yale-Penn study (*n* = 9,739)	• 18 genome-wide significant loci ▪ 5 associated with AUDIT-C and alcohol use disorder ▪ 8 associated with AUDIT-C only ▪ 5 associated with alcohol use disorder only ▪ Polygenic scores for both traits replicated in 2 independent samples
Liu et al. 2019[1]	Alcohol Smoking	GWAS & Sequencing Consortium of Alcohol and Nicotine use (GSCAN): • *N* of up to 1.2 million (depending on phenotype)	• Total of 566 variants in 406 loci associated with tobacco use and/or alcohol use • 150 loci showed evidence for pleiotropic association

Appendix 6A (continued)

Study	Phenotype(s)	Sample[a]	Selected results
Mallard et al. 2022[224]	Alcohol Smoking Psychiatric disorders	Discovery: • UK Biobank (*n* = 147,267); Netherlands Twin Register (*n* = 9,975); Avon Longitudinal Study of Parents and Children (*n* = 3,582) Replication: • UK Biobank different subset (*n* = 239,719); Collaborative Study on the Genetics of Alcoholism (COGA, *n* = 6,390); Vanderbilt University Medical Center (BioVU, *n* = 91,602)	• Identification of a 2-factor structure at the genetic level exemplified by "consumption" and "problems" which are: ▪ Highly correlated (r_g = 0.80) ▪ Strongly associated with alcohol dependence (r_g = 0.67) • Moderately associated with other psychiatric disorders, including cigarettes per day
Miller & Gizer 2024[225]	Alcohol Imaging	23andMe, GSCAN, UK Biobank, Million Veteran Program, AlcGen, Cohorts for Heart and Aging Research in Genomic Epidemiology (CHARGE), Psychiatric Genomics Consortium, FinnGenR6, Enhancing Neuro Imaging Genetics through Meta-Analysis (ENIGMA) Various combinations depending on phenotype sample sizes: • Sensation seeking: 710,971 • Alcohol consumption: 1,388,120 • Alcohol use disorder: 220,182 • Neuroimaging: ~30,000	• Sensation seeking, alcohol consumption, alcohol use disorder, and neuroimaging • Support for shared genetic architecture between sensation seeking and alcohol consumption, via genes expression in midbrain and striatal tissues, and genetic variants associated with increased cortical surface area • Alcohol consumption and alcohol use disorder show overlap with genetic variants associated with decreased frontocortical thickness • Mediation models provide evidence of alcohol consumption mediating associations between sensation seeking and alcohol use disorder
Rabinowitz et al. 2022[226]	Alcohol Cannabis Tobacco Common substance use factor Imaging	• 71 neuroimaging studies from the ENIGMA Consortium (*N* = ~33,000) • GWAS summary statistics for tobacco and alcohol use from 23andMe and GSCAN cohorts (*N* = ~1.2 million) • GWAS summary statistics for cannabis use from International Cannabis Consortium, UK Biobank, and 23andMe (*N* = 184,765) • Adolescent Brain Cognitive Development (ABCD) study (*N* = 10,403)	• 8 significant negative correlations ▪ Alcohol drinks per week and average cortical thickness ▪ Intracranial volume with age of smoking initiation • 5 positive genetic correlations ▪ Insula surface area and lifetime cannabis use ▪ Common substance use behavior genetic factor and pericalcarine surface area • There is a shared genetic etiology between cortical brain morphology and substance use behaviors

Appendix 6A (continued)

Study	Phenotype(s)	Sample[a]	Selected results
Risner et al. 2021[227]	Alcohol Smoking Psychiatric disorders	Pooled Database of Genotypes and Phenotypes (dbGAP) samples (*n* = 6,394, European ancestry); National Longitudinal Study of Adolescent to Adult Health (Add Health) Wave IV (*n* = 3,061, European ancestry)	• Nicotine dependence (cigarettes per day, AUDIT-C, AUDIT-P, neuroticism, schizophrenia, depression, BMI, self-perceived risk-taking, educational attainment) • Partial correlations between nicotine dependence and polygenic scores developed for each of the individual nine phenotypes • Multi-trait polygenic score also shows similar estimates with nicotine dependence, but depends on phenotype
Toikumo et al. 2024[13]	Tobacco	PsychMerge network biobanks: Vanderbilt University Medical Center (BioVU), Mass General Brigham Biobank, Penn Medicine BioBank, Million Veteran Program, UK Biobank *N* = 898,680 subjects (*n* = 495,005 European American, *n* = 114,420 African American, *n* = 44,365 Latin American ancestry, and *n* = 244,890 European ancestry from UK Biobank)	• Tobacco use disorder based on electronic health records • 88 independent risk loci • 461 potential risk genes, primarily expressed in the brain • Tobacco use disorder genetically correlated with many other health outcomes
Wang et al. 2023[228]	Alcohol	Michigan Longitudinal Study (*n* = 665; European ancestry)	• Problem alcohol use that combines frequency of alcohol use, maximum drinks in 24-hour period, and alcohol use problems at 4 assessments during young adulthood (11–29 years) • Problem alcohol use polygenic score was significantly associated with alcohol-related problems in young adulthood even after adjusting for: ▪ Demographic covariates ▪ Parental alcohol use disorder • Drinking and alcohol use problems in early and mid/late adolescence
Xu et al. 2020[229]	Smoking	Million Veteran Program (MVP; *n* = 286,118;), includes *n* = 209,915 European American, *n* = 54,867 African American, *n* = 21,336 Hispanic American) Meta-analysis with GSCAN (*n* = 842,717)	• EMR-based smoking trajectories ▪ In MVP: 18 loci for smoking trajectory in European Americans; 1 locus in African Americans; 1 locus in Hispanic Americans ▪ In MVP and GSCAN: 99 loci for smoking initiation ▪ In MVP and GSCAN: 13 loci for smoking cessation
Zhang et al. 2022[230]	Alcohol Nicotine Cannabis Opioid	National Epidemiologic Survey on Alcohol and Related Conditions-III (*n* = 22,848 subjects, *n* = 12,505 predominately European ancestry)	• 21 independent SNPs and 17 gene-based loci for substance use disorders • Some SNPs/loci are substance-specific, some are shared

Appendix 6A (continued)

Study	Phenotype(s)	Sample[a]	Selected results
Zhou et al. 2020[231]	Alcohol	*N* = 435,563 European ancestry subjects from Million Veteran Program Phase 1 (*n* = 202,004), MVP Phase 2 (*n* = 65,387), Psychiatric Genomics Consortium (*n* = 46,568), and UK Biobank (*n* = 121,604) Replication of polygenic score in Vanderbilt University Medical Center (BioVU) (*n* = 67,589)	• 29 independent risk variants (19 novel) • Problem alcohol use was genetically correlated with 138 phenotypes • Heritability analyses of problem alcohol use showed enrichments in the brain and in conserved and regulatory genomic regions
Zhou et al. 2022[232]	Alcohol Tobacco	5 cohorts (*N* = 13,551): • Thai study of genetics methamphetamine dependence – Global Screening Array (*n* = 532) • Thai study of genetics of methamphetamine dependence – Multi-Ethnic Global Array (*n* = 2,370) • Million Veteran Program-East Asian Americans (*n* = 6,955) • Han Chinese – Global Screening Array (*n* = 3,381) • Han Chinese – Cyto12 array (*n* = 313) • Independent evaluation of polygenic score in Genetic Epidemiology Research in Adult Health and Aging (*n* = 4,464 East Asians)	• Detected 2 well-known loci, one in *ADH1B* and one in *BRAP* near *ALDH2* • Alcohol use disorder polygenic score associated with days per week of alcohol consumption and nominally associated with pack-years of smoking and ever versus never smoking
Zhou et al. 2023[233]	Alcohol	Multiple cohorts (*N* = 1,079,947; *n* = 903,147 European ancestry; *n* = 122,571 African ancestry; *n* = 38,962 Latin American ancestry; *n* = 13,551 East Asian ancestry; *n* = 1,716 South Asian ancestry), including Million Veteran Program, UK Biobank, FinnGen, iPSYCH, QIMR Berghofer Medical Research Institute, Psychiatric Genomics Consortium, Yale-Penn 3, East Asian cohorts	• Problematic alcohol use based on DSM-IV alcohol dependence and AUDIT-P scale • 110 independent risk variants • High degree of cross-ancestral similarity in genetic architecture of problematic alcohol use • Fine mapping across ancestries allowed narrowing of putative functional variants • Transcriptome-wide association study and chromatin interaction analyses implicated brain-expressed genes • Polygenic score derived from multiple ancestries improves predictability and explains more variance relative to single-ancestry polygenic score

Note. ABCD = Adolescent Brain Cognitive Development; AUDIT-C/AUDIT-P = Alcohol Use Disorders Identification Test (C = Concise; P = Problem); BMI = body mass index; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders (Fourth Ed.); EMR = electronic medical record; GSCAN = GWAS and Sequencing Consortium of Alcohol and Nicotine use; GSCAN2 = GWAS and Sequencing Consortium of Alcohol and Nicotine use 2; GWAS = genome-wide association study; *N* = full study sample; *n* = subpopulation sample; MTAG = multi-trait analysis of GWAS; SNP = single nucleotide polymorphism. Four key GWAS of tobacco and alcohol[2,4,11,14] are presented in Table 6.1
[a]Population descriptors (e.g., European American, European ancestry) are quoted from the cited studies.

Appendix 6B Literature Search Strategy

To identify relevant studies, the following search strings were used in PubMed: "GWAS and nicotine"; "genetics and smoking"; "smoking and imaging studies". Searches were conducted between June and December 2023 to identify peer-reviewed studies published since PubMed inception through December 2023. Studies were also identified based on reviewing reference lists and review articles cited in key papers and/or presentations at scientific meetings, such as the NIDA Genetics and Epigenetics Cross-Cutting Research Team Meeting. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

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Chapter 7
Treatment Approaches for Tobacco and Alcohol Use

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Chapter 7
Treatment Approaches for Tobacco and Alcohol Use

Introduction

Previous research has identified the co-use of tobacco and alcohol as a major public health problem in need of innovative and combined treatment modalities.[1-4] Studies have shown that people who smoke are more likely to drink heavily and meet criteria for alcohol use disorder (AUD) than people who do not smoke, and those with AUD are less likely to quit smoking than those without AUD.[5-8]

This chapter reviews the evidence for treating cigarette smoking and alcohol use concurrently and for therapies specifically designed to address their use and co-use. People with alcohol or substance use disorder (SUD) are more likely to die from illnesses caused by smoking, including cancer caused by smoking, than the general population.[9] In addition, evidence indicates that people who smoke and have an SUD are more likely to die from a disease caused by tobacco than as a result of their SUD.[10] Together this underscores the need for integrated strategies to address both behaviors.

The evidence supports treating smoking concurrent with alcohol use treatment to enhance a person's ability to both quit smoking and achieve or sustain alcohol and substance use abstinence or reduced use.[11-13] With integrated treatment, people who smoke and have a co-occurring AUD can attain the same level of smoking cessation success[12] as people who smoke and do not have a co-occurring AUD.[14] Additionally, alcohol consumption can make quitting smoking more difficult and a relapse to cigarette smoking more likely, especially when alcohol use is heavy or meets criteria for AUD.[8,15-17] Providing smoking cessation treatment to people who smoke and are in treatment for alcohol and/or other substance use (1) does not harm the success of the alcohol and substance use treatment and (2) may improve the chance of success.[11-13] Analysis of longitudinal data from the Population Assessment of Tobacco and Health (PATH) study found that quitting smoking was associated with a 30% increase in the odds of recovery from substance use disorder.[18] Therefore, treating co-use of tobacco and alcohol may improve outcomes for both behaviors versus treating one substance at a time or focusing on treating one versus the other.

State of the Science

Quitting smoking usually takes several attempts to achieve long-term abstinence.[19] Reducing or quitting drinking can be challenging as well, especially for people with AUD; people seeking treatment for AUD often relapse within 6 months of treatment.[20-22] Tobacco and alcohol can exert mutually reinforcing effects, both behaviorally and neurobiologically (detailed in the section "Understanding and Leveraging Neurobiological and Neurobehavioral Connections" and in chapter 6), and therefore generate a unique need for integrated treatments.[23-30] There has been substantial progress in smoking cessation research since the mid-20th century, including development of novel behavioral treatments (e.g., quitline counseling) and pharmacotherapies

(e.g., nicotine replacement therapy [NRT], varenicline) and repurposed pharmacotherapies (e.g., bupropion).[31] The 2020 Surgeon General's report, *Smoking Cessation: A Report of the Surgeon General*, concluded that "the evidence is sufficient to infer that behavioral counseling and cessation medication interventions increase smoking cessation compared with self-help materials or no treatment." [31,p.10] In addition, as of 2021, more than three out of five U.S. adults who have ever smoked cigarettes have quit.[32] Although a majority of people who smoke cigarettes make a quit attempt each year, fewer than one-third use cessation medications approved by the U.S. Food and Drug Administration (FDA) or behavioral counseling to support their quit attempt.[31] The 2008 Public Health Service (PHS) Clinical Practice Guideline concludes that individual, group, and telephone counseling should be combined with first-line FDA approved medication (i.e., varenicline, NRT, and bupropion).[14] This recommendation applies to all people who smoke, including those with psychiatric disorders, such as AUDs and other SUDs, unless there is a medical contraindication or insufficient evidence for specific populations (pregnant women, people who use smokeless tobacco, people who smoke occasionally or lightly, and adolescents).[14] Further, the PHS Clinical Practice Guideline states, that "because alcohol is associated with relapse, the patient should consider limiting/abstaining from alcohol while quitting."[14,p.42] The U.S. Preventive Services Task Force (USPSTF) Recommendation Statement on smoking cessation concurs with the recommendation of combined pharmacotherapy and behavioral treatment, but does not make specific treatment recommendations for people who smoke and have an AUD and/or SUD.[33]

Smoking remains the leading cause of preventable death in the United States[34,35] and globally.[36] As discussed in chapter 2, more than half of adults who smoke or use tobacco also use alcohol. Although alcohol and substance use treatment programs often recognize the need to incorporate treatment for polysubstance use, tobacco use is a consistent exception.[37,38] Many people with AUDs express concern about their smoking or are interested in quitting smoking[39-44]; however, tobacco treatment is not consistently integrated into substance use treatment.[38,45] People with a past AUD are often not prompted by substance use treatment providers to make a quit attempt due to concerns about reducing AUD treatment success or because they are not advised and assisted to quit by alcohol treatment counselors, demonstrating another way that alcohol use can present a challenge to quitting smoking.[46] The Substance Abuse and Mental Health Services Administration (SAMHSA) has developed resources to aid providers in integrating tobacco cessation services into SUD treatment programs.[47-49] However, adoption of tobacco cessation services by SUD treatment facilities remains incomplete. For example, SAMHSA's 2024 National Substance Use and Mental Health Services Survey found that around two-thirds (72.6%) of U.S. substance use treatment facilities offered smoking/tobacco cessation counseling, and fewer than one-half offered NRT (43.2%) or nonnicotine smoking/tobacco cessation pharmacotherapies (39.1%).[50]

Chapter Structure

Conducting a literature review of combined treatment approaches for people who smoke cigarettes and use alcohol at different levels is challenging, due in large part to the heterogeneity observed from trial to trial regarding the many decision points (e.g., product types, outcome specification, candidate therapies, treatment setting, participant inclusion, and exclusion criteria). However, as this chapter will discuss, evidence is emerging on efficacious treatments for tobacco

and alcohol co-use. The primary goal of this chapter is to review and synthesize the evidence for treating tobacco use in the context of alcohol use, including among those with AUD. Conclusions are drawn from the existing evidence, while acknowledging limitations and identifying research needed to advance the field.

This chapter encompasses two major themes. The first theme addresses the neurobiological and behavioral mechanisms underlying tobacco smoking and alcohol consumption. This is not meant to duplicate material covered in chapter 6, but rather to highlight key mechanisms that drive co-use and could inform treatment targets. The second theme is a review of existing treatments for tobacco and alcohol use that have been evaluated in the literature, such as pharmacological monotherapies, combination pharmacotherapies, behavioral therapies, and combination behavioral and pharmacological therapies. People who have AUD may participate in mutual support groups, such as 12-step programs, to help them achieve and maintain abstinence from alcohol.[51] While many people with AUD have benefitted from these groups, this chapter focuses on the evidence-based treatments that have been used to treat tobacco and alcohol co-use, and no studies regarding the use of mutual support groups to treat co-use were found. Also, this chapter does not address forms of tobacco use other than cigarette smoking, as few studies have addressed concurrent treatment of tobacco products other than cigarettes with alcohol.

Understanding and Leveraging Neurobiological and Neurobehavioral Connections

Basic Underpinnings of Tobacco and Alcohol Dependence and Co-Use

Understanding the basic neurobiology and neurobehavioral connections between nicotine and ethanol is useful before discussing the literature on treatment implications. Preclinical data, and some clinical data, have established cross-tolerance between nicotine and ethanol, suggesting that as dosing escalates for ethanol, so it does for nicotine, and vice versa.[52-56] This cross-tolerance leads to increasing self-administration of each substance in order to obtain the same rewarding effects. When coadministered in rats, nicotine and ethanol cause changes in dopamine and acetylcholine levels throughout the nucleus accumbens and ventral tegmental area, and the nicotinic acetylcholine receptor system is activated via the mesolimbic system which expedites a path to dependence for both nicotine and ethanol.[28,57]

The nicotinic acetylcholine receptor system plays a critical role in the initiation, development, and maintenance of dependence to nicotine and alcohol.[28] For example, alcohol enhances the pleasurable effects of nicotine by increasing dopamine release in the brain's reward center, the nucleus accumbens, and by heightening the brain's sensitivity to nicotine through the activation of neuronal nicotinic acetylcholine receptors (nAChRs).[28,58] Conversely, nicotine may reduce some of the acute effects of alcohol consumption. Concurrent self-administration of the two substances has been described as a "pharmacological balancing act."[59] This may help explain why individuals may begin drinking alcohol, experience its depressant effects after a few drinks, and then smoke a cigarette to feel stimulated and counteract some of the sedative effects of alcohol consumption.[28,58] The primacy of the nicotinic acetylcholine receptor system when examining tobacco and alcohol co-use will come into focus further as the evidence for existing therapies is described in the section "Pharmacotherapy-Focused Approaches to Treat Tobacco and Alcohol Use."

Shared Genetic Vulnerability

Genetic evidence and growing specificity of neurobiological pathways and genetic markers could lead to further breakthroughs in developing personalized therapies to benefit individuals at disproportionate risk for smoking and alcohol drinking (described further in chapter 6). For example, genetic biomarkers can be used to distinguish between "fast" versus "slow" metabolizers of nicotine, and as a result, may enable healthcare providers to select a better suited pharmacotherapy based on the individual's rate of nicotine metabolism.[60] This type of research could advance personalized therapies among people who co-use tobacco and alcohol as well. However, as discussed in chapter 6, many of the genetic correlations identified as of 2026 concerning both tobacco and alcohol are not very strong. Further research is needed to determine whether there are suitable potential targets for interventions addressing tobacco and alcohol co-use based on genetic information and/or shared mechanistic pathways of initiation and maintenance.

Neurobehavioral Overlap

Several behavioral patterns emerge when an individual is smoking cigarettes and drinking alcohol that are critical to understanding the evaluation of treatment options. Alcohol use can escalate smoking by decreasing inhibition.[61] Using tobacco and alcohol together causes complementary rewarding effects; specifically, the use of alcohol acutely escalates tobacco smoking, while tobacco smoking increases consumption of alcohol.[62] Over time, this overlap leads to neurobiological changes that reinforce chronic use of these mutually reinforcing substances.[63] More specifically, these neurobiological changes and their underlying mechanisms are probably partially responsible for the bi-directional and complementary reinforcing effects of these substances (i.e., the use of one will increase the reinforcing effect of the other). Alcohol acutely enhances the subjective effects of nicotine, including pleasure and satisfaction.[64,65] Among people who drink heavily and smoke, alcohol use increases the urge to smoke and the rewarding effects of smoking, as well as acutely escalating tobacco smoking,[26,62,66-69] whereas tobacco smoking increases alcohol craving, decreases the subjective effects of alcohol, and increases alcohol consumption.[62,67,70] In fact, even individuals who are given a single, "priming" alcoholic beverage in a controlled setting find it more difficult to abstain from smoking after an episode of drinking has begun, compared to trying to abstain from smoking when not drinking.[26] In sum, tobacco and alcohol co-use may mitigate short-term negative effects and expedite a path to dependence on both substances.

Treatment Implications

Critical clinical outcomes of chronic tobacco and alcohol co-use are withdrawal and craving; these outcomes can drive a return to use and are the oft-cited targets for treatment development.[17,28,71] Return to use, or relapse, can often present as impulsive, driven by a need for immediate gratification, or compulsive, driven by a need to perform ritualistic behaviors. Once behavioral patterns develop and key neural circuits adapt to these new patterns, an inability to self-administer nicotine and alcohol is experienced as craving and withdrawal.[72] If the decision to use nicotine and/or alcohol is not deliberate but compulsive, it is often driven by environmental cues or external sources of stressors.[73] Compulsive tobacco and alcohol use behaviors are also often driven by heightened anxiety and stress and can lead to feelings of

relief from such negative emotional states during acute administration.[72,73] In part, impaired decision-making and compulsive self-administration make it very difficult for the individual to accurately assess the risk of tobacco and alcohol consumption.[73-75] Moreover, the compulsive nature of the substance use behavior makes it significantly more difficult to learn from the many negative consequences that result from intoxication.[76] Described succinctly by Nocente and colleagues (2013), "addiction to alcohol and tobacco is caused by pathologic brain neuroplasticity, in which drug seeking and compulsive intake are overlearned behaviors and relapse is due to unconscious working memory stimulated by impaired neural transmission."[28,p.454]

This bi-directional interaction poses various challenges for treating populations that co-use tobacco and alcohol because untreated dependence of one type can worsen the long-term outcomes of another untreated dependence.[7,8,77,78] Even moderate drinking can reduce smoking cessation success,[8] but the effects of heavier drinking appear stronger. Several studies on smoking cessation point to heavy episodic drinking (e.g., ≥4 drinks per day for women; ≥5 drinks per day for men) as a common predictor of poor smoking outcomes,[79-83] with more than 40% of all smoking lapses occurring concomitant to drinking.[84] Among individuals with a history of AUD or undergoing treatment for AUD, smoking is associated with greater urge to drink,[85,86] increased risk of relapse,[7,87] and greater consumption of drinks upon relapse.[86,88] Although the interplay between the use of tobacco and alcohol can create challenges for individuals trying to quit smoking or reduce their drinking, this relationship can also be leveraged to support their success in treatment, as quitting one substance might help with abstaining from the other.

Evidence Base for Smoking Cessation Therapies in the Presence of Alcohol Use

This section reviews clinical practice guidelines along with systematic reviews conducted as of 2026 on how available therapies for smoking cessation are moderated by alcohol use or whether these therapies have any specific guidelines or recommendations for treating co-use.

Clinical Practice Guidelines as of 2026

The PHS Clinical Practice Guideline, *Treating Tobacco Use and Dependence: 2008 Update,* states that smoking is a chronic disease that often requires multiple interventions and multiple cessation attempts, and existing therapies can significantly increase the odds of achieving and maintaining smoking abstinence.[14] The PHS Clinical Practice Guideline is consistent with the 2021 recommendations of USPSTF, which recommends that clinicians advise adults to stop using tobacco and provide behavioral interventions and FDA-approved pharmacotherapy for cessation to nonpregnant adults who use tobacco.[33] The PHS Clinical Practice Guideline emphasizes that health care providers and systems should incorporate screening for tobacco use, taking care to consistently document current tobacco use status and treating or referring individuals to treatment accordingly.[14] The Guideline also states that, across a broad range of populations, counseling combined with pharmacotherapy is more effective than either approach alone. Furthermore, even brief interventions can be effective for many individuals. Key elements of behavioral smoking cessation counseling include skills training, problem-solving, and social support, which are integral to individual and group counseling. These counseling approaches can be delivered in person and via telephone or digital tools (e.g., spoken conversation, text

messaging, smartphone applications). The Guideline recommends the use of FDA-approved medications to support cessation efforts unless medically contraindicated; these have been found to significantly improve long-term quit rates.[14] FDA-approved medications that should be considered are several NRTs (i.e., gum, lozenge, nasal spray, and patch) and two oral medications, varenicline and bupropion sustained-release. Neither the PHS Clinical Practice Guideline nor USPSTF makes specific recommendations for people who consume alcohol in low, moderate, or heavy amounts or for people who have AUD or other SUDs. However, their recommendations for treating tobacco use apply to individuals who consume alcohol, regardless of the amount or frequency consumed or the presence of AUD. Moreover, the PHS Clinical Practice Guideline notes that alcohol use is associated with relapse and thus patients who are trying to quit tobacco "should consider limiting/abstaining from alcohol while quitting." Guidelines from PHS, USPSTF, and other organizations related to treatment of tobacco and alcohol use are summarized in Table 7.1.

Table 7.1 Recommendations for Addressing Tobacco Use and Alcohol Use Disorder or Alcohol Use

Organization and recommendation	Year	Recommendations (excerpts): Screening	Recommendations (excerpts): Pharmacological and behavioral interventions	Recommendations (excerpts): Addressing co-use
Recommendations for treatment of alcohol use				
American Psychiatric Association (APA), Practice Guideline for the Pharmacological Treatment of Patients with Alcohol Use Disorder[89]	2018	The initial psychiatric evaluation of a patient with suspected AUD should include a quantitative behavioral measure to detect the presence of alcohol misuse and assess its severity.	Patients with AUD should have a documented, comprehensive, and person-centered treatment plan that includes evidence-based nonpharmacological and pharmacological treatments. Evidence-based psychotherapeutic treatments for AUD, including CBT, TSF, and MET play a major role in the treatment of AUD. APA recommends that naltrexone or acamprosate be offered to patients with moderate to severe AUD. APA suggests that disulfiram be offered to patients with moderate to severe AUD who meet certain criteria. APA suggests that topiramate or gabapentin be offered to patients with moderate to severe AUD who meet certain criteria.	The initial psychiatric evaluation of a patient with suspected AUD should include assessment of current and past use of tobacco and alcohol as well as any misuse of other substances, including prescribed or over-the-counter medications or supplements.
Department of Veterans Affairs (VA) and Department of Defense (DoD), Clinical Practice Guideline for the Management of Substance Use Disorders[90]	2021	For patients in general medical and mental health care settings, screen for unhealthy alcohol use periodically using the three-item AUDIT-C or single item SASQ.	For patients without documented AUD who screen positive for unhealthy alcohol use, provide a single initial brief intervention regarding alcohol-related risks and advising to abstain or drink within established limits for daily and weekly consumption. For patients with moderate to severe AUD, offer naltrexone or topiramate. For patients with moderate to severe AUD, offer acamprosate or disulfiram. For patients with moderate to severe AUD for whom first-line pharmacotherapy is contraindicated or ineffective, offer gabapentin. For patients with AUD, provide one or more of the following interventions, considering patient preference and availability: behavioral couples therapy, CBT, community reinforcement approach, MET, TSF.	Quitting tobacco use has clear benefits for improving health and decreasing mortality and is strongly encouraged for all patients with SUD. Consistently offering tobacco use disorder treatment throughout SUD treatment supports the principles of patient-centered care, shared decision-making, and recovery
Substance Abuse and Mental Health Services Administration (SAMHSA), Medication for the Treatment of Alcohol Use Disorder: A Brief Guide[91]	2015	Clinicians should routinely screen their patients for at-risk drinking, provide brief interventions as needed, and assess for AUD when indicated.	Clinicians should consider prescribing an FDA-approved medication (acamprosate calcium, disulfiram, oral naltrexone, extended-release injectable naltrexone) when treating a patient who is dependent on alcohol or who has stopped drinking but is experiencing problems including cravings or relapse. Medications should be prescribed as part of a comprehensive treatment approach that includes counseling and other psychosocial therapies (through referral to a psychiatrist, psychologist, or professional counselor) and social supports (through participation in Alcoholics Anonymous and other mutual-help programs).	The treatment plan for individuals with AUD should include a plan for treating co-occurring medical or psychiatric conditions and other SUDs, including smoking.

Table 7.1 (continued)

Organization and recommendation	Year	Recommendations (excerpts): Screening	Recommendations (excerpts): Pharmacological and behavioral interventions	Recommendations (excerpts): Addressing co-use
U.S. Preventive Services Task Force (USPSTF), Unhealthy Alcohol Use in Adolescents and Adults: Screening and Behavioral Counseling Interventions[92]	2018	All adults: Screen for unhealthy alcohol use in primary care settings in adults 18 years or older, including pregnant women.	All adults: Provide people engaged in risky or hazardous drinking with brief behavioral counseling interventions to reduce unhealthy alcohol use.	N/A
Recommendations for treatment of tobacco use				
American Society of Addiction Medicine (ASAM), Integrating Tobacco Use Disorder Intervention in Addiction Treatment[93]	2022	Screen all patients for tobacco use disorder. Patients entering addiction treatment should be screened for nicotine and tobacco use.	Offer evidence-based treatment to all patients with tobacco use disorder. • Address tobacco-related problems and goals in treatment planning. • Provide barrier-free access to evidence-based tobacco cessation interventions. Use motivational and harm reduction strategies for patients ambivalent about quitting. • Motivational interviewing should be used to support patients that are ambivalent about stopping nicotine or tobacco use. • Utilize harm reduction principles to reduce harms associated with tobacco use.	Correct misconceptions about tobacco use disorder and addiction treatment. Patients might be concerned that quitting tobacco could disrupt their efforts to stop other substance use. These beliefs are not supported by evidence, which demonstrates that tobacco cessation can help, not hinder, addiction treatment interventions. Clinicians should educate patients on the effectiveness of concurrent treatment for tobacco use disorder and other SUDs. Implement organizational policies to support tobacco cessation. • Implement tobacco-free policies in addiction treatment settings. • Educate and train staff to support tobacco-free policies.
National Comprehensive Cancer Network (NCCN), NCCN Clinical Practice Guidelines in Oncology, Smoking Cessation[94]	2022	Smoking status should be documented in the patient health record. Patient health records should be updated at regular intervals to indicate changes in smoking status, quit attempts made, and interventions utilized.	Treatment plans should be offered for all patients with cancer who smoke, regardless of cancer stage, site, prognosis, or treatment modalities. Treatment plans should include: evidence-based motivational strategies and behavior therapy (counseling), evidence-based pharmacotherapy, and close follow-up with retreatment as needed. Combining pharmacologic therapy and behavior therapy is the most effective approach for achieving and maintaining abstinence from smoking, or smoking reduction for those not ready to quit.	Providers should evaluate and document the risk of relapse for patients with more than 30 days' abstinence. Drug or alcohol use is a risk factor for relapse.

Table 7.1 (continued)

Organization and recommendation	Year	Recommendations (excerpts): Screening	Recommendations (excerpts): Pharmacological and behavioral interventions	Recommendations (excerpts): Addressing co-use
Public Health Service (PHS) Clinical Practice Guideline, Treating Tobacco Use and Dependence: 2008 Update[14]	2008	It is essential that clinicians and health care delivery systems consistently identify and document tobacco use status and treat every tobacco user seen in a health care setting.	Brief tobacco dependence treatment is effective. Clinicians should offer every patient who uses tobacco at least the brief treatments shown to be effective. Numerous effective medications are available for tobacco dependence, and clinicians should encourage their use by all patients attempting to quit smoking—except when medically contraindicated or with specific populations for which there is insufficient evidence of effectiveness (i.e., pregnant women, smokeless tobacco users, light smokers, and adolescents). Counseling and medication are effective when used by themselves for treating tobacco dependence. The combination of counseling and medication, however, is more effective than either alone. Thus, clinicians should encourage all individuals making a quit attempt to use both counseling and medication. If a tobacco user currently is unwilling to make a quit attempt, clinicians should use the motivational treatments shown to be effective in increasing future quit attempts.	Because alcohol is associated with relapse, the patient should consider limiting/abstaining from alcohol while quitting.
U.S. Preventive Services Task Force (USPSTF), Tobacco Smoking Cessation in Adults, Including Pregnant Persons: Interventions[95]	2021	Nonpregnant adults: Clinicians should ask all adults about tobacco use. Pregnant adults: Clinicians should ask all pregnant people about tobacco use.	Nonpregnant adults: Clinicians should advise adults to stop using tobacco and provide behavioral interventions and FDA-approved pharmacotherapy for cessation to nonpregnant adults who use tobacco. Pregnant adults: Clinicians should advise pregnant people to stop using tobacco and provide behavioral interventions for cessation to pregnant people who use tobacco.	N/A

Note. Table content is paraphrased from cited sources. APA = American Psychiatric Association, AUD = alcohol use disorder, AUDIT-C = Alcohol Use Disorders Identification Test-Concise, CBT = cognitive-behavioral therapy, FDA = Food and Drug Administration, MET = motivational enhancement therapy, N/A = Not applicable, SASQ = Single Item Alcohol Screening Questionnaire, SUD = substance use disorder, TSF = twelve-step facilitation.

Systematic Review Evidence as of 2026

Available systematic reviews contribute up-to-date evidence pertaining to how the use of alcohol affects smoking cessation outcomes, as well as how smoking cessation treatments delivered in the context of alcohol use treatment affect both smoking and alcohol use outcomes. Understanding smoking cessation in the context of alcohol use at various levels and frequencies, including among those who engage in heavy drinking or are in treatment for AUD, is an area of active research. This section summarizes the systematic reviews conducted as of 2026 that are relevant to the central questions of this chapter: (1) the extent to which smoking cessation interventions are affected by concurrent alcohol use and (2) whether addressing co-use of smoking and alcohol or AUD improves smoking cessation outcomes. The effect of providing smoking cessation treatment in the context of treatment for alcohol use on the alcohol use behavior is also addressed in some reviews and discussed in this chapter. Systematic reviews are summarized in Table 7.2.

Table 7.2 Treatment Approaches to Tobacco and Alcohol Cessation: Systematic Review Findings

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
Apollonio, Philipps, & Bero 2016[11] Meta-analysis 34 studies	January 1970–August 2016	Assess the impact of tobacco cessation interventions in the context of SUD treatment	**Language:** No restriction **Publication**: Peer reviewed and grey literature **Population**: Adults ages 15 and older undergoing inpatient or outpatient treatment for alcohol or other drug dependence, or in recovery from alcohol and other drug dependence **Study Design**: RCTs, cluster RCTs **Intervention**: Tobacco cessation interventions, including counseling, pharmacotherapy, and combination of both **Outcomes**: Point prevalence tobacco abstinence	**Outcomes**: Studies that did not report tobacco abstinence; studies that reported reduced smoking rather than abstinence	Point prevalence tobacco abstinence (intervention compared to placebo or usual care) *Pharmacotherapy alone*: RR = 1.88, 95% CI = 1.37–2.57 *Counseling alone*: RR = 1.33, 95% CI = 0.90–1.95 *Combined pharmacotherapy and counseling*: RR = 1.74, 95% CI = 1.39–2.18 *Interventions among people in SUD treatment*: RR = 1.99, 95% CI = 1.59–2.50 *Interventions among people in AUD treatment:* RR = 1.47, 95% CI = 1.20–1.81 *Interventions among people in other substance use treatment:* RR = 1.85, 95% CI = 1.43–2.40 *Interventions among people in SUD recovery*: RR = 1.33, 95% CI = 1.06–1.67 Point prevalence abstinence from alcohol and other drugs (intervention compared to placebo or usual care): RR = 0.97, 95% CI = 0.91–1.03

Table 7.2 (continued)

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
McCambridge & Jenkins 2008[96] Systematic review and meta-analysis 41 studies, 7 in meta-analysis	1995–2005	Examine the secondary effects of brief alcohol interventions on cigarette smoking	Language: English Publication: Peer-reviewed publications Population: Adults ages 18 and older Study Design: RCT or quasi-experimental Intervention: Brief alcohol interventions Outcomes: Smoking cessation	Studies not meeting inclusion criteria	Smoking cessation rate *Among 7 studies*: 9.4% (95% CI, 6.4%–12.3%) in intervention group; 10.3% (95% CI, 8.2%–12.3%) in control group, $p = .042$ *Among 6 studies (randomized studies only)*: 10.5% (95% CI, 7.3%–13.7%) in intervention group; 10.4% (95% CI, 8.2%–12.6%) in control group, $p = .42$

Table 7.2 (continued)

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
Prochaska et al. 2004[12] Meta-analysis 19 studies (12 of individuals in treatment, 7 of individuals in recovery)	January 1966–September 2003	Examine outcomes of smoking cessation interventions among individuals in current treatment or recovery for substance use (alcohol or drug use)	**Language**: Not specified **Publication**: Peer-reviewed articles, dissertations, and conference abstracts **Population**: Adults older than age 18 in treatment or recovery for substance use **Study Design**: RCTs **Intervention**: Smoking cessation interventions (pharmaceutical or behavioral) **Outcomes**: Quantitative assessment of smoking cessation	**Publication:** Multiple publications related to the same study **Study Design**: Quasi-experimental or single group design **Outcomes**: Outcomes other than smoking cessation (e.g., smoking reduction)	Smoking abstinence (intervention compared to control) *Post-treatment, participants in current substance use treatment*: RR = 2.03, 95% CI = 1.21–3.39 *Post-treatment, participants in recovery:* RR = 1.77, 95% CI = 1.37–2.30 *Post-treatment, all participants*: RR = 1.82, 95% CI = 1.45–2.29 *Long-term follow-up (≥6 months, participants in current substance use treatment):* RR = 1.00, 95% CI = 0.64–1.57 *Long-term follow-up (≥6 months, participants in recovery)*: RR = 1.31, 95% CI = 0.92–1.86 *Long-term follow-up (all participants):* RR = 1.18, 95% CI = 0.89–1.56 Abstinence from drugs and alcohol (intervention compared to control) *Post-treatment, participants in current substance use treatment*: RR = 1.10, 95% CI = 0.93–1.29 *Long-term follow-up (≥6 months, participants in current substance use treatment)*: RR = 1.25, 95% CI = 1.07–1.46

Table 7.2 (continued)

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
Thurgood et al. 2016[13] Systematic review 17 studies	January 1990–August 2014	Evaluate smoking cessation interventions during SUD treatment and recovery	**Language:** Not specified **Publication**: Peer reviewed and grey literature **Population**: Adults older than 18 who smoked and who had recently completed or were receiving inpatient or outpatient treatment for a SUD (drugs or alcohol) **Study Design**: RCTs **Intervention**: Pharmacological or nonpharmacological smoking cessation interventions **Outcomes**: Biochemically verified continuous smoking abstinence and 7-day point prevalence smoking abstinence at 6 or 12 months	**Study Design:** Trials comparing different timings, such as concurrent versus delayed implementation of the same intervention **Outcomes**: Studies with <6 months follow-up	A total of 5 out of 17 studies reported significant effects on smoking cessation at 6 or 12 months. Compared to placebo or usual care: • Nicotine patches improved continuous smoking abstinence at 6 months. • Nicotine gum improved continuous smoking abstinence at 12 months. • Counseling, contingency management, and relapse prevention improved continuous smoking abstinence at 6 and 12 months. • CBT + NRT improved point prevalence abstinence at 6 months. • Bupropion, NRT, counseling, and contingency management improved point prevalence abstinence at 6 months. Out of 10 studies that reported substance use outcomes, 2 observed differences in substance use outcomes between conditions, and 8 reported no difference between conditions. No study observed a negative effect of smoking cessation interventions on substance use outcomes.

Table 7.2 (continued)

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
van Amsterdam & van den Brink 2022[7] Systematic review 43 studies	1990–January 25, 2022	Investigate the potential moderating effect of smoking on behavioral and pharmacological treatment for AUD	**Language:** English, German, French, Dutch **Publication:** Studies available in PubMed **Population:** People diagnosed with AUD **Study Design:** Longitudinal studies, cohort studies, RCTs **Intervention:** Behavioral or pharmacological treatment of AUD; smoking cessation interventions for people with AUD **Outcomes:** Studies with information available on smoking status; studies that measured changes in drinking behaviors or relapse to drinking at follow-up in association with smoking status or changed smoking behavior	**Population:** People with comorbid psychiatric disorders or other SUDs (except tobacco or nicotine use); studies with a sample size of <40	Smoking cessation or decreased smoking frequency was associated with improved alcohol use outcomes in 16 out of 30 studies. Of 15 studies of behavioral treatment for AUD, smoking was associated with worse drinking outcomes in 10 studies. Of 15 studies of pharmacological treatment for AUD, smoking was associated with increased risk of relapse in 6 studies, and had no significant effect on alcohol use outcomes in 6 studies. A total of 13 studies included treatment for smoking cessation along with treatment for AUD. Only 1 study resulted in both reduced drinking and smoking; 6 other studies resulted in reduced smoking, but this was not associated with reduced drinking.

Table 7.2 (continued)

Study Publication type Number of studies included	Dates of studies included	Purpose	Inclusion criteria	Exclusion criteria	Main findings
van Amsterdam & van den Brink 2023[8] Systematic review 27 studies	Through August 19, 2022	Investigate the impact of alcohol consumption on smoking cessation	**Language:** Not specified **Publication**: Peer-reviewed literature **Population**: People who smoke **Study Design**: RCTs, naturalistic studies, and experimental laboratory studies **Intervention**: Smoking cessation **Outcomes**: Studies that recorded drinking status and changes in alcohol consumption among people who smoked tobacco	**Study Design**: Case reports, reviews **Population**: Studies in those who smoked and had other comorbid SUDs (except AUD) or other mental disorders; studies of specific groups like pregnant, unhoused, or incarcerated people or people with cancer; studies with a sample size of <40	Alcohol use was associated with less successful smoking cessation in 20 out of 27 studies about effects of alcohol use during attempts to quit smoking. In 19 out of 20 naturalistic studies with long-term follow-up, the duration of smoking abstinence was shorter among people with higher levels of alcohol consumption. In 12 out of 13 experimental studies about the effect of exposure to alcohol on smoking urges and craving, exposure to alcohol or alcohol cues among those who smoked induced a strong propensity to smoke.

Note. AUD = alcohol use disorder; CBT = cognitive-behavioral therapy; CI = confidence interval; NRT = nicotine replacement therapy; RCT = randomized controlled trial; RR = risk ratio; SUD = substance use disorder.

Smoking Cessation Therapies Delivered During Alcohol and Substance Use Treatment

Prochaska and colleagues (2004)[12] investigated whether smoking cessation treatment is effective when delivered during treatment for addiction or recovery, including AUD. The meta-analysis focused on randomized controlled trials (RCTs) of smoking cessation interventions in substance using populations published from January 1966 through September 2003. The analysis included 19 RCTs; 12 focused on adults in treatment for current addiction and 7 focused on smoking cessation in individuals in recovery from addiction (the authors note that they included studies of individuals with alcohol and/or other drug dependencies because of the frequency of comorbidity between substances and because treatment often addresses numerous addictions). The findings demonstrated that those who received smoking cessation interventions delivered during current addictions treatment were twice as likely to be abstinent from smoking at short-term follow-up as those in the control condition (pooled risk ratio [RR] = 2.03, 95% confidence interval [CI] = 1.21–3.39). Those in recovery who received smoking cessation treatment were 77% more likely to be abstinent from smoking at short-term follow-up as those in the control condition (pooled risk ratio [RR] = 1.77, 95% confidence interval [CI] = 1.37–2.30). When combining studies of participants in both addiction treatment and recovery, the summary RR was 1.82 (95% CI, 1.45–2.29).[12] The smoking cessation effect during treatment was more pronounced in the five studies that provided NRT as part of the smoking cessation intervention, compared with the six studies that provided only counseling. This finding is consistent with the 2008 PHS Clinical Practice Guideline, which recommends the provision of both an FDA-approved smoking medication that supports cessation and behavioral counseling.[14] However, at long term follow-up, the significant differences between intervention and control conditions was not sustained. The studies that focused on addictions treatment and recovery included participants with a history of any alcohol use problems; however, the authors did not present a subgroup analysis for those with alcohol and substance use compared with those with AUD alone. Additionally, a major conclusion of the review is that the provision of smoking cessation in the context of alcohol and SUD treatment or recovery did not compromise alcohol and substance use outcomes; rather, it appeared to enhance treatment and recovery outcomes. The authors note "the current findings suggest smoking cessation interventions may help with long-term sobriety even if long-term smoking cessation is not achieved."[12,p.1152]

Two systematic reviews published in 2016 also focused on the effectiveness of smoking cessation interventions delivered in the context of SUD treatment and recovery.[11,13] The first review, by Thurgood and colleagues (2016),[13] examined 17 studies conducted between 1990 and August 2014. This review included only RCTs of smoking cessation interventions with at least 6 months follow-up, involving adults (18 years or older) who smoked and who had recently completed or were currently receiving inpatient or outpatient treatment for a SUD. Five of the 17 studies reported significant effects on smoking cessation at follow-up of 6 or 12 months, specifically: (1) at 6 months, nicotine patches had a positive impact on continuous smoking abstinence; (2) at 12 months, nicotine gum had a positive impact on continuous smoking abstinence; (3) at 6 and 12 months, relapse prevention, counseling, and contingency management had a positive impact on continuous smoking abstinence; (4) at 6 months, NRT combined with cognitive behavioral therapy (CBT) had a positive impact on 7-day point prevalence smoking abstinence; and (5) at 6 months, bupropion, NRT, counseling, and contingency management had a positive impact on 7-day point prevalence smoking abstinence. Among the five RCTs that reported significant effects on smoking, four were focused on treatment of AUD and in some,

the primary outcome was alcohol and other substance use. Notably, no study found that treating smoking led to a deleterious impact on the other substance use outcomes, including those for alcohol. The authors characterized the evidence as tentative, noting that few studies reported sufficient detail to assess quality, and that additional studies are needed to strengthen the evidence base.

The second review, a meta-analysis conducted for the Cochrane Database of Systematic Reviews by Apollonio and colleagues (2016),[11] included 34 RCTs published between 1970 and August 2016. These studies were inclusive of both inpatient and outpatient settings, and both patients in treatment and in recovery from SUDs, including AUDs. The studies included 3 types of smoking cessation interventions: counseling only (11 studies), pharmacotherapy only (11 studies), and combination of counseling and pharmacotherapy (12 studies). Each study compared these treatment approaches with usual care or no intervention. Although counseling alone did not demonstrate a significant impact on smoking cessation (RR = 1.33, 95% CI = 0.90–1.95), pharmacotherapy alone significantly increased smoking cessation (RR = 1.88, 95% CI = 1.37–2.57), as did pharmacotherapy plus counseling (RR = 1.74, 95% CI = 1.39–2.18). Providing smoking cessation interventions increased the likelihood of smoking abstinence among both people currently in SUD treatment (RR = 1.99, 95% CI = 1.59–2.50) and those in recovery (RR = 1.33, 95% CI = 1.06–1.67). When the studies were divided by type of SUD being treated, both those treated for AUD (RR = 1.47, 95% CI = 1.20–1.81) and other SUD (RR = 1.85, 95% CI = 1.43–2.40) demonstrated significant effects regarding smoking cessation. Another important finding of this review is that the delivery of smoking cessation therapies to people in treatment for or recovery from AUD and/or SUD did not statistically significantly affect their alcohol and substance use treatment outcomes (RR = 0.97, 95% CI = 0.91–1.03).

Finally, a 2022 review by van Amsterdam and van den Brink[7] focused on smoking as an outcome moderator in the treatment of AUDs and concluded that smoking while in treatment predicts poorer alcohol treatment outcomes. Smoking cessation or decreased smoking frequency was associated with improved alcohol use outcomes (e.g., increased alcohol abstinence, reduced drinking, later relapse) in 16 out of 30 studies of behavioral or pharmacological treatment for AUD.

These four publications are the most up-to-date systematic reviews addressing the impact of smoking cessation delivered alongside alcohol and substance use treatment available as of 2026. Together, they provide strong evidence that (1) smoking cessation pharmacotherapies and counseling are effective for people in treatment for or recovery from AUD and (2) providing smoking cessation treatment does not harm alcohol use outcomes; conversely, the evidence suggests it may improve them. Collectively, these reviews provide foundational knowledge for developing future therapies targeting smoking and alcohol co-use.

Impact of Brief Interventions for Alcohol on Smoking Behavior

The development of brief interventions for alcohol use raises the question of whether these interventions may have a more generalized health promotion effect or influence related but nontargeted behaviors (e.g., also increase or decrease cigarette smoking). For example, because alcohol use can be a barrier to quitting smoking, if someone who is motivated to quit smoking also receives a brief intervention for their alcohol use and they successfully reduce or quit their

alcohol use, they could be more likely to quit smoking. In this regard, a 2008 systematic review and meta-analysis examined the potential secondary effects of brief interventions focused solely on alcohol use on smoking behavior outcomes in seven studies for which data were available on smoking status both at baseline and at follow-up.[96] This review found no secondary impact of brief interventions for alcohol use on smoking outcomes, including no difference in smoking cessation rates or frequency of smoking (mean cigarettes per day). The overall smoking cessation rate was 10.5%, regardless of treatment arm. However, this number should be interpreted with caution, as there was considerable heterogeneity in the efficacy of the interventions on reducing or changing smoking behavior across studies, and other factors may have contributed to the cessation rates observed in both study arms (which were higher than typical unassisted cessation rates). This review's primary conclusion was that providing a brief intervention for alcohol use will not also reduce smoking or increase cessation rates unless smoking is also actively targeted.

The Effects of Alcohol Use on Smoking Cessation Treatment

A 2023 systematic review[8] examined a related, but distinct question: What is the impact of alcohol consumption (without regard to alcohol treatment or recovery setting) on smoking cessation outcomes? The review identified 27 experimental or naturalistic studies published before August 19, 2022, that focused on smoking cessation which also measured alcohol use (e.g., nondrinking, heavy drinking, AUD).[8] In 20 of the 27 studies, alcohol use was associated with lower rates of smoking cessation. In 19 of 20 long-term follow-up studies, the duration of smoking abstinence was shorter among people with higher alcohol consumption compared with those who consumed less or no alcohol at all. Furthermore, 12 of 13 experimental studies demonstrated that consuming alcohol or even being exposed to a cue for alcohol was significantly associated with a propensity for smoking cigarettes. This review also found evidence that even moderate drinking was associated with greater difficulty resisting the opportunity to smoke cigarettes when consuming alcohol (compared with drinking a placebo beverage). These findings are consistent with robust evidence demonstrating the link between smoking and alcohol use neurobiologically and neurobehaviorally. The difficulty in resisting smoking after drinking alcohol is largely attributed to the disinhibiting and rewarding effects of alcohol, which can hinder attempts to quit smoking. The authors concluded that individuals interested in smoking cessation who drink alcohol should stop or substantially reduce their alcohol use before and during a quit attempt or consider an integrated approach to cease alcohol consumption and cigarette smoking.[8] These findings highlight the need for the investigation of integrated therapies.

Summary of Systematic Review Evidence

There is consistent evidence that engaging in smoking cessation treatment while in treatment for alcohol or SUD is effective for helping people quit smoking and does not jeopardize efficacious alcohol or substance use outcomes. Additionally, providing evidence-based smoking cessation treatment appears to be similarly effective for people who smoke who are receiving treatment for AUD and for people who are in recovery from AUD. People with a past diagnosis of AUD who try to quit smoking are just as likely to be successful as those with no history of AUD. A challenge remains that people in AUD treatment are often not prompted to make a smoking cessation attempt or offered smoking cessation treatment due to providers' concerns about interfering with alcohol treatment success. Still, a promising pattern has emerged from the

literature—that is, coordinated treatment of both tobacco and alcohol use can improve smoking cessation outcomes and does not harm the success of alcohol treatment outcomes. Indeed, coordinated treatment of both substances may enhance alcohol treatment outcomes.

Additionally, studies have found that alcohol consumption interferes with successful smoking cessation. This is an important finding considering that most people who smoke also drink: more than three-quarters of adult participants in the 2014–2015 and 2019–2020 National Alcohol Surveys who reported past-year tobacco use also reported past-year alcohol use, and more than one-half of participants in the 2015–2019 National Survey on Drug Use and Health who reported past-month cigarette smoking also reported past-month alcohol use (see chapter 2). The prevalence of alcohol use among people who smoke highlights the importance of advising people trying to quit smoking of the benefits of avoiding alcohol use during their quit attempt and the need for the investigation of integrated therapies for both tobacco and alcohol use.

Integrated Therapies for Smoking and Alcohol Use

This section focuses on two domains of treatment research—pharmacotherapy and behaviorally focused studies—for the concurrent treatment of smoking and alcohol use. This section builds on the systematic reviews, particularly the meta-analysis conducted by Apollonio and colleagues (2016)[11] and therefore describes results from some of the same studies and ones published after that review. As described earlier in this chapter, Apollonio and colleagues found that providing smoking cessation treatment to people with AUD in treatment or recovery is effective and does not undermine AUD treatment success.

The first part of this section summarizes pharmacotherapy approaches to treating smoking and alcohol co-use at various levels, often including heavy drinking or diagnosis of AUD. Although these trials always included a behavioral or psychosocial therapy in addition to pharmacotherapy, the focus of these trials was on testing a pharmacotherapy. Following that, this section then reviews and summarizes the findings of trials that primarily are designed to test the efficacy of a behavioral treatment. Although some of the trials also included medications, the focus of these trials was squarely on testing a behavioral or psychosocial intervention. Any exceptions are noted in the text. The search strategy and search strings used to identify relevant studies are described in Appendix 7A.

Pharmacotherapy-Focused Approaches to Treat Tobacco and Alcohol Use

This section provides an overview of findings from RCTs (Table 7.3A) and human laboratory experiments (Table 7.3B) published in or after 2002 that investigated the effectiveness of pharmacotherapies for treating tobacco and alcohol use concurrently. These studies typically included a behavioral or psychosocial adjunct that was held constant in all trial arms.

When considering the existing literature on testing pharmacotherapies (alongside behavioral interventions) to treat smoking and alcohol use, studies can be largely divided by the type of pharmacotherapy delivered. All studies were conducted in the United States, and virtually all are phase 2 clinical trials conducted in outpatient treatment research settings where the type of pharmacotherapy was the primary distinguishing factor across the trials. The types of pharmacotherapies delivered in the reviewed studies were FDA-approved medications for

smoking cessation (NRT, varenicline, and bupropion) and/or naltrexone, an FDA-approved medication for treating AUD. Although acamprosate is also an FDA-approved medication for treating AUD, no studies were identified that focused on acamprosate and reported both smoking and alcohol use outcomes. The primary outcome measures in these studies differed between smoking and alcohol use, with primary outcomes for smoking typically being complete abstinence, and primary outcomes for alcohol typically being reduction in alcohol use, such as reduction in number of drinks consumed or reduction in number of heavy drinking days.

Table 7.3A Selected Pharmacotherapy-Focused Treatment Studies Addressing Smoking and Alcohol Use Discussed in This Chapter

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Nicotine replacement therapy				
Cooney et al. 2009[97] *N* = 96 (45i, 51c) Outpatient alcohol treatment; Veterans Administration	Adults with a diagnosis of alcohol abuse or dependence and who smoked 15 or more cigarettes per day (at least a 3-year smoking history)	*All participants:* Received a behavioral alcohol and smoking treatment delivered in 3 months of weekly outpatient sessions followed by 3 monthly booster sessions. Alcohol treatment was based on CBT. *Intervention*: Open-label transdermal nicotine patch for 12 weeks (21 mg daily for 8 weeks, 14 mg daily for 2 weeks, 7 mg daily for 2 weeks) plus 2 mg nicotine gum for 24 weeks (ad libitum, 6-20 pieces per day) *Control*: Nicotine patch + placebo gum	3, 6, and 12 months	Smoking *Prolonged smoking abstinence, verified by breath carbon monoxide <10 ppm (intervention vs. control):* 3 months: 40% vs. 35% 6 months: 20% vs. 12% 12 months: 13% vs. 0%, $p < .01$ Alcohol use *Alcohol abstinence (intervention vs. control):* 3 months: 45% vs. 28% 6 months: 38% vs. 32% 12 months: 43% vs. 32% (Differences not statistically significant)
Varenicline				
King et al. 2022[98] *N* = 122 (61i, 61c) Outpatient clinics	Adults who smoked between 5 and 30 cigarettes per day and drank heavily (>14 drinks per week for men or >7 drinks per week for women, and 1 or more heavy drinking days per month [≥5 drinks per occasion for men, ≥4 drinks per occasion for women])	*All participants:* Received nicotine patch at manufacturer-recommended doses for 10 weeks (<10 cigarettes per day: 14 mg/day for 6 weeks, 7 mg/day for 4 weeks; 10+ cigarettes per day: 21 mg/day for 6 weeks, 14 mg/day for 2 weeks, 7 mg/day for 2 weeks) *Intervention*: Varenicline 2 mg/day for 12 weeks (starting at 0.5 mg once daily for 3 days, 0.5 mg twice daily for 4 days, and 1 mg twice daily starting on the quit date through 12 weeks) *Control*: Placebo pills twice daily for 12 weeks	Weeks 9–12	Smoking *Continuous cigarette abstinence (self-reported abstinence through weeks 9 to 12, verified by breath carbon monoxide ≤10 ppm [intervention vs. control] at week 12):* 44.3% vs. 27.9%, $p = .047$ Alcohol use *Weekly drinking days:* Among both groups, weekly drinking days decreased by 25%. There was no significant difference in weekly drinking days ($p = .81$) by group. *Weekly heavy drinking days (≥5 drinks per occasion for men, ≥4 drinks per occasion for women:* Among both groups, weekly heavy drinking days decreased by 54%.There was no significant difference in weekly heavy drinking days ($p = .57$) by group.

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Litten et al. 2013[99] *N* = 200 (99i, 101c) Outpatient clinics	Adults with DSM-IV diagnosis of alcohol dependence in the past year and who consumed ≥28 alcoholic drinks/week for women or ≥35 alcoholic drinks/week for men in the 5 weeks prior to randomization	*All participants*: Viewed 1 of 6 computerized modules for alcohol cessation at each of 5 clinic visits. *Intervention*: Varenicline 2 mg per day (0.5 mg once per day on days 1–3, 0.5 mg twice per day on days 4–7, 1 mg twice per day during weeks 2–13) *Control:* Placebo	Weeks 2–13 (alcohol); Weeks 6, 10, 14 (smoking)	Smoking *Cigarettes per day among participants who at baseline smoked ≥1 cigarettes per day in the last week (n = 78), averaged over Weeks 6, 10, 14 (intervention vs. control):* 7.4 vs. 11.7, *p* = .002; *d* = 0.73 Alcohol use *Weekly percent heavy drinking days (≥5 drinks/day for men, ≥4 drinks/day for women), Weeks 2–13 (intervention vs. control):* 37.9% vs. 48.4%, *p* = .03; *d* = 0.31
O'Malley et al. 2018[100] *N* = 131 (64i, 67c) Outpatient substance abuse treatment facilities in New York and Connecticut	Adults who smoked (daily smoking of ≥5 cigarettes or nondaily smoking at least 2 days per week) and who met DSM-IV criteria for alcohol dependence and reported heavy drinking 2 or more times a week for the previous 90 days and 7 or fewer consecutive days of abstinence	*Intervention*: Varenicline (0.5 mg once per day on days 1–3, 0.5 mg twice per day on days 4–7, 1 mg twice per day weeks 2–16) *Control*: Placebo	Last 8 weeks of treatment	Smoking *Self-reported abstinence in the last 28 days, verified by plasma cotinine levels of <6 ng/ml (intervention vs. control):* 13% vs. 0%, *p* = .003, *h* = .72 Alcohol use *Least-square mean difference in percent heavy drinking days (≥5 drinks/day for men, ≥4 drinks/day for women; intervention vs. control):* 1.69 vs. 1.77, *p* = .80 There was a significant medication by sex-by-time interaction (*p* = .03) such that varenicline, compared to placebo, resulted in a greater decrease in percent heavy drinking days in men (*d* = 0.45) than in women (*d* = -0.53).

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Bupropion				
Grant et al. 2007[101] *N* = 58 (28i, 30c) Substance use disorder treatment programs in Eastern Nebraska	Adults with DSM-IV criteria for alcohol dependence/abuse, attending SUD treatment programs, who smoked at least 20 cigarettes per day	*Intervention*: Nicotine patches+ bupropion (150 mg once per day for 3 days, then 150 mg twice per day for 60 days) *Control*: Nicotine patches + placebo	Week 4 Week 9 Month 6 (Week 26)	Smoking *7-day point prevalence abstinence (intervention vs. control)* Respondents: Week 4: 36% vs. 21%, *p* = .24 Week 9: 23% vs. 30%. *p* = .74 Month 6 (Week 26): 28% vs. 36%, *p* = .74 ITT (includes nonrespondents): Week 4: 30% vs. 18%, *p* = .36 Week 9: 17% vs. 25%. *p* = .52 Month 6 (Week 26): 17% vs. 29%, *p* = .35 Alcohol use *Continuous abstinence in past 30 days (intervention vs. control):* Week 4: 80% vs. 100%, *p* = .050 Week 9: 73% vs. 91%, *p* = .13 Month 6: 67% vs. 82%, *p* = .30 *Mean drinks per day in past 30 days (intervention vs. control):* Week 4: 1.3 vs. 0.04, *p* = .051 Week 9: 0.84 vs. 0.89, *p* = .12 Month 6: 1.4 vs. 0.91, *p* = .076 *Mean drinks per drinking day in past 30 days (intervention vs. control):* Week 4: 7.6 vs. 8.0, *p* = .80 Week 9: 5.1 vs. 8.5, *p* = .19 Month 6: 6.9 vs. 5.4, *p* = 1.0 *Mean percent days abstinent in past 30 days (intervention vs. control):* Week 4: 83% vs. 99%, *p* = .051 Week 9: 86% vs. 89%, *p* = .13 Month 6: 80% vs. 86%, *p* = .083

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Hays et al. 2009[102] *N* = 110 (56i, 54c) Outpatient clinic	Adults who smoked ≥20 cigarettes per day in the previous year who met DSM-IV criteria for alcohol abuse/dependence and who had at least 1 year of continuous abstinence from alcohol and substance use	*Intervention*: 8 weeks of tailored nicotine patch therapy; after confirmed smoking abstinence during week 8, bupropion sustained release 150 mg per day for 3 days, followed by 150 mg twice per day through week 52; brief behavioral counseling *Control*: 8 weeks of tailored nicotine patch therapy; after 8 weeks, placebo through week 52; brief behavioral counseling	Weeks 9–12, Week 16, Week 20, Week 24, Week 28, Week 32, Week 36, Week 40, Week 44, Week 48, Week 52, Week 53, Week 64, Week 76	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide <8 ppm (intervention vs. control):* Week 52: 39.3% vs. 40.7%, *p* = 1.0 Week 76: 35.7% vs. 37.0%, *p* = 1.0 *Continuous smoking abstinence (intervention vs. control):* Week 52: 41.1% vs. 40.7%, *p* = 1.0 Week 76: 37.5% vs. 38.9%, *p* = 1.0 Alcohol use *Relapse to alcohol:* 2 participants in the placebo group and 2 in the bupropion group relapsed to alcohol.
Oral naltrexone				
Fridberg et al. 2014[103] *N* = 315 (161i, 154c) Outpatient clinics in Chicago	Adults with smoking history of 10–40 cigarettes per day for at least 2 years Participants were categorized into 3 subgroups: heavy drinking and smoking (average of ≥2 heavy drinking episodes/month); moderate to light drinking and smoking (1 drink to <2 heavy drinking episodes/month on average), and those who smoked but did not drink.	*Intervention*: Naltrexone (50 mg titrated; 12.5 mg on day 1, 25 mg on days 2 and 3, and then 50 mg daily) + NRT (patch) + behavioral therapy *Control*: Placebo + NRT (patch) + behavioral therapy	12 weeks, 6 months, 12 months	Smoking *Prolonged abstinence, verified by breath carbon monoxide <10 ppm (intervention vs. control):* People who heavily drank and smoked: 32% vs. 14%, *p* = .023 People who moderately drank and smoked: 25% vs. 24%, *p* = .60 People who smoked but did not drink: 10% vs. 32%, *p* = .24 Among those assigned to the naltrexone intervention, the participants who drank heavily significantly increased smoking abstinence at 12 weeks compared to participants who did not drink heavily. The intervention effects on quit rates at 6 months or 12 months did not differ by drinking subgroup, *p*s > .39. Alcohol use *Drinks per week (intervention vs. control):*Compared to placebo, naltrexone significantly reduced weekly alcohol consumption among people who drank heavily and smoked (*p* = .005), but not among people who drank moderately to lightly (*p* = .26).

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Kahler et al. 2017[4] *N* = 150 (75i, 75c) Outpatient setting in Rhode Island	Adults who smoked cigarettes regularly for ≥1 year and smoked ≥5 cigarettes per day and currently reported drinking heavily at least once per month (≥4 drinks for women, ≥5 drinks for men)	*All participants:* Received 6 weeks of transdermal nicotine patch (14–21 mg for 2 weeks, 14 mg for 2 weeks, 7 mg for 2 weeks); 6 sessions of counseling on smoking and heavy alcohol use *Intervention*: 50 mg naltrexone (titrated from initial doses of 12.5 mg and 25 mg) for 10 weeks *Control*: Placebo	Weeks 2, 8, 16, 26	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤4 ppm and salivary cotinine ≤15 ng/ml (intervention vs. control)* Week 2: 37.3% vs. 28.0% Week 8: 20.0% vs. 22.7% Week 16: 16.0% vs. 18.7% Week 26: 12.0% vs. 12.0% There was no significant difference in 7-day point prevalence abstinence between treatment groups at any point (intervention vs. control). Alcohol use *Percent heavy drinking days (≥5 drinks/day for men, ≥4 drinks/day for women) (intervention vs. control):* Weeks 1 to 8: 15.3% vs. 12.4% Weeks 9 to 16: 18.7% vs. 18.7% Weeks 17 to 26: 17.0% vs. 16.0% There was no significant difference in mean percent heavy drinking days between treatment groups at any time point.

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
King et al. 2009[104] *N* = 78 (34i, 44c) Outpatient setting in Chicago	Adults who smoked, did not currently or previously meet criteria of alcohol dependence, and who reported drinking at least 1 alcoholic drink in the 2 weeks prior to study enrollment. Participants were further classified as heavy drinkers if they consumed ≥4 alcoholic drinks for women or ≥5 alcoholic drinks for men on 1 or more occasions in 2-week baseline period (*n* = *36*).	*All participants*: Received 1 month of nicotine patch (NicoDerm CQ) 21 mg daily for 2 weeks, 14 mg daily for week 3, and 7 mg daily for week 4; all participants received 6 individual weekly behavioral counseling sessions *Intervention*: 25 mg naltrexone 3 days prior to quit date, 50 mg naltrexone beginning on quit date and for the remaining 8 weeks *Control*: Placebo tablets on the same schedule	Weeks 1–4	Smoking *Smoking quit rates at Week 4, verified by breath carbon monoxide ≤10 ppm (intervention vs. control)*: Overall sample: 71% vs. 66%, *p* = .66 Heavy drinkers only: 80% vs. 52%, *p* = .09 Among heavy drinkers, heavy drinking days significantly mediated the relationship between medication and smoking quit rates (*p* < .05). Alcohol use *Mean total drinks per week at Week 2 (intervention vs. control):* 3.6 vs. 6.3 *Weekly heavy drinking, reporting 1 or more heavy drinking episodes (≥5 drinks on 1 occasion for men, ≥4 drinks on 1 occasion for women) at Week 2 (intervention vs. control):* 10% vs. 40%
King et al. 2013[105] *N* = 315 (161i, 154c) Outpatient clinics in Chicago	Adults who smoked 12–40 cigarettes daily for at least 2 years and reported a desire to quit smoking.	*All participants*: Received once weekly behavioral counseling for 4 weeks and open-label nicotine patch for 4 weeks starting on the quit date *Intervention*: 12.5 mg naltrexone 6 days prior to quit date, 25 mg for subsequent 2 days, 50 mg daily continuing through quit date and subsequent 12 weeks *Control:* Placebo tablets on the same schedule	1 week pre-quit, 4 weeks after quit date, 12 weeks after quit date	Smoking *Reduction in number of cigarettes smoked daily during pre-quit phase (intervention vs. control):* 4.21 fewer cigarettes daily vs. 2.93 fewer cigarettes daily (*p* = .02) *Mean number of cigarettes smoked per week 4 weeks after quit date (intervention vs. control)*: 2.6 vs. 5.2 Alcohol use *Mean total number of alcoholic drinks consumed (intervention vs. control):* Through week 4: 19.2 vs. 25.5 Through week 12: 53.1 vs. 68.4

Table 7.3A (continued)

Study *N* (intervention [i] vs. control [c]) Setting	Sample	Pharmacological intervention and control condition	Assessment points	Results
Varenicline and oral naltrexone				
Ray et al. 2021[106] *N* = 165 (83i, 82c) Outpatient research facility in Los Angeles	Adults who smoked daily (≥5 cigarettes/day) and drank heavily (for men: >14 drinks/week or ≥5 drinks per occasion at least once per month over the past 12 months; for women: >7 drinks/week or ≥4 drinks per occasion at least once per month over the past 12 months)	*All participants:* Received varenicline at 2 mg daily (titration schedule: 0.5 mg once daily for 3 days, 0.5 mg twice daily for 4 days, 1 mg twice daily for remainder of 12 weeks) and received a 30–45-minute counseling session. *Intervention*: Varenicline (schedule described above) + naltrexone at 50 mg daily (titration schedule: 25 mg once daily for 5 days, 50 mg once daily for remainder of 12 weeks). *Control*: Varenicline + placebo	12-week treatment phase with follow-up at 16 weeks and 26 weeks	Smoking *7-day point prevalence abstinence at week 26, verified by breath carbon monoxide ≤5 ppm*: Significantly higher in the varenicline plus placebo condition (n = 37 [45.1%]) compared to the varenicline plus naltrexone condition (n = 22 [26.5%]), p = .015 Alcohol use *Drinks per drinking day*: Trend toward a main effect favoring the combination of varenicline plus naltrexone over varenicline alone (β = 0.82, SE = 0.47, $t(1,484) = 1.76$, p = .080)

Note. CBT = cognitive behavioral therapy. DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; mg = milligrams; ml = milliliter; ng = nanogram; NRT = nicotine replacement therapy; ppm = parts per million; *SE* = standard error.

Table 7.3B Selected Pharmacotherapy-Focused Treatment Studies in a Laboratory Setting Discussed in this Chapter

Study *N*	Sample	Pharmacological intervention and control condition	Assessment points	Results
Varenicline and oral naltrexone combined				
Ray et al. 2014[107] *N* = 130	Adults (21–55 years old) who smoked daily (10 or more cigarettes per day) and currently reported heavy drinking (for men, >14 drinks per week or ≥5 drinks/occasion at least once per month over the past 12 months; for women, >7 drinks per week or ≥4 drinks/occasion at least once per month over past 12 months)	*Intervention Group 1*: Varenicline 0.5 mg daily on days 1–2, 1 mg daily on days 3–5, 2 mg (1 mg twice per day) on days 6–9 *Intervention Group 2*: Naltrexone 25 mg per day for 9 days *Intervention Group 3:* Varenicline + naltrexone (at titration described above) *Control*: Placebo	*Craving*: After 12 hours of cigarette abstinence, after receiving alcohol to reach a target BrAC of 0.060 g/dl, and after receiving the first cigarette of the day Cigarettes and drinks per day: Days 1–9	Smoking *Mean cigarette craving:* Post alcohol: 8.14 (varenicline alone), 7.93 (naltrexone alone), 6.88 (varenicline + naltrexone), 7.88 (placebo); significant difference between combination group vs. varenicline alone, combination group vs. naltrexone alone, and combination group vs. placebo ($p < .05$) Post cigarette: 3.00 (varenicline alone), 3.52 (naltrexone alone), 3.65 (varenicline + naltrexone), 3.91 (placebo); significant difference between combination group and placebo ($p < .05$) *Mean cigarettes per day:* Compared to placebo or naltrexone alone, the varenicline + naltrexone group had significantly fewer cigarettes per day ($p < .05$). Alcohol use *Mean alcohol craving:* Post alcohol: 17.31 (varenicline alone), 15.95 (naltrexone alone), 15.65 (varenicline + naltrexone), 15.71 (placebo); no significant difference between groups Post cigarette: 14.68 (varenicline alone), 13.44 (naltrexone alone), 14.44 (varenicline + naltrexone), 16.34 (placebo); no significant difference between groups *Mean drinks per drinking day:* Compared to placebo, the varenicline + naltrexone group and naltrexone alone group had significantly fewer drinks per drinking day ($p < .05$).

Table 7.3B (continued)

Study *N*	Sample	Pharmacological intervention and control condition	Assessment points	Results
Roberts et al. 2018[108] *N* = 30 (9 varenicline alone; 11 varenicline + naltrexone; 10 placebo)	Adults who smoked 10 or more cigarettes per day and drank heavily (≥14 drinks/week for men, ≥7 drinks/week for women, with at least 1 weekly binge drinking episode [≥5 drinks for men, ≥4 drinks for men] in past 30 days) and did not meet DSM-IV criteria for alcohol dependence	*Intervention Group 1*: Varenicline 2 mg daily for 7 days (0.5 mg once daily on days 1–2, 0.5 mg twice daily on days 3–5, 1 mg twice daily on days 6–7) *Intervention Group 2*: Varenicline 2 mg daily (same titration as above) + naltrexone 25 mg daily (0 mg daily for days 1–3, 12.5 mg daily on day 4, 25 mg daily on days 5–7) for 7 days *Control*: Placebo	Day 8 after alcohol dose to achieve blood alcohol level of 0.03 g/dL	Smoking *Smoking delay: Time to smoking after alcohol dose:* Varenicline vs. placebo: aHR = 0.13 (95% CI, 0.02–0.75), *p* = .023 Varenicline + naltrexone vs. placebo: aHR = 0.40 (95% CI, 0.12–1.37), *p* = .145 Varenicline + naltrexone vs. varenicline: aHR = 3.03 (95% CI, 0.72–12.71), *p* = .129 *Number of cigarettes smoked ad libitum:* Varenicline vs. placebo: aOR = 0.05 (95% CI, 0.01–0.48), *p* = .01 Varenicline + naltrexone vs. placebo: aOR = 0.09 (95% CI, 0.01–0.63), *p* = .02 Varenicline + naltrexone vs. varenicline: aOR = 1.76 (95% CI, 0.31–9.86), *p* = .52 Alcohol use There was no significant difference in alcohol craving (score on the Alcohol Urge Questionnaire) between groups (*p* > .11).

Note. aHR = adjusted hazard ratio; aOR = adjusted odds ratio; BrAC = Breath Alcohol Concentration; CI = confidence Interval; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; DSM-V = Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; g/dl = grams per deciliter; mg = milligram.

Nicotine Replacement Therapies

NRT is an evidence-based, FDA-approved treatment for smoking cessation that is available in several different forms (i.e., gum, lozenge, nasal spray, and patch).[31] The nicotine patch, lozenge, and gum are available over the counter. As of 2026, the nicotine inhaler is no longer available, as its sole manufacturer discontinued it due to shortage of ingredients needed to manufacture the inhaler.[109] Using 2 forms of NRT in combination is a best practice, evidence-based treatment approach. In this approach, a person uses a long-acting form (e.g., nicotine patch which delivers passive, continuous cutaneous dosing for up to 24 hours) and a short-acting form (e.g., nicotine lozenge which delivers the dose buccally over about 20 minutes) to actively manage acute cravings.[31] This treatment approach appears to also be effective for smoking cessation among people who smoke and have an AUD. An RCT conducted by Cooney and colleagues (2009)[97] involving 96 people who smoked, were diagnosed with alcohol abuse or dependence (DSM-IV criteria), and in the early stages of their outpatient treatment, found that nicotine gum augmented with a nicotine patch produced better smoking abstinence, but not better alcohol use outcomes, when compared with patch alone. The study specifically targeted individuals in the early stages of their outpatient treatment, providing insights into how NRT might work within this phase of recovery from alcohol dependence.

Varenicline

The introduction of varenicline for smoking cessation has arguably helped shape the science regarding tobacco and alcohol co-use significantly because of interest in the preclinical evidence indicating that it acts on brain pathways that are important for inducing the rewarding effects of both substances.[28] Varenicline, a selective α4β2 nicotinic acetylcholine receptor partial agonist, is a frontline medication to treat nicotine dependence[31]; provision of varenicline with concurrent counseling significantly increases smoking cessation rates.[14,110-112] The evidence base supports varenicline's viability as a treatment for smoking in those with co-occurring AUD. Although varenicline is not an FDA-approved medication for the treatment of alcohol use, clinical and preclinical studies have shown that varenicline significantly reduces alcohol cravings[99,113-115] and can concurrently decrease use of tobacco and alcohol, with more moderate treatment effects for alcohol use compared to the stronger treatment effects for tobacco use.[28,99,113,116-118] For example, McKee and colleagues (2009)[114] found that varenicline significantly reduced self-administration of alcohol and increased alcohol abstinence rates. Varenicline has also been shown to reduce subjective reinforcing effects of alcohol,[114] the number of drinks consumed,[115] and cue-induced alcohol relapse.[118,119] In addition, in animal studies, varenicline reduced seeking and consumption of alcohol in a dose-dependent manner,[118,120] promising signs for further development of the medication for the treatment of alcohol use. These findings confirm the underlying neurobiology and neurobehavioral evidence pointing to the importance of considering the nicotinic acetylcholine receptor system when developing therapies for people who use tobacco and alcohol.

Although varenicline has been shown to reduce alcohol use in some studies, the evidence of its effectiveness is mixed. In a Cochrane meta-analysis and systematic review of 34 studies of interventions for tobacco cessation among people in substance abuse treatment or recovery, Apollonio and colleagues (2016)[11] found no evidence to support the efficacy of varenicline for reducing alcohol use and a high level of study heterogeneity. However, this review only included two studies of varenicline. Results of a 2023 meta-analysis of 22 RCTs (N = 1,421) investigating

the effectiveness of varenicline for alcohol dependence were mixed.[115] Phimarn and colleagues (2023)[115] found that varenicline showed improvement compared to placebo in the percentage of abstinent days, drinks per day, drinks per drinking day, and alcohol craving, but not on abstinence rates, percentage of drinking days, or percentage of very heavy drinking days.[115]

With regard to the use of varenicline among people who use both tobacco and alcohol, in a 2021 systematic review and meta-analysis of 9 RCTs of the effects of pharmacological interventions on smoking cessation, Guo and colleagues (2021)[121] reported that varenicline was effective at promoting smoking cessation among people who had an AUD, while naltrexone and bupropion demonstrated no clear effect among the same population. Further, an RCT by O'Malley and colleagues (2018)[100] also found that varenicline combined with medical management (i.e., intervention that emphasized medication adherence for 4 weeks followed by support for changing drinking) was associated with reductions in drinking compared to placebo for patients who met alcohol dependence criteria (according to the DSM-IV-TR) and had comorbid cigarette smoking. Notably, they also found a sex difference, such that men benefited more than women from varenicline plus medical management.[100] One of the largest varenicline RCTs to demonstrate a reduction in drinking was performed by Litten and colleagues (2013),[99] with a sample of 200 individuals who were diagnosed with DSM-IV-TR alcohol dependence, 78 of whom also smoked cigarettes. They found that varenicline was associated with moderate—but significant—reductions in percent heavy drinking days (adjusted mean difference = 10.4; $d = 0.31$) while also decreasing cigarettes per day (i.e., 4.3 fewer cigarettes per day; $d = 0.73$) among people who smoked. The effects on smoking were large, while the effects on alcohol were consistent but more modest and similar in magnitude for people who smoked or did not smoke. These results are promising and indicate that varenicline may represent an important tool to leverage for people who smoke and have AUD; moreover, they are also an important signal to further evaluate sex as a variable and consider future therapies that boost quit rates among women. Another double-blind RCT testing the efficacy of varenicline for people who smoked and reported heavy drinking ($N = 122$) found that varenicline plus nicotine patch was more effective for smoking cessation than placebo pills plus nicotine patch at 9–12 weeks post-quit date (odds ratio [OR] = 2.20; 95% CI = 1.01–4.80; $p = .047$).[98] Participants in both conditions reduced any heavy drinking, with no statistical difference between groups in the amount of reduction. Notably, there were statistically more adverse events (digestive, sleep issues, headache) in the varenicline condition compared to the placebo condition and there were two serious adverse events in the varenicline condition and none in the placebo condition. It is important to note that varenicline can affect alcohol tolerance; extra care and monitoring is likely to be needed for these trials as well as in clinical practice. Additionally, the FDA package insert for varenicline warns of a potential medication interaction with alcohol and advises reducing alcohol use until the patient determines how varenicline affects their alcohol tolerance.[122]

Bupropion

Bupropion, an antidepressant with multiple pharmacologic effects including blocking the reuptake of dopamine and norepinephrine, has been approved for the treatment of cigarette smoking.[31] Only two studies were identified that tested the efficacy of bupropion for the reduction of both smoking and alcohol use. Grant and colleagues (2007)[101] conducted a small, double-blind, placebo-controlled trial ($N = 58$) of bupropion for the reduction of smoking and alcohol; all participants received nicotine patches and brief counseling. This trial found no

evidence of efficacy for bupropion at improving smoking cessation at 6-month follow-up, and discontinuing use of bupropion was common.[101] Participants who quit smoking also reduced their drinking, but this outcome did not vary by randomized group. A second double-blind trial conducted by Hays and colleagues[102] compared bupropion to placebo; all participants ($N = 110$) also received nicotine patches and brief counseling. This study found no statistically significant reductions in smoking and relapse to alcohol use among those who received bupropion compared with placebo. Based on these two studies, bupropion has not been found to be efficacious for the treatment of smoking and heavy drinking among people who engage in both behaviors. Moreover, due to the potential for increased seizure risk, bupropion is contraindicated among individuals undergoing abrupt discontinuation of alcohol use, and patients are encouraged to minimize or avoid alcohol consumption while taking bupropion.[123]

Oral Naltrexone

Naltrexone, an opioid receptor antagonist originally designed to treat opioid use disorder, has also been used as a therapy with modest efficacy for AUD.[124] Naltrexone is available both as an oral medication and in an extended-release intramuscular injectable form.[125] Oral naltrexone has been shown to be effective in reducing heavy drinking days and alcohol craving among people who smoke and are seeking AUD treatment[126,127] and is more effective than acamprosate, another FDA-approved medication for the treatment of AUD, at reducing alcohol use among people who smoke.[126] A 2013 Cochrane review and meta-analysis of naltrexone for smoking cessation that included 8 trials and more than 1,200 individuals found no evidence of an effect of naltrexone, either as an adjunct to NRT or as a stand-alone therapy, on long-term smoking abstinence.[128] Both the point estimate and the CIs suggest that an important clinically meaningful treatment effect is unlikely (naltrexone versus placebo: RR = 1.00, 95% CI = 0.66–1.51; naltrexone + NRT versus placebo + NRT: RR = 0.95, 95% CI = 0.70–1.30). However, that Cochrane review focused on naltrexone's utility as a smoking cessation therapy and not as a treatment for smoking and alcohol co-use. This section reviews the evidence for naltrexone as a potential treatment for people who smoke cigarettes and drink alcohol.

Two reviews focused on understanding naltrexone as a treatment for smoking and alcohol co-use have been published since 2013.[17,121] The Yardley and colleagues review (2015)[17] found evidence to support that using naltrexone, alone or in combination with varenicline or transdermal nicotine patches, is particularly effective in reducing alcohol use among individuals who smoke and are seeking treatment for AUD or who use alcohol heavily. They concluded that the combination of varenicline and oral naltrexone is more promising for treating co-use in people who smoke and drink heavily than NRT plus oral naltrexone. Yardley and colleagues' review[17] examined some naltrexone studies not included in a 2021 review by Guo and colleagues:[121] a study by O'Malley and colleagues (2008)[129] and a study by King and colleagues (2013).[105] O'Malley and colleagues reported naltrexone monotherapy demonstrated significantly higher total abstinence from alcohol (35%) compared with placebo (12%, $p = .03$) in a sample in which 59% of participants reported current smoking.[129] King and colleagues found reductions in both alcohol consumption and number of cigarettes smoked when naltrexone was compared to placebo and that naltrexone also reduced the urge to smoke.[105] This is consistent with human laboratory studies that have verified naltrexone's ability to attenuate cigarette craving and reduce smoking urges among people who both smoke and drink heavily.[130,131] Another double-blind clinical trial reported a similar pattern such that naltrexone reduced smoking among participants

(87% of whom reported current drinking).[132] A survival analysis revealed that compared with placebo, naltrexone significantly improved lapse-allowed quit rates over the course of the 12 weeks of treatment ($\chi^2_1 = 4.30$, $p < .05$). Additional evidence exists indicating that, when combined with NRT, naltrexone may be an effective therapy for reducing smoking.[133,134]

Some evidence suggests that oral naltrexone may be effective in reducing smoking among people who smoke and also drink heavily.[103,104,135] However, a 2021 review by Guo and colleagues identified three RCTs of people who smoked and had dependence on alcohol that did not find significant reductions in smoking.[121] Included in the review was an RCT conducted by Kahler and colleagues (2017)[4] which found that among people who smoked and drank heavily, naltrexone did not enhance participants' ability to quit smoking or drinking compared to placebo (all trial participants received the nicotine patch). However, there were significant reductions in both smoking and alcohol use compared to baseline. In an RCT conducted by Fridberg and colleagues (2014),[103] naltrexone reduced smoking urges and increased smoking abstinence only among people who smoked and drank heavily, but not among people who smoked and drank lightly or moderately, or who smoke and do not drink alcohol. The third study, an exploratory RCT by King and colleagues (2009),[104] found a similar pattern of results where naltrexone appeared to have some effect on increasing quit rates among people who drank heavily, but not among the entire study sample; however, these results were not statistically significant ($p = .09$). The potential greater efficacy of naltrexone in people who smoke and drink heavily, rather than any level of alcohol consumption, is not surprising, as naltrexone is FDA-approved for use in those with AUD. Moreover, although naltrexone did not significantly decrease smoking for the entire sample, the findings among people who smoked and drank heavily warrant further investigation of naltrexone to treat people who smoke and drink heavily or have an AUD. Although the evidence remains mixed for the specific role of naltrexone in the treatment of tobacco and alcohol use, there are promising signs that this medication may be effective.

Varenicline and Oral Naltrexone Combined

Given the separate effects seen for varenicline in treating tobacco use and naltrexone for treating heavy drinking and AUD, studies have also investigated the combination of the two to treat co-use. Varenicline has some level of cross-over effects from treating smoking as the primary target to alcohol consumption, but it has not proven effective enough at significantly reducing alcohol consumption to represent a stand-alone therapy for AUD. An experimental human laboratory study was conducted with 130 people who reported smoking 10 or more cigarettes/day and met the National Institute on Alcohol Abuse and Alcoholism definition of heavy drinking (for men, >14 drinks per week or ≥5 drinks per occasion at least once per month over the past 12 months; for women, >7 drinks per week or ≥4 drinks per occasion at least once per month).[107] The study found that the combination of varenicline (2 mg daily) and low-dose oral naltrexone (25 mg daily) was more effective than placebo, and at times monotherapy, in attenuating cigarette craving, reducing subjective feeling of "cigarette high" and "alcohol high," and reducing smoking and alcohol use during the 9-day medication period.[107] Roberts and colleagues (2018)[108] conducted a human laboratory study ($N = 30$) that compared placebo,

varenicline (2 mg/day), and varenicline (2 mg/day) plus low-dose naltrexone (25 mg/day) on cigarette smoking outcomes and alcohol craving and other subjective effects. There was a reduction in the number of cigarettes smoked in the two groups that received varenicline compared to placebo, but the administration of low-dose naltrexone did not have a significant effect on number of cigarettes smoked or on alcohol craving.

Ray and colleagues (2021)[106] also examined the use of varenicline and oral naltrexone in an RCT comparing varenicline (2 mg/day) to varenicline (2 mg/day) plus oral naltrexone (50 mg/day) in people who smoke and drink heavily ($N = 165$). They found the combination to be effective in reducing risky drinking levels, with trends toward a lower number of drinks per day during treatment; however, these results were not statistically significant ($p = .80$).[106] However, this study also found that varenicline monotherapy was more effective in promoting smoking cessation at 26-week follow-up than varenicline plus oral naltrexone.[106]

Cytisine

Cytisine (also called cytisinicline), a compound derived from the *Cytisus laburnum* plant, is a partial agonist of the $\alpha_4\beta_2$ nicotinic acetylcholine receptor and has been used to treat tobacco dependence in Europe for decades.[136] Several RCTs conducted outside of the United States (mainly Europe and New Zealand) have found cytisine to be effective for smoking cessation.[137,138] A 2024 meta-analysis of RCTs comparing cytisine to placebo and/or FDA-approved pharmacotherapies found that cytisine was more effective but had more side effects than placebo and NRT, but was comparable in effectiveness and had fewer side effects than varenicline.[137] In the last decade, research on cytisine in the United States has increased, and studies have demonstrated efficacy for smoking cessation compared to placebo.[139-141] The RCTs conducted in the United States were funded by Achieve Life Sciences to inform a new drug application to the FDA for approval of cytisinicline medication for smoking cessation.[142,143] As of 2026, there was one published study investigating the efficacy of cytisine to treat both tobacco and alcohol use. Tindle and colleagues (2022)[144] conducted an RCT to test the effectiveness of varenicline, cytisine, and NRT for treating daily smoking and risky drinking among people with HIV in Russia. In this four-arm trial (active varenicline and placebo NRT, placebo varenicline and active NRT, active cytisine and placebo NRT, and placebo cytisine and active NRT), all of the active medications were equally successful for smoking cessation and alcohol reduction (number of heavy drinking days in the past 30 days and 30-day alcohol abstinence). In post hoc analyses, participants in all arms who quit smoking at all follow-up timepoints reported fewer heavy drinking days compared with those who continued smoking.[144] This finding suggests that further investigation of cytisine as a treatment for tobacco and alcohol co-use may be warranted.

Summary: Pharmacotherapy-Focused Approaches to Treat Tobacco and Alcohol Use

Effective pharmacological treatments exist for treating tobacco use and alcohol dependence separately, and some studies have investigated the effects of pharmacological treatments on both smoking and alcohol outcomes. Both of the FDA-approved medication classes for smoking cessation (i.e., NRT and the oral medications varenicline and bupropion, which do not contain nicotine) have been evaluated for their effects on drinking when treating smoking, with unclear evidence for reducing alcohol use. One of the approved smoking cessation medications,

varenicline, has been independently investigated for treating alcohol use, with some studies finding it reduced alcohol cravings and alcohol use and others finding no effect. Studies have also evaluated the effects of naltrexone, an FDA-approved medication for the treatment of AUD, on smoking and alcohol use outcomes. Some evidence suggests that oral naltrexone may be effective in treating smoking among people who smoke and also drink heavily. In sum, as found in previous reviews, pharmacotherapy and pharmacotherapy with counseling appears to be effective in the context of treatment for AUD and does not negatively affect AUD treatment outcomes.

Behavioral Treatments for Smoking Cessation in the Context of Alcohol Use (With or Without Medications)

This section describes studies focused on testing the efficacy of behavioral smoking cessation interventions delivered in the context of alcohol treatment, and interventions testing integrated tobacco and alcohol treatment, with or without a pharmacological treatment, among RCTs published between 2002 and 2024. As stated above, this section builds on the findings of the systematic reviews, especially the meta-analysis by Apollonio and colleagues (2016)[11] that examined smoking cessation efficacy in people with AUD who received treatment. As such, this section includes discussion of some studies that have been included in the previously published systematic reviews and studies published since those reviews (summarized in Table 7.4).[83,145-162] The search strategy and inclusion criteria are described in Appendix 7A. The studies that tested combined behavioral interventions targeting smoking and alcohol behaviors are categorized by setting: (1) intensive therapy for substance use in inpatient or outpatient clinical settings, and (2) non-substance-use treatment settings.[11-13] Notably, as of 2026 there has not been a review focused on combined tobacco and alcohol behavioral treatment studies that took place in settings outside of substance use and/or other clinical treatment settings

Tobacco and Alcohol Use Treatment in Inpatient and Outpatient Settings

This section summarizes the literature pertaining to smoking cessation interventions in the context of alcohol treatment that are delivered in intensive substance use treatment settings. Since the most recent systematic reviews summarizing the literature on interventions to treat smoking and AUD in substance use treatment settings,[11,13] two new RCTs were published.[148,151] Cooney and colleagues (2017)[148] assessed the impact of contingency management (CM) added to a CBT and NRT intervention compared to the intervention without CM. They found that the intervention with the addition of CM was more effective than the intervention alone at the end of treatment (60% vs. 29%, $p = .004$), However, the effect was not sustained at follow-up time points. Das and colleagues (2017)[151] examined the efficacy of a smoking cessation intervention with stage-tailored counseling and NRT compared to usual care (brief counseling and NRT) in the context of inpatient intensive alcohol or substance use treatment. They found the stage-tailored intervention was more effective than usual care at 12-month follow-up (RR = 2.01, 95% CI = 1.05–3.83, $p = .03$) with no differences in reported past-30-day alcohol use.[151]

Smoking cessation in the context of AUD treatment studies have been conducted specifically with people receiving inpatient treatment for SUD,[151,155,162] alcohol detoxification,[159] or general psychiatric disorders.[151] Across studies reviewed here, the smoking cessation treatment approaches provided were motivational interviewing[162] and stage-tailored interventions,[151,155] brief advice,[162] and CBT.[159] All studies provided participants with NRT.[151,155,159,162] Across the 4 studies, smoking cessation rates (7-day point prevalence abstinence) at 6 months ranged from 0%[159] to 13.5%.[151] The alcohol outcomes assessed varied across all studies. Key findings included: alcohol abstinence at 6 months (47.6% vs. 39.1% in the intervention group and control group, respectively, *p* value not presented)[159]; alcohol abstinence at 12 months (42.9% vs. 52.7% in the intervention group and control group, respectively, difference not statistically significant)[151]; 6-months sustained abstinence assessed at 6, 12, and 18 months after study enrollment, with concurrent smoking cessation treatment having lower alcohol abstinence at 6 months (41% versus 56% in concurrent versus delayed implementation, respectively), but with statistically equivalent abstinence rates by 12 and 18 months[155]; and number of days with any alcohol use (data not presented).[162]

Two trials considered the impact of a concurrent or delayed smoking cessation intervention in the context of AUD treatment; both interventions provided CBT-based counseling and NRT.[155,160] Joseph and colleagues (2004)[155] found greater engagement with the smoking cessation treatment in the concurrent delivery group compared to delayed group, but no differences between groups in 7-day point prevalence abstinence at 18-month follow-up (12.4% vs 13.7%, respectively, $p > .05$). Nieva and colleagues (2011)[160] also did not find a statistically significant difference in 7-day point prevalence smoking abstinence between the two groups at 6-month follow-up (13.7% vs. 2.4%, $p > .05$). Notably, with regard to the alcohol outcomes assessed, differing definitions make comparisons of the results challenging. Although Nieva and colleagues found no differences in alcohol abstinence at any of the follow-up timepoints,[160] Joseph and colleagues found statistically significantly lower rates of sustained alcohol abstinence at 6-month follow-up ($p = .001$), but the differences were not statistically significant at the 12- and 18-month follow-up timepoints.[155] As noted by Joseph and colleagues, this finding was contrary to the extant literature at that time.[155] Additionally, as noted in systematic reviews,[11,12] there are only a few studies that used a concurrent vs. delayed intervention delivery design, and the overall literature demonstrates that concurrent delivery of smoking cessation and treatment for AUD does not harm alcohol treatment outcomes.

Studies conducted with military veterans examined smoking cessation treatment in the context of U.S. Department of Veterans Affairs outpatient alcohol and substance use treatment programs. The smoking cessation behavioral treatment approaches tested varied across the studies, from 3-session intensive smoking cessation counseling in accordance with the PHS Clinical Practice Guideline[149] to 5 sessions of group-based counseling,[154] to 16 CBT-based sessions plus contingency management.[147] All interventions included the provision of nicotine patches, with two interventions offering NRT in gum or lozenge form as well.[147,154] All but one of the four studies biochemically verified smoking abstinence. Three out of the four studies with veterans found that the intervention resulted in significantly higher cigarette smoking abstinence rates overall or early follow-up periods compared to the control group.[147-149] However, at 6-month follow-up, 7-day abstinence rates were not statistically different between the intervention and control groups and ranged from 9.1% to 12% in the intervention groups.[148,149] Yet, the quit rates these studies observed among veterans in both intervention and control conditions were

comparable to quit rates seen among general populations who receive similar smoking cessation treatment.[141] In addition, three of the four studies indicated that alcohol treatment was not affected by the provision of smoking cessation treatment.[147-149] Only one small study ($N = 42$) reported higher alcohol use in the intervention group than in the control group, but these differences were not statistically significant.[154]

Overall, among participants in an alcohol/substance use treatment setting reviewed above,[147-149,151,154,155,159,160,162] the smoking cessation treatment results were somewhat mixed, with several studies finding a positive effect of the behavioral smoking cessation intervention and some finding no effect compared to the comparison condition (often usual care). Notably, among veterans in outpatient treatment settings, the provision of a behavioral smoking cessation treatment (together with NRT) resulted in increased smoking abstinence in the short term but not at longer-term follow-up[147-149,154]; while in inpatient psychiatric care, abstinence rates were significantly higher for patients who received a behavioral smoking cessation intervention.[151] Consistent with previous reviews of the literature,[11,12] this summary finds that in these treatment settings, incorporating behavioral smoking cessation treatment (with provision of NRT) into alcohol use treatment can be effective for helping people quit smoking and does not negatively affect the success of the alcohol use treatment. As discussed in chapter 9, smokefree policies in substance use treatment settings also do not deter individuals from seeking treatment for their substance use. Moreover, the benefits of concurrent smoking cessation and AUD treatments on alcohol treatment outcomes may be indirect. Although two of the most recently conducted (2017) studies showed no statistically significant difference in alcohol use by condition, participants who did quit smoking during the trials were less likely to report alcohol use at follow-up.[148,151] Specifically, Das and colleagues (2017),[151] found that having quit smoking was associated with past 30-day alcohol abstinence at 12-month follow-up. Cooney and colleagues (2017)[148] found that having quit smoking at the end of treatment mediated the relationship between the treatment (CBT and NRT plus contingency management) and alcohol abstinence at 1-month follow-up.

Table 7.4 Selected Studies of Behavioral Interventions for Smoking Cessation and Alcohol Use Targeting Adults Discussed in This Chapter

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Interventions in inpatient and outpatient substance use treatment settings				
Carmody et al. 2012[147] *N* = 162 (82i, 80c) Outpatient clinic; Veterans Administration In-person USA	Adult veterans who smoked at least 5 cigarettes per day, were abstinent from alcohol for at least 7 days, and were interested in quitting smoking	*Theoretical approach*: CBT *Intervention:* NRT + 16 sessions of individual counseling for smoking cessation, incorporating mood management Session 1–5: Focused on the health consequences of smoking and preparation for the quit date Session 6–16: Modules addressing mood management skills, cognitive restructuring, behavioral activation, social support, and weight management *Comparison:* NRT + Usual care with referral to the medical center's smoking cessation clinic	12 weeks; 26 weeks (end of intervention); 38 weeks; and 52 weeks 16 weeks of nicotine patches 26 weeks of nicotine lozenges	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide <10 ppm):* Exact abstinence rates not presented. *p* value for condition = .0229, favoring intervention. Alcohol *Drinking during past 30 days:* Exact abstinence rates not presented. *p* value for condition = .08

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Cooney et al. 2007[149] *N* = 118 (55i, 63c) Outpatient clinic; Veterans Administration In-person USA	Adults who met DSM-IV criteria for nicotine and alcohol dependence during the past 3 months and smoked at least 10 cigarettes per day	*Theoretical approach*: CBT *All participants*: Completed a 3-week intensive outpatient alcohol and substance use treatment program. *Intervention:* 3 individual counseling sessions (60 minutes each) with PHS Clinical Practice Guideline. Content included setting a targeted quit date (~1 week into 3-week program), identifying antecedents for smoking urges and behavior, developing behavioral and cognitive coping skills for high-risk smoking situations, and acquiring extra-treatment support for smoking cessation. On the scheduled quit date, participants were provided NRT. *Comparison:* 15-minute smoking cessation treatment session + 5-minute follow-up within 3 days of quit date. No NRT provided.	2 weeks post-discharge, 3- and 6-months post-discharge 4 weeks of 21 mg nicotine patches, 2 weeks of 14 mg, and 2 weeks of 7 mg.	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide <10 ppm (intervention vs. comparison):* Week 2: 27.5% vs. 6.6%, $p < .01$ Month 3: 4.3% vs. 5.7%, $p > .05$ Month 6: 9.1% vs. 2.1%, $p > .05$ Alcohol *Proportion heavy drinking days (≥6 drinks for men, ≥4 drinks for women; intervention vs. comparison):* Week 2: 0.04 vs. 0.02, $p > .05$ Month 3: 0.07 vs. 0.10, $p > .05$ Month 6: 0.02 vs. 0.01, $p > .05$ *For alcohol abstinence (intervention vs. comparison):* Week 2: 85.5% vs. 88.7%, $p > .05$ Month 3: 71.4% vs. 64.8%, $p > .05$ Month 6: 58.7% vs. 61.2%, $p > .05$

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Cooney et al. 2017[148] *N* = 83 (42i, 41c) Outpatient clinic; Veterans Administration In-person USA	Adult veterans who met DSM-IV criteria for alcohol abuse or dependence, last alcoholic drink within 90 days, smoked 10 or more cigarettes per day, had 3 years of smoking history, and completed substance detoxification prior to randomization	*Theoretical approach:* CBT *All participants*: Enrolled in a 3-week intensive outpatient alcohol and substance use treatment program. CBT (120 minutes total) focused on preparation to quit smoking, coping with nicotine withdrawal, and relapse prevention. *Intervention:* Combination intervention: CBT + NRT + contingency management. CBT was delivered concurrently with alcohol and drug treatment program. Twelve, 10-minute CBT sessions daily, corresponding with daily contingency management sessions. Contingency management provided escalating financial incentives for biochemically verified smoking abstinence (starting at $5, potential total maximum of $140 over 8 days). *Comparison:* Combination intervention: CBT + NRT. CBT delivered concurrently with alcohol treatment program. Three, 40-minute CBT sessions at weekly intervals.	End of behavioral treatment (~1.5 weeks after quit date), 1 month, and 6 months after quit date 8 weeks of nicotine patches; 21 mg for 4 weeks. 14 mg for 2 weeks, 7 mg for 2 weeks	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤5 ppm (intervention vs. comparison):* End of treatment: 60% vs. 29%, *p* = .004 Month 1: 41% vs. 27%, *p* = .21 Month 6: 12% vs. 5%, *p* = .27 Alcohol *Abstinence:* Exact abstinence rates at the end of treatment and at each follow-up were not presented; *p* values at all points > .05.

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Das et al. 2017[151] *N* = 216 (105i, 111c) Inpatient facility In-person USA	Adults who smoked at least 5 cigarettes daily; these results restricted to those who had a SUD (positive substance abuse screening test score ≥3 or positive Alcohol Use Disorders Identification Test (AUDIT) score ≥8)	*Theoretical approach:* Transtheoretical model (stages of change) *Intervention:* Usual care plus the Transtheoretical model stage tailored computer-delivered intervention with printed report, stage-matched treatment manual, and on-unit individual cessation counseling. Offered 10-week course of nicotine patches available at time of hospital discharge or anytime up to 6-month follow-up assessment when ready to quit. The 15–30-minute counseling provided motivational enhancement and stage-tailored strategies. Repeated the computer interventions at 3 and 6 months posthospitalization. *Comparison:* Usual care which included brief advice to quit, NRT during their smokefree hospitalization, and a quit smoking pamphlet	3 months; 6 months; 12 months Nicotine patches in hospital and 10-week course for intervention.	Tobacco *7-day point prevalence abstinence, verified by breath carbon monoxide ≤10 ppm (intervention vs. comparison):* Month 3: 12.5% vs. 3.7% Month 6: 13.5% vs. 5.5% Month 12: 22.1% vs. 11.0% Tobacco abstinence over 12 months was higher in the intervention condition than in the comparison condition (OR = 2.30, 95% CI = 1.08–4.90, $p < .05$). Alcohol *Alcohol abstinence in past 30 days (intervention vs. comparison):* Month 12: 42.9% vs. 52.7%, (RR = 1.21, 95% CI, = 0.88–1.67, $p > .05$)

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Grant et al. 2003[154] *N* = 42 (21i, 21c) Outpatient clinic; Veterans Administration In-person USA	Adult veterans who met DSM-IV criteria for alcohol abuse/dependence, smoked at least 10 cigarettes per day, and were enrolled in outpatient treatment for alcohol use	*Theoretical approach:* N/A *All participants*: Engaged in the same outpatient alcohol treatment program *Intervention:* 5 weeks of 30-min education and 1-hour group therapy sessions, education sessions addressed nicotine dependence, nicotine withdrawal, and other behavioral conditions. An 8-week trial of NRT gum or patch was offered. *Comparison:* Participants were not discouraged from attempting to discontinue smoking; had access to smoking cessation resources at the Medical Center (1 educational session and NRT).	2 weeks, 1 month, 6 months, 12 months An 8-week trial of nicotine gum or patch offered	Smoking *Past 7-day smoking (intervention vs. comparison):* Week 2: 78.6% vs. 87.5% Month 1: 63.6% vs. 86.7% Month 6: 88.9% vs. 92.9% Month 12: 100% vs. 93.3% $p > .20$ (all) Alcohol *Any drinking (intervention vs. comparison):* Week 2: 13.3% vs. 6.3%, Month 1: 38.5% vs. 17.6% Month 6: 38.5% vs. 35.3% Month 12: 53.8% vs. 43.8% $p > .20$ (all) *Having more than 1 drink (intervention vs. comparison):* Week 2: 6.7% vs. 0%, ($p > .20$) Month 1: 33.3% vs. 0% ($p = .024$) Month 6: 33.3% vs. 13.3% ($p > .20$) Month 12: 53.8% vs. 40.0% ($p > .20$) *Heavy drinking in the past 2 days (intervention vs. comparison):* Week 2: 0.0% vs. 0.0%, Month 1: 16.7% vs. 6.3% Month 6: 10.0% vs. 18.8% Month 12: 27.3% vs. 26.7% $p > .20$ (all)

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Mueller et al. 2012[159] *N* = 103 (53i, 50c) Inpatient facility In-person Switzerland	Adults (ages 18–65) who were alcohol dependent according to ICD-10 criteria, currently smoked tobacco, and were motivated to quit or reduce their tobacco use	*Theoretical approach:* CBT *Intervention:* Five, 30-minute group sessions. Received a modified version of a manual CBT program with additional information regarding the harm of concomitant use of tobacco and alcohol. Sessions included motivational processes, education about nicotine and its effects, psychological factors in SUD, and elements of relapse prevention. Nicotine patches offered during inpatient stay. *Comparison:* Practiced the relaxation technique autogenic training. Nicotine patches offered during inpatient stay.	End of intervention; 6 months Nicotine patches (21 mg).	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide <10 ppm and urinary cotinine level (intervention vs. comparison):* End of intervention: 7.5% vs. 4%, *p* value not presented Month 6: 0% vs. 6%, *p* value not presented Alcohol *Month 6 alcohol abstinence (of n = 44; intervention vs. comparison):* 47.6% vs. 39.1%, *p* value not presented

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Nieva et al. 2011[160] *N* = 92 (51i, 41c) Outpatient clinic In-person Spain	Adults (ages 18–65) who met DSM-IV criteria for nicotine and alcohol dependence, smoked more than 5 cigarettes per day for more than 1 year, and wanted to quit drinking and smoking	*Theoretical approach:* CBT *Intervention:* Simultaneous group started smoking cessation treatment on Day 1 of enrollment. Ten, 30- to 45-minute individual sessions based on CBT with emphasis on skill training, problem-solving strategies, and relapse prevention. Self-help materials were also provided. All patients used NRT. Received standard alcohol treatment concurrently with smoking cessation treatment. *Comparison:* Delayed group started smoking cessation treatment on day 180. Received alcohol treatment at the start of the study and smoking cessation treatment 180 days after enrollment.	30 days; 60 days; 90 days; 180 days Nicotine patches (size reduced after 4 weeks and lasted approximately 3 months), and/or nicotine gum, or nicotine lozenges	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤9 ppm and urinary cotinine level (intervention vs. comparison):* Day 30: 33.3% vs. 19.5%, *p* value not presented. Day 60: 21.6% vs. 9.8%, *p* value not presented. Day 90: 21.6% vs. 4.9%, *p* = .04 Day 180: 13.7% vs. 2.4%, *p* > .05 Alcohol *7-day abstinence (intervention vs. comparison):* Day 30: 76.5% vs. 70.7%, *p* value not presented. Day 60: 66.6% vs. 58.5%, *p* value not presented. Day 90: 49.0% vs. 51.2%, *p* value not presented. Day 180: 25.5% vs. 34.1%, *p* value not presented.

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Pengpid et al. 2015[161] *N* = 620 (215i, 199 tobacco control, 206 alcohol control) Outpatient clinic In-person Thailand	Adults (ages 18–60) who were assessed at moderate risk for tobacco and alcohol use with ASSIST	*Theoretical approach:* N/A *Intervention:* 3 sessions of brief counseling for alcohol use reduction and tobacco use cessation. Sessions based on the ASSIST-linked brief intervention in primary care for hazardous and harmful substance use. Also included feedback and discussion on the relationship between drinking and smoking and the effects of alcohol consumption on smoking cessation, advice to avoid or minimize drinking during the smoking cessation process, and self-efficacy for change. *Comparison (tobacco only):* 3 sessions of brief counseling on tobacco use cessation only using the ASSIST-linked brief intervention for hazardous and harmful substance use manual for use in primary care *Comparison (alcohol only):* 3 sessions of brief counseling on alcohol use reduction only using the ASSIST-linked brief intervention for hazardous and harmful substance use manual for use in primary care	3 months; 6 months None	Tobacco *ASSIST Tobacco Score (intervention vs. tobacco only vs. alcohol only)*: Month 3: 7.0 vs. 9.1 vs. 8.9 Month 6: 6.3 vs. 9.1 vs. 8.5 *p* for GEE intervention x time = .004 *Self-reported 7-day point prevalence abstinence (intervention vs. tobacco only vs. alcohol only)*: Month 3: 19.1 vs. 41.2 vs. 24.3 Month 6: 17.7 vs. 49.0 vs. 26.7 *p* for GEE intervention x time = .007; the tobacco only condition had higher smoking abstinence than the alcohol only condition ($p < .001$), which in turn had higher smoking abstinence than the intervention ($p = .028$) Alcohol *ASSIST Alcohol Score (intervention vs. tobacco only vs. alcohol only)*: Month 3: 5.6 vs. 7.5 vs. 6.3 Month 6: 6.1 vs. 8.6 vs. 4.8 *p* for GEE intervention x time = 0.179

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Rohsenow et al. 2014[162] *N* =165 (80i, 85c) Inpatient facility In-person USA	Individuals who met DSM-IV criteria for alcohol dependence and smoked ≥10 cigarettes per day for the past 6 months, and were not using NRT or bupropion	*Theoretical approach:* Motivational interviewing *Booster session*: Half of intervention participants and half of control participants received a 5–15-minute booster session 7–30 days after the initial session. *Intervention:* Initial 45-minute session involving pros and cons of smoking, interpreting health risks of carbon monoxide levels, costs of smoking relative to their income, smoking rate compared to norms, relationship of smoking to ongoing alcohol use and sobriety and the barriers to change. Patients selected stage-appropriate goals and methods, were informed of free access to NRT, and provided with smoking cessation pamphlets. Patients who attended booster sessions were asked about progress toward goals and focused on barriers self-efficacy. The second booster session reviewed feedback from 1 month assessment. *Comparison:* Smoking cessation counseling based on PHS Clinical Practice Guideline: 15-minute initial session assessing smoking rate and interest in quitting, advice on quitting and useful methods, and setting a quit date within 2 weeks. Provided with a consumer guide for smoking cessation and a list of smoking services in the state and informed of free access to NRT and smoking cessation pamphlets. Booster sessions involved checking on progress toward smoking cessation, engaged in problem-solving around barriers, noted successes and reminded patients of methods available.	1 month; 3 months; 6 months; 12 months Nicotine patches or nicotine gum	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤10 ppm:* Data not presented. *p* values for treatment and booster effect described as nonsignificant. Alcohol *Number of days alcohol and/or drugs used during each follow-up period:* Data not presented. *p* values for treatment and booster effect described as nonsignificant.

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Interventions in non-substance use treatment settings				
Ames et al. 2010[146] *N* = 41 (22i, 19c) Community In-person USA	Young adults (ages 18–30) who were interested in quitting smoking, who smoked 10 or more cigarettes per day during the past 6 months, and who reported binge drinking (≥4 drinks in a row for women; ≥5 drinks in a row for men) on 2 or more occasions per month in the past 3 months	*Theoretical approach*: N/A *Intervention (alcohol + tobacco):* 8 sessions of individual-based, semi-structured counseling integrated with PHS Clinical Practice Guideline, with Brief Alcohol Screening and Intervention for college students Week 1–6: Focused on behavioral treatments related to alcohol consumption, the link between tobacco and alcohol use, and addressing high-risk drinking's role in maintaining smoking Week 7–8: Brief visits (dispense nicotine patches, monitor participant safety) *Comparison (tobacco only):* 8 individual-based, semi-structured counseling sessions aligned with PHS Clinical Practice Guideline Week 1–6: Behavioral treatment Week 7–8: Brief visits (dispense nicotine patches, monitor participant safety)	Tobacco use evaluated at weeks 12 and 24 All participants received 8 weeks of nicotine patches; 21 mg/24 hours for 4 weeks, 14 mg/24 hours for 2 weeks, and 7 mg/24 hours for 2 weeks.	Tobacco *7-day point prevalence abstinence (intervention vs. comparison):* Week 12 (verified by breath carbon monoxide <8 ppm): 45% vs. 26%, *p* = .33 Week 24 (verified by salivary cotinine <15 ng/ml): 23% vs. 11%, *p* = .42 Alcohol *Number of drinks per month (intervention vs. comparison)* Week 12: 34.2 vs. 67.1, *p* =.259 Week 24: 39.3 vs. 60.8, *p* = .596 *Number of binge drinking episodes per month (intervention vs. comparison):* Week 12: 3.2 vs. 5.8, *p* = .242 Week 24: 3.8 vs. 6.3, *p* = .365

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Ames et al. 2014[145] *N* = 95 (48i, 47c) Community In-person USA	Young adults (ages 18–30) who smoked 1 or more cigarettes per day over the past 6 months and reported binge drinking (≥4 drinks in a row for women, ≥5 drinks in a row for men) on 1 or more occasions per month during the past 3 months	*Theoretical approach:* N/A *Intervention:* 8 sessions of individual-based, semi-structured counseling with PHS Clinical Practice Guideline + NRT Week 1–6: NRT and addressing alcohol-related behaviors, the alcohol-tobacco relationship, and the role of high-risk drinking in smoking maintenance. Behavioral aspects included developing a relapse prevention plan and implementing strategies for sustaining abstinence. Week 7–8: NRT and assessing compliance *Comparison:* 8 sessions of individual-based, semi-structured counseling with PHS Clinical Practice Guideline + NRT Week 1–6: Behavioral treatment Week 7–8: NRT and assessing compliance	Tobacco use evaluated at weeks 12 and 24. All participants received 8 weeks of nicotine patches with dosing based on their cigarette usage: *≥10 cigarettes/day* Week 1–4: 21 mg/24 hours Week 5–6: 14 mg/24 hours Week 7–8: 7 mg/24 hours *≤9 cigarettes/day* Week 1–6: 14 mg/24 hours Week 7–8: 7 mg/24 hours	Tobacco *7-day point prevalence abstinence (intervention vs. control):* Week 12 (verified by breath carbon monoxide <8 ppm): 19% vs. 9%, *p* = .078 Week 24 (verified by salivary cotinine <15 ng/ml indicative of abstinence): 21% vs. 9%, *p* = .050 Alcohol *Number of drinks per month (intervention vs. comparison)* Week 24: 49.2 vs. 51.3, *p* = .665 *Number of binge drinking episodes per month (intervention vs. comparison):* Week 24: 4.5 vs. 4.7, *p* = .703

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Correa-Fernandez et al. 2017[150] *N* = 202 (101i, 101c) Community Telephone; printed materials USA	Adults in Puerto Rico who smoked daily, were interested in quitting smoking, and met at least 1 at-risk drinking criterion in the past 30 days (≥2/≥1 drinks per day for men/women, drinking at least 5/4 drinks on 2 or more occasions for men/women, or driving after consuming ≥3 drinks on 1 or more occasions)	*Theoretical approach:* MAPS *All participants*: Received Spanish-language self-help materials on tobacco and alcohol use and received up to 7 telephone counseling calls (20–30 min each) focusing on increasing motivation to change smoking behavior and enhancing participant's self-efficacy to make a quit attempt. *Intervention:* Enhanced Motivation and Problem Solving (MAPS+). Components described above; in calls, the counselor also utilized motivational enhancement techniques to address at-risk drinking. *Comparison:* Standard Motivation and Problem Solving (S-MAPS), described above. No additional components.	12 weeks; 26 weeks; 52 weeks None	Smoking *Self-reported 7-day point prevalence abstinence:* Data not presented. *p* values by condition overall and for each time point > .05 Alcohol *Overall at-risk drinking*: Full data not presented, *p* values for condition overall (Beta = -.01, 95% CI = -.27–.24) and for each time point > .05 *Average drinks per day*: Full data not presented, *p* values for condition overall (Beta = .04, 95% CI = -.26–.34) and for each time point > .05 *Number of binge drinking episodes*: Full data not presented. *p* values for condition overall (Beta = .50, 95% CI = -.49–1.49) and for each time point > .05 *Number of drinking and driving episodes*: Full data not presented. *p* values for condition overall (Beta = -.-14, 95% CI = -.63–.35) and for each time point > .05

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
				Note: participants who successfully quit smoking and received the MAPS+ intervention were more likely to reduce their at-risk drinking behaviors than those who successfully quit and received the standard MAPS intervention.
Fridberg et al. 2015[152] *N* = 33 (11i [BI-S], 10i [BI-ARF], 11c) Community In-person USA	Young adults (ages 21–29) who reported current smoking, were not seeking treatment for tobacco or alcohol use, and reported heavy drinking (1–5 binge drinking episodes weekly and consumed 10 to 40 drinks per week), and who smoked 10–70 cigarettes/week	*Theoretical approach:* N/A *Intervention 1 (standard alcohol brief intervention: BI-S):* Participants completed assessment on desire to change smoking and drinking behavior and alcohol-related consequences and then two, 20–30-minute behavioral intervention sessions which included feedback on alcohol-related problems, education, and motivational enhancements to change drinking behavior. *Intervention 2 (standard alcohol brief intervention with personalized alcohol response feedback: BI-ARF):* Participants completed assessment on desire to change smoking and drinking behavior and alcohol-related consequences and then two, 20–30-minute behavioral intervention sessions which included feedback on alcohol-related problems, education, and motivational enhancements to change drinking behavior. An additional alcohol response session was included in the first behavioral intervention session where respondents consumed an alcoholic beverage and completed a questionnaire and then received feedback related to their individual responses to alcohol and how it serves as a potent cue to increase smoking urges and behavior. *Control:* Preintervention alcohol response session without feedback or discussion of results. Behavioral intervention sessions focused on general health and progressive muscle relaxation for stress reduction.	1 month; 6 months None	Smoking *Reduction in self-reported number of smoking days at Month 6*: 39% reduction (BI-ARF) vs. 3% reduction (control group), $p = .004$, BI-S estimate not presented Alcohol *Reduction in binge drinking at Month 6*: 64% (BI-ARF) vs. 16% (BI-S group), 25% (control group). $p < .001$ *Reduction in drinking frequency at Month 6*: 32% reduction (BI-ARF) vs. 2% increase (BI-S), $p < .001$, control group estimate not presented *Reduction in drinks per drinking day at Month 6*: 26% (BI-ARF) vs. 3% (BI-S), $p < .001$, control group estimate not presented Co-use *Reduction in smoking-alcohol co-use at Month 6*: 39% (BI-ARF) vs. 27% (BI-S) vs. 11% (control group)

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Fucito et al. 2020[153] *N* = 26 (13i, 13c) Community In-person USA	Adults who smoked on average ≥5 cigarettes on smoking days for a year or more, were interested in tobacco or alcohol treatment, and reported heavy drinking (>14/7 drinks per week for men/women or >5/4 drinks per day once per month for men/women over the past year)	*Theoretical approach:* Social cognitive theory *Intervention:* During first 4 weeks, participants attended weekly counseling and then could return weekly or biweekly for 4 weeks. Sessions addressed tobacco and alcohol and incorporated motivation and confidence to change, goal setting, stimulus control, managing urges and triggers, and behavior-change maintenance. *Comparison:* During first 4 weeks, participants attended weekly counseling and then could return weekly or biweekly for 4 weeks. Sessions addressed tobacco or alcohol, depending on a participant's primary presenting concern, and incorporated motivation and confidence to change, goal setting, stimulus control, managing urges and triggers, and behavior-change maintenance.	4 weeks after treatment; 12 weeks, and 6 months 12 weeks of varenicline	Smoking *7-day point prevalence abstinence at Month 6, confirmed by breath carbon monoxide ≤ 4 ppm (intervention vs. comparison):* 46% vs. 23%, $p = .17$, $d = .59$ Alcohol *Percent heavy drinking days*: There was no difference by condition overall or over time by condition, $p > .60$, full data not shown
Joseph et al. 2004[155] *N* = 499 (251i, 248c) Outpatient clinic and inpatient facility In-person USA	Adults (ages 21 to 75) who met DSM-IV criteria for alcohol abuse/ dependence, smoked more than 5 cigarettes per day for more than 1 year, and had completed 1 week of treatment for alcohol use	*Theoretical approach*: Stages of change *Intervention:* Concurrent tobacco and alcohol cessation treatment, 1-hour face-to-face session and up to 3 follow-up sessions. Treatments were based on the PHS Smoking Cessation Clinical Practice Guideline and content varied depending on the participant's stage of change. Addressed the relationships between smoking and alcohol use and the potential advantages of recovery from all substance use. Participants in the action stage of change received a free prescription for NRT. Behavioral and pharmacological treatments were available for 1 year following initiation of smoking cessation treatment. Participants received reminder postcards indicating treatment was available every 3 months. *Comparison:* Delayed cessation treatment; identical treatment to concurrent treatment but timed to begin 6 months after study enrollment	3 months; 6 months; 12 months; 18 months Nicotine patches (21 mg for 6 weeks, 14 mg for 2 weeks, and 7 mg for 2 weeks) and gum.	Smoking *7-day point prevalence abstinence verified by breath carbon monoxide (intervention vs. comparison):* Month 3: 15.5% vs. 4.4%, $p < .001$ Month 6: 10.8% vs. 5.2%, $p = .02$ Month 12: 12.8% vs. 10.1%, $p > .05$ Month 18: 12.4% vs. 13.7%, $p > .05$ Alcohol *6-month abstinence (intervention vs. comparison):* Month 6: 41% vs. 56%, $p = .001$ Month 12: 33% vs. 42%, $p = .06$ Month 18: 41% vs. 48%, $p = .14$

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Kahler et al. 2008[157] *N* = 236 (119i, 117c) Community In-person USA	Adults who smoked cigarettes regularly for at least 1 year, currently smoked at least 10 cigarettes per day, reported heavy drinking (>14/7 drinks per week for men/women or ≥5/4 drinks per occasion for men/women at least once per month over the past 12 months), and did not meet full DSM-IV criteria for alcohol dependence in the past 12 months	*Theoretical approach:* Motivational interviewing *All participants*: Had 4 individual counseling sessions with the quit date occurring at session 2, plus 6 weeks of nicotine patch. Counseling sessions were focused on problem-solving regarding high-risk situations for smoking relapse, providing support within the treatment, and encouraging participants to seek support for quitting smoking outside of treatment. *Intervention*: Brief alcohol information was included in sessions 1 and 2, including open-ended discussions of drinking and smoking patterns, normative feedback on drinking levels, and the risk of smoking relapse associated with drinking. Participants were encouraged to abstain from alcohol while on the nicotine patch. *Comparison:* Brief advice to avoid or reduce drinking while quitting smoking	2 weeks; 8 weeks; 16 weeks; 26 weeks after quit date Nicotine patches (21 mg for 4 weeks, 14 mg for 2 weeks, and 7 mg for 2 weeks).	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤10 ppm at weeks 2 and 8 and salivary cotinine ≤15 ng/ml at weeks 16 and 26 (intervention vs. comparison):* Week 2: 57.3% vs. 45.4%, *p* = .07 Week 8: 39.3% vs. 31.1%, *p* = .18 Week 16: 18.0% vs. 17.7%, *p* = .95 Week 26: 19.1% vs. 17.7%, *p* = .76 Alcohol *Heavy drinking in past 2 weeks (intervention vs. comparison):* Week 2: 45.9% vs. 58.7%, *p* = .06 Week 8: 51.9% vs. 57.7%, *p* = .39 Week 26: 60.0% vs. 68.0%, *p* = .23 *Drinks per week*: Intervention participants reported fewer drinks per week than participants in the comparison condition, exp(*Beta*) = .81 (95% CI = 0.67–0.98), *p* = .027; exact estimates of drinks per week not presented.

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Kahler et al. 2020[156] *N* = 119 (58i, 61c) Internet Online; text messages USA	Adults who currently smoked daily and reported heavy drinking (>14/7 drinks per week for men/women or ≥5/4 drinks per occasion at least once in the last month for men/women)	*Theoretical approach:* N/A *Intervention:* Participants were given access to self-administered modules on BecomeAnEx.org with standard smoking cessation content plus content specific to heavy drinking + 2 text messages/day before quit date, 3–5 messages/day for 2 weeks after their quit date, 1–2 messages/day for 6 weeks past quit date; 24 of the messages were alcohol-focused messages. *Comparison:* BecomeAnEx.org with standard content and up to 128 text messages using the schedule described above; only 1 text addressed alcohol use	1 month; 6 months None	Smoking *7-day abstinence (intervention vs. comparison):* Month 1 (self-reported): 17% vs. 16%, *p* = .90 Month 6 (verified by salivary cotinine <15 ng/ml): 5% vs. 2%, *p* = .36 Note: Participants who received BecomeAnEx.org modules with added alcohol content were less likely to report having a smoking lapse while drinking alcohol compared with those in the standard program. Alcohol *Number of heavy drinking days in the last 30 days (intervention vs. comparison):* Month 1: 4.4 vs. 2.9, *p* = .36 Month 6: 3.8 vs. 2.6, *p* = .92 *Total drinks in last 7 days (intervention vs. comparison):* Month 1: 8.8 vs. 10.6, *p* = .29 Month 6: 7.7 vs. 8.1, *p* = .57

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Meacham et al. 2021[158] *N* = 179 (85i, 94c) Internet Online USA	Young adults (ages 18–25) who used Facebook at least 4 days per week, currently smoked at least 1 cigarette per day at least 4 days a week, ≥100 lifetime cigarettes, and reported at least 1 heavy episodic drinking day in the past month (≥5/4 drinks on an occasion for men/women)	*Theoretical approach*: Motivational interviewing, CBT, and transtheoretical model *All participants*: Received tailored daily Facebook posts over 90 days that focused on use patterns, quitting strategies, and awareness of tobacco industry targeting. Participants also had access to live weekly group smoking cessation counseling for 12 weeks. Counseling content was based on readiness to quit, motivational interviewing, and coping skills. *Intervention:* Tailored posts addressed cigarette and alcohol co-use but did not aim to eliminate alcohol use. Tobacco-only posts represented a smaller proportion of overall post material (31%–55%). *Comparison:* Tailored posts to readiness to quit smoking only, did not address alcohol.	3 months; 6 months; 12 months 14 days of nicotine patches, either 14 mg for 5–9 cigarettes/day or 21 mg patches for 10 or more cigarettes/day	Smoking *7-day point prevalence abstinence, verified by salivary cotinine <11 ng/ml (intervention vs. comparison):* Month 3: 3.5% vs. 4.3%, $p > .05$ Month 6: 0% vs. 1.2%, $p > .05$ Month 12: 3.5% vs. 0%, $p > .05$ Alcohol *Days of binge drinking (intervention vs. comparison):* Month 3: 4.8 vs. 7.6, Month 6: 3.8 vs. 6.0, Month 12: 3.2 vs. 5.6, *p* value for condition = .322 *Number of drinks in the past week (intervention vs. comparison):* Month 3: 4 vs. 6, Month 6: 5 vs. 7, Month 12: 1 vs. 4, *p* values not provided *Alcohol Use Disorders Identification Test-Concise (AUDIT-C) scores (intervention vs. comparison):* Month 3: 3.9 vs. 5, Month 6: 3.7 vs. 4.8, Month 12: 3.5 vs. 4.1, *p* value for condition = .416

Table 7.4 (continued)

Study *N* (intervention [i], comparison [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Toll et al. 2015[83] *N* = 1,948 (975i, 973c) Community; tobacco quitline Telephone USA	Adults who reported smoking and heavy drinking (≥14/7 drinks/week for men/women or ≥5/4 drinks per day for men/women at least once in the past year)	*Theoretical approach*: Motivational interviewing *All participants*: Received an intake call that included medical screening for a 2-week starter package of nicotine patches or gum, the New York State Smoker's Quitline booklet ("Break Loose!"), and a smoking cessation tip sheet. Callers received a quit-day telephone call and NRT. *Intervention:* Alcohol + tobacco counseling included additional assessment questions in their interview focused on how smoking and drinking are paired and the ways drinking may inhibit smoking cessation. Research findings on how alcohol undermines smoking cessation efforts were shared and callers were recommended to never drink more than 3 (for women) or 4 (for men) drinks per sitting. Callers also received an additional NIAAA booklet on alcohol use ("Rethinking Drinking"). *Comparison:* The tobacco-only counseling condition focused on standard smoking cessation advice and received "Clearing the Air" booklet.	7 months Nicotine patch or nicotine gum	Smoking *Self-reported 7-day point prevalence abstinence (intervention vs. comparison):* 13.5% vs. 10.3%, OR = 1.37, 95% CI = 1.04–1.80, *p* = .03 Alcohol *7-day abstinence from heavy drinking (intervention vs. comparison):* 74.6% vs. 70.3%, OR = 1.29, 95% CI = 0.98–1.69, *p* = .07

Note. ASSIST = Alcohol, Smoking, and Substance Involvement Screening Test; CBT = cognitive behavioral therapy; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; CI = confidence interval; GEE = generalized estimating equations; ICD-10 = International Classification of Diseases, 10th Revision; mg = milligram; ml = milliliter; N/A = not applicable; NIAAA = National Institute on Alcohol Abuse and Alcoholism; ng = nanogram; NRT = nicotine replacement therapy; OR = odds ratio; PHS = Public Health Service; ppm = parts per million; SUD = substance use disorder; USA = United States of America.

Behavioral Interventions Outside of Substance Use Treatment Settings

The majority of clinical trials that have focused on treating smoking in people with SUD (including AUD) have been conducted in substance use treatment settings. Less is known about the feasibility and efficacy of intervening with tobacco and alcohol co-use outside of substance use treatment settings. As of 2026, there are no published review of studies testing the efficacy of behavioral interventions for smoking cessation that also addressed alcohol use in settings outside of intensive alcohol use treatment (e.g., community settings). Therefore, this section provides an overview of this subset of studies and the details of the individual studies noted in this section are in Table 7.4. The studies span a wide variety of settings and modalities,[83,145,146,150,152,153,156-158,161] including interventions delivered online. There has also been variation in the definitions of tobacco and alcohol use for defining the target population in these studies. For example, the tobacco use inclusion criteria have included callers to a state tobacco quitline who report any level of smoking,[83] individuals in the community who report regular smoking for a year and currently smoking at least 10 cigarettes per day,[157] and young adults in the community who report smoking at least 1 cigarette per day during the past 6 months.[145] By contrast, the alcohol use inclusion criteria have been more consistent, with most studies focusing on binge and heavy drinking.[83,145,146,148,150,152,153,156-158,161] Several studies reviewed in this section excluded potential participants meeting criteria for AUD.[153,156,157,161] In general, the results of these studies of combined tobacco and alcohol interventions offered outside of substance use treatment settings may be broadly generalizable to adults who smoke cigarettes and engage in frequent and/or heavy episodic drinking, but not those meeting the diagnostic criteria for AUD.

Across the small set of studies reviewed in this section, there was substantial variation in study design and population of focus, and many of the studies are small or pilot studies.[145,146,152,153,156,158] Study populations included young adults from the general community,[145,146,152,158] callers to a state quitline,[83] and people being seen in an outpatient hospital setting (not specific to SUD treatment) who were screened for tobacco and alcohol use, among other behaviors.[161] The majority of these behavioral smoking cessation and alcohol use interventions focused on integrating alcohol reduction content, largely focused on addressing alcohol as a barrier to quitting smoking, within the context of a smoking cessation intervention.[83,145,146,150,152,153,156-158,161] Several interventions were grounded in motivational interviewing to support smoking cessation and reduce drinking with alcohol reduction content added to the foundational smoking-only content.[83,157,158]

Some of the interventions were delivered entirely remotely via phone, website, or app-based platforms and text messaging. Specifically, Toll and colleagues (2015)[83] screened callers to a state tobacco quitline for heavy drinking and tested the efficacy of an augmented smoking cessation quitline protocol using specific content to address how alcohol use may interfere with successful smoking cessation. They found significantly higher smoking abstinence rates among the group that received the combined intervention (smoking cessation and addressed alcohol use) compared with the usual care quitline protocol (focused only on smoking cessation) intervention (7-day point prevalence abstinence at 7-month follow-up: 13.5% vs. 10.3%, $p = .03$). Meacham and colleagues (2021)[158] compared the efficacy of a Facebook-based smoking cessation intervention that also addressed heavy episodic drinking with a Facebook-based, smoking cessation-only intervention among young adults who engage in co-use. There were no

statistically significant intervention effects for the smoking abstinence or on the alcohol use outcomes. Kahler and colleagues (2020)[156] tested an integrated intervention consisting of a smoking cessation web-based program, and a tailored text messaging program designed to address both tobacco and alcohol use. There were no statistically significant effects for the integrated intervention compared to the standard care intervention for either the smoking or alcohol use outcomes, although participants in the integrated intervention were less likely to report drinking during a smoking lapse compared to those in the standard intervention.[156] More research employing remotely-delivered interventions for tobacco and alcohol co-use is warranted given their relatively low implementation costs and potential for broad reach.

NRT was offered in 5 of 10 studies but with varying implementation protocols.[83,145,146,157,158] Compared with the studies that implemented tobacco use treatment in substance use treatment settings, NRT was offered less consistently in studies conducted in other settings. Only one community-based study investigated the potential benefit of using varenicline as part of their behavioral counseling intervention designed to treat both tobacco use and heavy drinking.[153] Smoking cessation outcomes were consistently measured using 7-day point prevalence abstinence,[83,145,146,150,153,156-158,161] and some studies also included biochemical verification of self-reported cessation.[145,146,153,156-158] Primary and secondary alcohol outcomes varied and included outcomes such as ASSIST alcohol score,[161] percentage of heavy drinking days in the last 4 weeks,[153] any drinking in the past month,[83,152] drinks per week[156,157] or month,[145,146] abstinence from heavy drinking,[83] and number of binge drinking episodes in the last week[158] or month.[145,146,156]

The studies reviewed here on behavioral interventions to treat smoking in the context of alcohol use outside of substance use treatment settings offer some evidence that combined smoking cessation and alcohol use treatments can be effective for smoking cessation. In addition, results from several studies suggest that successfully quitting smoking may confer a benefit for alcohol reduction.[83,150] For example, Toll and colleagues (2015)[83] found that the combined intervention (alcohol + tobacco use treatment) was more effective than usual care for smoking cessation but did not find a main effect on reducing alcohol use. However, an interaction effect demonstrated that the combined intervention reduced drinking among participants who successfully quit smoking and may aid the maintenance of smoking abstinence.[83]

Summary: Behavioral Treatments for Smoking Cessation in the Context of Alcohol Use

In summary, the combined behavioral smoking cessation and alcohol reduction interventions delivered in inpatient and outpatient SUD settings and outside of substance use treatment settings appear to have one of two effects: (1) they improve smoking cessation outcomes (better cessation rates in enhanced versus usual or standard care) or (2) they have equal smoking cessation rates for enhanced compared with usual or standard care. The interventions did not exacerbate alcohol use, nor did they harm the success of AUD treatment.

In addition, in some of the studies conducted with populations not in a substance use treatment setting where no main effect of the combined intervention on alcohol use was found, interaction effects in two studies suggest that addressing alcohol use as part of a smoking cessation intervention may help those who successfully quit smoking during the study to reduce drinking and stay abstinent from tobacco.[83,150]

One potential limitation of the extant studies is that none attempted an explicitly adaptive approach to the tobacco or alcohol treatments, where the interplay of the tobacco and alcohol use behaviors was periodically assessed and informed the content of the intervention in real time. The theoretical treatment paradigms used in the present studies, such as motivational interviewing, motivational enhancement, CBT, and quitline counseling sessions, lend themselves to iterative and adaptive study designs that may be needed to better understand effective delivery of multiple behavior interventions, as these approaches focus on identifying goals, triggers and coping strategies in an iterative process.

Treatment of Tobacco and Alcohol Use in Adolescent and Young Adult Populations

Most substance use, including cigarette smoking and alcohol use, begins in adolescence, and addressing these behaviors early in life can reduce the risk of tobacco and alcohol-related health consequences.[163,164] Importantly, the brain is still developing through adolescence and until about age 25. This development includes brain structures involved in executive function and reward, such as the mesocorticolimbic system and the prefrontal cortex, which are key brain structures for executive function, learning, and memory. Both nicotine and alcohol can interfere with and impair this development, putting adolescents at risk for impaired learning and memory and priming the brain for the rewarding effects of other drugs.[73,165,166] Intervening early to interrupt progression of tobacco and/or alcohol use and to treat established use and/or use disorder is critical for adolescent health and to prevent the long-term consequences of use, including cancers. This section and Table 7.5 describe several studies of interventions specific to the adolescent population.

One meta-analysis has examined whether brief interventions to reduce alcohol use (with or without directly addressing tobacco use) would have effects on tobacco use among adolescents and young adults who use both substances.[167] This meta-analysis of 17 randomized or quasi-experimental controlled studies (ages 13–18 years, N = 2,709) published between 1980 and 2012 showed that brief interventions to reduce alcohol use among adolescents and young adults had a statistically significant effect on reducing alcohol use but not tobacco use, regardless of whether tobacco use was a direct target of intervention. However, the authors note that there was a statistically significant degree of heterogeneity among studies on the tobacco outcomes, indicating that variation across studies could be contributing to the null effect.

Myers and Prochaska (2008)[168] conducted an exploratory secondary analysis of data collected during an RCT for smoking cessation for adolescents (ages 13–18 years, N = 54) in treatment for SUDs (including alcohol), where the smoking cessation treatment was effective at 3-month follow-up. The secondary analysis also examined whether the tobacco cessation treatment affected the alcohol and substance use outcomes. The findings demonstrate that intervening with adolescents to treat tobacco use in the context of alcohol treatment may enhance success of the alcohol treatment; adolescents who received the smoking cessation intervention were significantly more likely to have reduced the number of days using alcohol and/or other substances at 3-month follow-up compared with those in the waitlist control group who did not receive the intervention. However, this reduction was not sustained at 6 months, and all findings could be considered preliminary due to small sample size.

Jaszyna-Gasior and colleagues (2007)[169] examined the influence of alcohol use on smoking cessation outcomes among a sample of adolescents (ages 13–17 years, *N* = 101) enrolled in an RCT testing NRT plus CBT-based group counseling for smoking cessation. The authors found that, among adolescents receiving the smoking cessation intervention, those who reported using alcohol were significantly less likely to have quit smoking. The level of alcohol use pre-enrollment in the trial was not associated with cessation success. These findings are concordant with adult studies where reducing or abstaining from alcohol while in treatment for tobacco dependence supports successful cessation.[83] This implies that including advice to abstain from alcohol while quitting smoking is important to treatment success among adolescents as well as that of adults.

Pedersen and colleagues (2022)[170] conducted a secondary analysis of a pilot study of a brief text message smoking cessation program that augmented usual care (brief counseling plus NRT) for unhoused young adults (ages 18–25 years; *N* = 77) receiving care/assistance through drop-in centers. The pilot study showed efficacy of the augmented program compared to usual care. In their secondary analysis to understand whether the intervention influenced alcohol use, other tobacco product use, and/or cannabis use at 3-month follow-up, they found that young adults receiving the enhanced intervention reported a significantly greater reduction in number of drinks on drinking days at 3-month follow-up than those in the control condition. These results are concordant with the adult literature where the tobacco intervention may also have a positive effect of reducing alcohol use, thus maximizing overall health benefit for people who use multiple substances.

Haug and colleagues (2017)[171] conducted a cluster RCT in Switzerland to compare the efficacy of a smoking cessation intervention that incorporated stage-specific text messages with additional alcohol content (MobileCoach Tobacco + [MCT +]) to that of an intervention with tobacco only content (MobileCoach Tobacco [MCT]) among 1,471 vocational and upper secondary school students. Effects on smoking abstinence at 6-month follow-up were moderated by maximum number of alcoholic drinks per day at baseline. MCT+ produced significantly greater 7-day point prevalence smoking abstinence than MCT for participants with heavier peak drinking at baseline (10+ drinks on at least 1 day) (16.5% for MCT+ versus 9.5% for MCT), whereas for participants with only 0–9 drinks on their heaviest drinking occasion, MCT+ and MCT produced similar 7-day point prevalence smoking abstinence at 6-month follow-up (0–2 drinks: 15.7% for MCT+ versus 15.9% for MCT; 3–9 drinks: 13.4% for MCT+ versus 16.0% for MCT).

One study tested an intervention designed to interrupt early progression of tobacco and alcohol use.[172] Preventing escalation from infrequent/intermediate use to regular use may be an important intermediate target for intervention efforts because adolescents may find it easier to achieve long-term abstinence at a time when dependence has not become entrenched. Evers and colleagues (2012)[172] conducted an RCT testing the efficacy of a computer administered transtheoretical model-based tobacco, alcohol, and other substance cessation program for middle and junior high school students. At 3-month follow-up, students randomized to receive the intervention were more likely to quit smoking and reduce alcohol use than students randomized to the control condition.

Summary: Treatment of Tobacco and Alcohol Use in Adolescent and Young Adult Populations

Taken together, the literature on treating adolescent tobacco use in the context of alcohol use has described findings similar to those seen in studies of adults. In particular, intervening on tobacco use either alone or with alcohol-specific intervention content may improve smoking cessation outcomes. In addition, the one intervention that treated adolescent tobacco use in the context of alcohol and SUD treatment contributes preliminary evidence that treating tobacco use may improve short-term alcohol use outcomes,[168] which aligns with some of the adult literature on this topic. Moreover, the promise of the interventions that have been tested is strengthened by their brevity and sound theoretical and evidence base with some evaluating and finding effectiveness in real-world settings, such as schools and clinics serving homeless youth. Overall, treating both tobacco and alcohol use concurrently may be an important and efficient approach for improving adolescent and young adult tobacco cessation outcomes and adolescent health more generally.

However, the existing literature on this topic has several limitations. Though some of the studies focused on treating tobacco use in populations that experience greater burden (e.g., youth with a SUD, unhoused youth),[168,170] only one study focused on intervening with adolescents who are members of racial and ethnic populations that experience tobacco-related differences in health outcomes.[173] In addition, all the studies focus on one type of tobacco product use—cigarette smoking. This is a notable limitation in understanding how to address tobacco and alcohol co-use in adolescents, especially because e-cigarettes were the most commonly used tobacco product among adolescents as of 2026.[174] Moreover, adolescents frequently report concurrent use of multiple tobacco products.[175] More research is needed to understand what interventions are effective for treating adolescents who report co-use of e-cigarettes or other tobacco products and alcohol.

Table 7.5 Selected Studies of Interventions for Smoking Cessation and Alcohol Use Targeting Adolescents Discussed in This Chapter

Study *N* (intervention [i], control [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Evers et al. 2012[172] *N* = 1,590 students who ever used alcohol, tobacco, cannabis, or other drugs (865i, 725c) Middle and junior high schools Computer-based in classroom setting with teacher present USA	Students from 22 middle and junior high schools (ages not reported; grades included were 6th through 9th grade)	*Theoretical approach:* Transtheoretical model *Intervention:* Up to three, 30-minute, internet-based, individualized sessions. Computerized tailored intervention program based on the transtheoretical model, with interactive features such as assessment questions, feedback, images, and QuickTime movies. Sessions delivered once per month. *Comparison:* No intervention	1 pretest assessment and 2 post-test (3 months after pretest and 14 months after).	Any substance use *Cessation of use of any substances (alcohol, tobacco, cannabis, other drugs) (intervention vs. comparison):* Post-test 1: 39.6% vs. 24.6% Post-test 2: 36.9% vs. 32.1% Random effects logistic regression models showed that differences between control and intervention groups were statistically significant at post-test 1 ($p < .05$) but not at post-test 2.
Haug et al. 2017[171] *N* = 1,471 (741i, 730c) Schools Online; text messages Switzerland	Students in vocational schools, mean age 18.6 years	*Theoretical approach*: HAPA *Mobile Coach Tobacco (MCT) system*: Both MCT and MCT+ interventions were developed using the MobileCoach system and received individually tailored text messages to support smoking cessation over a 3-month period. *Intervention:* Mobile Coach System + (MCT+). Subjects in the MCT+ group received integrated intervention, including web-based tailored feedback on alcohol consumption. For those who binge drank, personalized text messages were sent to encourage maintaining drinking within low-risk limits over a 3-month period. *Comparison:* Standard MCT intervention	Baseline survey; 6-month follow-up assessment	Smoking *7-day point prevalence abstinence (intervention vs. comparison):* 15.0% vs. 13.9% (CC $p = .61$, ITT) $p = .82$). *Mean number of cigarettes smoked per day (intervention vs. comparison):* Decrease of 2.7 vs. 2.8 (CC $p = .97$, ITT $p = .93$) MCT+ was more effective at improving smoking abstinence among participants who reported a higher drinking at baseline compared to those who reported less drinking (CC $p = .008$, ITT $p = .049$) *7-day point prevalence abstinence by number of drinks on heaviest drinking occasion at baseline (intervention vs. comparison):* 10+ drinks: 16.5% vs. 9.5% 3–9 drinks: 13.4% vs. 16.0% 0–2 drinks: 15.7% vs. 15.9% Alcohol *Mean alcoholic drinks per week at follow-up (intervention vs. comparison):* Decrease of 4.8 vs. 4.2 (CC $p = .74$, ITT $p = .28$)

Table 7.5 (continued)

Study *N* (intervention [i], control [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Jaszyna-Gasior et al. 2007[169] *N* = 101 analyzed (120 randomized) Community In-person USA	Adolescents aged 13–17 who smoked and were interested in quitting	*Theoretical approach:* CBT *All participants*: Attended a 45-minute CBT group session led by a trained social worker during each treatment visit. Visits occurred weekly during weeks 1–6 and every other week during weeks 7–12. *Group 1*: Active nicotine gum + placebo patch *Group 2*: Active nicotine patch + placebo gum *Group 3*: Placebo gum + placebo patch	Baseline, weeks 1–6, week 8, week 10, week 12, week 26 Nicotine gum or patch	Smoking *7-day point prevalence abstinence, verified by breath carbon monoxide ≤6 ppm:* Those reporting drinking since the previous visit had lower odds of abstinence (unadjusted OR = 0.42, 95% CI = 0.23–0.78, *p* = .0057; adjusted OR = 0.30, 95% CI = 0.16–0.58, *p* = .0003) compared to those who did not reporting drinking. Alcohol On average, participants reported alcohol use between visits on approximately 20% of visits (18.6% ± 26.4%).
Myers et al. 2008[168] *N* = 54 with baseline information. 39 (20i, 19c) completed follow-up assessments Outpatient clinic In-person USA	Adolescents aged 13–18 who reported weekly cigarette smoking, recruited from 4 outpatient substance abuse treatment programs in Southern California	*Theoretical approach:* N/A *Intervention:* 6, weekly, hour-long sessions intended to motivate and encourage changes in smoking behavior. *Comparison:* No intervention. Participation in the intervention was offered after completion of the 6-month follow-up interview.	Baseline, 3 months, 6 months	Substance use *Past-30-day abstinence from alcohol and other drugs:* Odds of abstinence were not significantly different for the treatment group vs. the comparison group at 3 months (OR = 3.6, 95% CI = 0.73–17.38, *p* = .12) or 6 months (*p* = .75). *Days of substance use (alcohol and other drugs):* Participants in the treatment condition had significantly fewer days of substance use than the comparison group at 3 months (*p* = .015) but not at 6 months (*p* = .31).

Table 7.5 (continued)

Study *N* (intervention [i], control [c]) Setting Intervention mode Country	Sample	Theoretical approach(es) Intervention and comparison condition	Assessment points Smoking cessation medication protocol	Results
Pedersen et al. 2022[170] *N* = 77 (40i, 37c) Community Text messages USA	Participants ages 18–25 who smoked ≥5 cigarettes/day on at least 20 days in the past month, were ready to set a quit date in the next 30 days, had a cell phone that could receive text messages, and were seeking services at 1 of 3 drop-in centers for youth experiencing homelessness in Los Angeles	*Theoretical approach*: NA *All participants:* Received a 30-minute group smoking cessation counseling session following the 5 As protocol (i.e., Ask, Advise, Assess, Assist, Arrange). *Intervention:* 174 automated text messages for 6 weeks, designed to provide strategies for getting quit support, calculations for money saved by quitting, information on health and social benefits of quitting. *Comparison:* No text messaging intervention	Baseline, 3 months All participants received a supply of nicotine patches with instructions for use and written materials on quitting.	Tobacco *Maximum of noncigarette tobacco use days in past month (intervention vs. comparison):* Change in outcomes at 3 months: -9.21 vs. 3.30, Cohen's *d* = 0.53, *p* = .05 *Sum of noncigarette tobacco use days in past month (intervention vs. comparison)*: Change in outcomes at 3 months: -23.11 vs. -7.30, Cohen's *d* = 0.58, *p* = .04 Alcohol *Days of alcohol use in past month (intervention vs. comparison):* Change in outcomes at 3 months: -4.53 vs. -0.77, Cohen's *d* = 0.33, *p* = .19 *Typical number of drinks on drinking days (intervention vs. comparison):* Change in outcomes at 3 months: -2.94 vs. 1.64, Cohen's *d* = 0.69, *p* = .01

Note. CBT = cognitive behavioral therapy; CC = complete case; CI = confidence interval; HAPA = Health Action Process Approach; ITT = intention to treat; OR = odds ratio; USA = United States of America.

Treatment of Tobacco and Alcohol Use in Cancer Survivors

Cancer survivors are an important population for treating both tobacco and alcohol use. As described in chapter 3, similar to the general population, current or former smoking is associated with more frequent alcohol use and binge drinking among cancer survivors. The co-use of these substances continues exposure to two significant, and potentially synergistic, risk factors for cancer. Tobacco and alcohol use also increases the risk for health effects in addition to cancer (e.g., cardiopulmonary disease) and diminished quality of life (e.g., financial toxicity). Intervening after a cancer diagnosis is critical given the known effects of tobacco use on cancer treatment, cancer-specific mortality and secondary primary cancer development,[35,176] as well as the potential for alcohol use to reduce cancer treatment effectiveness, increase the risk of recurrence or second primary cancers, and decrease overall survival rates.[177-180] Though there are limited data specifically on treatment for co-use of tobacco and alcohol among cancer survivors, clinical guidance for addressing tobacco and alcohol use in cancer survivors is similar to that indicated for the general population (as discussed in detail in this chapter). Clinicians are encouraged to screen for tobacco and alcohol use in the oncology setting (as well as other clinical settings), provide FDA-approved medications paired with behavioral counseling, or refer patients to programs that can provide the treatment.[94,176,181] For alcohol use, Karam-Hage and colleagues (2026)[181] note that due to the lower level of awareness of alcohol as a cause of cancer, clinicians screening for alcohol use may also need to educate their cancer patients about this risk before, or alongside, providing treatment or referral to an alcohol treatment program. Clinical guidance also encourages treating mental health conditions that often co-occur with tobacco and alcohol use and the experience of having cancer, as the stress of cancer diagnosis may cause or exacerbate mental health issues that need to be addressed in parallel with cancer treatment. Being diagnosed with cancer, especially a cancer caused by tobacco or alcohol, is often a key moment for patients in terms of motivation to quit or reduce use, and for doctors in terms of timing to provide counseling to quit or reduce use and assist with treatment.

Chapter Summary

Quitting smoking is challenging; it usually takes numerous attempts to achieve long-term abstinence. In the context of alcohol use, especially heavy drinking, the likelihood of successful smoking cessation is hampered further. Reducing and quitting drinking can be challenging as well, especially for people with alcohol use disorder. People seeking treatment for alcohol use disorder often relapse within 6 months of treatment. Effective pharmacological and behavioral treatments exist for treating tobacco use and for treating alcohol use disorder, yet these treatments are underutilized. Treatments that target tobacco and alcohol co-use have the potential to improve quit outcomes and health benefits. Tobacco and alcohol use and the development of use disorders are linked neurobiologically and behaviorally, which may inform new treatment targets. A line of research has sought to design and deliver integrated pharmacological and behavioral therapies that work together to optimize quit outcomes for both smoking and drinking. Engaging people who use both tobacco and alcohol in treatment for one substance provides an opportunity to treat the other, which could lead to successful treatment of both

behaviors. For example, offering treatment for tobacco use in the context of alcohol treatment can lead to successful smoking cessation and may also reduce drinking. In addition, increasing provider education about the benefits of treating smoking in the context of alcohol use and encouraging screening for both tobacco use and alcohol use, especially alcohol use disorder, to improve the use of effective treatments would further efforts to reduce cancer risks.

This chapter reviewed the available evidence from published systematic reviews and studies published after the systematic reviews were conducted. Some of the available evidence combines alcohol with other substance use when evaluating how best to deliver smoking cessation treatment to people with substance use disorder. This makes some aspects of understanding the role of alcohol use in the context of smoking cessation treatment more difficult to extrapolate. Nonetheless, currently available studies of smoking cessation in the context of alcohol treatment reviewed in this chapter have returned two consistent conclusions: (1) integration of smoking cessation treatment into alcohol use disorder treatment settings is not detrimental to the goals of reduced drinking or abstinence, and (2) treatment integration can reduce use of both tobacco and alcohol. Smoking cessation interventions that included alcohol-specific content in a motivation-based approach appeared to enhance the durability of the successful quit attempts and reduce drinking among those who had quit. However, integrated treatment development is still fairly nascent, with no single breakthrough treatment identified. Both of the Food and Drug Administration-approved medication classes for smoking cessation (i.e., nicotine replacement therapy and the oral medications varenicline and bupropion, which do not contain nicotine) have been evaluated for their effects on drinking when treating smoking, with unclear evidence for reducing alcohol use. The few studies conducted among adolescents find that, similar to adults, treating smoking and alcohol use in combination generally results in improved outcomes.

The health risks, including cancer risk, of concurrent smoking and alcohol consumption are well documented in the literature (see chapters 4 and 5), and warrant attention in provider-, education-, and systems-level interventions to support treatment of tobacco and alcohol use, in combination, routinely in the clinical and public health context.

Research Needs

New drug discovery is needed to effectively treat both tobacco and alcohol use independently, as well as new research to develop integrated pharmacologic and behavioral therapies for co-use and attention to high-risk subgroups. Future therapies would ideally leverage the overlapping neurobiological and neurobehavioral systems related to substance use dependence (described in chapter 6) to reduce the use of both substances. This is an emerging area of scientific research. For example, glucagon-like peptide-1 (GLP-1) agonist medications are approved for treatment of diabetes and obesity by the Food and Drug Administration (see chapter 6); as the use of these agents has become common, interest in understanding their effects on tobacco and alcohol use is increasing.[182] Although preclinical, animal research, and early clinical trial research with human participants indicates this potential concurrent effect, research is needed to more fully understand the effects of GLP-1 on the brain and the

implications of GLP-1 as a treatment for tobacco, alcohol, and/or other substance use.[182] The added benefit of counseling to support and sustain behavioral changes also warrants investigation. The genetics findings presented here and in chapter 6 are also important to consider when evaluating the utility of personalized integrated approaches.

More research is needed to understand how best to combine and schedule integrated treatments. People who smoke cigarettes and drink heavily may not be ready to directly tackle changing both behaviors at once. However, as this chapter has shown, progress made in changing one risk factor may have indirect effects on changing another and may indirectly support motivation to change another. Leveraging the process of change in one behavior (e.g., as elaborated in the Transtheoretical Model) to facilitate change in another highlights the need for integrated treatment strategies that more fully consider the interplay between smoking and alcohol use behaviors.[83,183] Moreover, research is needed to understand how societal trends in tobacco (e.g., e-cigarette use) and alcohol use (e.g., greater awareness of alcohol as a cause of cancer) interact and whether they encourage people to seek treatment.

The field needs standardized methods and reporting protocols for clinical trials that focus on the integrated treatment of tobacco and alcohol use. Studies ought to clearly define co-use, heavy use, and/or dual disordered use in stating their inclusion criteria. Definitions need to consider changes in tobacco and alcohol products and use behaviors. For example, there has been a decrease in the proportion of daily versus nondaily smoking (see chapter 2), and an increase in the use of e-cigarettes and other tobacco products (e.g., heated tobacco, nicotine pouches). Researchers should clearly specify the primary versus secondary intervention target(s) in their trial, whether the proposed therapy is aimed at treating both substances concurrently, and any hypothesized mediation effects. In addition, the paucity of evidence on treating other tobacco product use in the context of alcohol use presents an important research opportunity.

Investigators focused strictly on smoking cessation should measure alcohol use and analyze changes in alcohol use as an outcome and potential mediator and consider broadening inclusion criteria. In addition, there is substantial heterogeneity in the measurement of alcohol use outcomes (e.g., drinks per day, heavy drinking, alcohol abstinence), which indicates a need for developing consensus around common alcohol use outcomes to include in studies to improve interpretation of findings across studies. Moreover, there are relatively few studies that include people who report levels of drinking that do not exceed the guidelines and/or do not meet the criteria for alcohol use disorder. Those studies demonstrate potential for improving smoking cessation and reducing drinking among people who successfully quit. Understanding how to refine smoking cessation interventions to increase quitting in people who drink alcohol would advance smoking cessation while potentially reducing drinking. In addition, the enrollment criteria for many smoking cessation trials have excluded people who report other substance use, including alcohol use, especially heavy drinking. Although additional safety measures may be needed, broadening for the inclusion of people who drink heavily will increase study generalizability.

More research on treating young people who engage in tobacco and alcohol co-use is warranted. Although most initiation of tobacco product use and alcohol use occurs in adolescence and young adulthood, there have been relatively few treatment intervention studies for tobacco and alcohol co-use focused on that population. The research reviewed in this chapter indicates that treating both tobacco and alcohol use in adolescence and young adulthood is feasible and effective for quitting smoking and may be effective for reducing drinking. Research in treating co-use in young people has largely been conducted for cigarette smoking. Yet, as adolescent smoking has decreased, the use of e-cigarettes and other types of tobacco products has become more prevalent. Because co-use of these products and alcohol is widespread among youth and because there are few studies addressing co-use in youth, greater efforts are needed to develop interventions addressing co-use of e-cigarette and other noncigarette tobacco products and alcohol.

Conclusions

1. **The efficacy of smoking cessation treatment can be enhanced by reducing alcohol use, especially heavy alcohol use**. Most studies addressing tobacco cessation in the context of alcohol use have been conducted with people who engage in heavy drinking and/or have alcohol or substance use disorders. The preponderance of evidence shows that continued use of alcohol during smoking cessation treatment is a barrier to quitting smoking. For this reason, people trying to quit smoking should be advised and assisted to decrease or stop their alcohol use. Integrated tobacco use treatments that address both tobacco and alcohol use may increase reach and effectiveness across multiple settings, including inpatient and outpatient clinical settings and community settings.
2. **Coordinated treatment of concurrent tobacco and alcohol use is warranted**. Health care providers should screen for both tobacco and alcohol use, provide advice to quit tobacco and to reduce or quit alcohol to maximize health benefits, assess readiness to quit, and offer coordinated care and/or referral to evidence-based treatment that includes both pharmacological and behavioral interventions.
3. **Smoking cessation interventions are effective for people who smoke and have an alcohol use disorder, and providing smoking cessation treatment does not harm alcohol use outcomes; conversely, the evidence suggests it may improve them.** Engaging in smoking cessation treatment during treatment for alcohol use disorder does not jeopardize sobriety outcomes. Evidence-based treatments for quitting smoking in the general population are also effective for people who smoke and have an alcohol use disorder.
4. **Effective pharmacological and behavioral therapies to treat tobacco use and to treat alcohol use are available; however, evidence is still emerging to inform the development of combined treatment approaches for co-use.** The high prevalence of co-use of tobacco and alcohol points to the need for additional research to develop pharmacological and/or behavioral treatments that can effectively address treatment needs for people who co-use both substances.

5. **Measuring co-use in tobacco and alcohol treatment studies is critical for improving tobacco use treatment.** Regardless of the tobacco focus of the treatment study, systematically measuring alcohol use in that context will improve understanding of the effect of alcohol use on tobacco use treatment. This can also inform how tobacco use treatment affects alcohol use, thus enhancing understanding of how to treat both causes of cancer. Finally, measuring and addressing both tobacco and alcohol use in cancer treatment trials is important both to continue to better understand the effects of continued use of these substances on cancer treatment and patient outcomes.
6. **Pharmacological and behavioral tobacco treatment trials could be improved by taking alcohol use into account**. The high prevalence of tobacco and alcohol co-use means that excluding people who drink alcohol at any level (e.g., infrequent drinking, heavy drinking) from smoking cessation trials limits the generalizability of these trials. Additionally, because the use of other substances (e.g., cannabis or illicit drugs) is common among people who smoke and drink alcohol, differentiating between alcohol and "other drug use" in trials is important to determine the effect of alcohol on the efficacy of tobacco use treatment.

Appendix 7A Literature Search Strategy

Searches for relevant studies of pharmacotherapy and behavioral treatments were conducted with the assistance of a National Institutes of Health (NIH) librarian, using Boolean logic combining indexed terms (e.g., MeSH, Emtree) and keywords. PubMed (National Library of Medicine), EMBASE (Elsevier), Cochrane Library (Wiley & Sons), and Web of Science (Clarivate) were searched for this review. In addition, relevant clinical trials listed in the ClinicalTrials.gov database and reference lists of the included articles were also searched. Search terms were selected to encompass cigarette smoking, other tobacco product use, alcohol use, alcohol use disorder, and tobacco cessation.

Inclusion Criteria: Overall

In general, an article must have (a) been peer-reviewed and published between January 1, 2002, and January 31, 2023, in English, German, or Spanish; (b) described the outcomes of a randomized controlled trial (RCT), quasi-randomized (pre/post) clinical trial, or a nonrandomized (participants/researchers select group) clinical trial; and (c) been an intervention study for tobacco and alcohol cessation/reduction that reported intervention effects (i.e., results that report on outcomes for both behaviors separately), including interventions for reducing tobacco and alcohol use along with other substances, such as those conducted in a treatment facility or part of a modifiable risk behavior-focused intervention. Articles were excluded if they were baseline intervention data analyses, epidemiologic studies, study protocols, prevention interventions (i.e., to prevent smoking and/or drinking), economic/cost analyses, or preclinical, basic science, or mechanistic studies.

Inclusion Criteria: Pharmacotherapy Treatments

The section on pharmacotherapy treatments focused on RCTs only. Articles were excluded if they included people younger than 18 years of age, did not include tobacco and alcohol use as primary or secondary outcomes, focused on psychosocial/behavioral efficacy exclusively, and did not include behavior change as an outcome (e.g., quality of life).

Inclusion Criteria: Behavioral Treatments Among Adults

The section on behavioral treatments among adults excluded articles if they included people younger than 18 years of age, used tobacco use and drinking indicators as secondary outcomes, focused on medication/pharmacotherapy product efficacy exclusively, and those that did not include behavior change as an outcome (e.g., quality of life).

Inclusion Criteria: Behavioral Treatments Among Adolescents

The section on behavioral treatments among adolescents included articles if they were conducted with a population of adolescents (younger than 18 years of age) who reported any use of tobacco and alcohol and examined effects of an intervention to quit and/or reduce tobacco use and measured and reported effects of alcohol use.

Search Terms

Search strings are listed below. Search strings included terms for each tobacco and alcohol use and cessation behaviors:

- Alcohol: “Alcohol-Related Disorders”[MeSH] OR “Alcohol-Induced Disorders, Nervous System”[MeSH] OR “Drinking Behavior”[MeSH] OR “alcohol*”[Title/Abstract] OR “drinking behavior*”[Title/Abstract] OR “binge drinking”[Title/Abstract] OR “heavy drink*”[Title/Abstract] OR “drinking problem*”[Title/Abstract] OR “drinking motiv*”[Title/Abstract]
- Tobacco Smoking: “Smoking Devices”[MeSH] OR “Tobacco Use”[MeSH] OR “Tobacco Use Disorder”[MeSH] OR “smoking”[Title/Abstract] OR “smoker*”[Title/Abstract] OR “nicotine*”[Title/Abstract] OR “tobacco”[Title/Abstract] OR “cigarette*”[Title/Abstract] OR “cigar*”[Title/Abstract] OR “e-cig*”[Title/Abstract] OR “vaping”[Title/Abstract] OR “vape”[Title/Abstract] OR “snuff”[Title/Abstract] OR “snus”[Title/Abstract] OR “hookah”[title/abstract] OR “waterpipe”[title/abstract] OR “smokeless”[title/abstract]
- Cessation Behavior: “Treatment Outcome”[MeSH] OR “treatment*”[Title/Abstract] OR “Therapeutics”[MeSH:NoExp] OR “Behavior Therapy”[MeSH] OR “therap*”[Title/Abstract] OR “behavior chang*”[Title/Abstract] OR “Counseling”[MeSH] OR “counseling*”[Title/Abstract] OR “motivational interviewing*”[Title/Abstract] OR “mindfulness”[Title/Abstract] OR “Recurrence”[MeSH] OR “recurrence*”[Title/Abstract] OR “relapse*”[Title/Abstract] OR “remission*”[Title/Abstract] OR “abstinence*”[Title/Abstract] OR “Alcoholics Anonymous”[Title/Abstract] OR “stop”[Title/Abstract] OR “stopped”[Title/Abstract] OR “stopping”[Title/Abstract] OR “quit”[Title/Abstract] OR “quitting”[Title/Abstract] OR “Tobacco Use Cessation”[MeSH] OR “Smoking Cessation”[MeSH] OR “Smoking Reduction”[MeSH] OR “cessation*”[Title/Abstract] OR “abstinence*”[Title/Abstract] OR “Telemedicine”[MeSH] OR “telemedicine*”[Title/Abstract] OR “telehealth*”[Title/Abstract] OR “mHealth*”[Title/Abstract] OR “m-Health*”[Title/Abstract] OR “eHealth*”[Title/Abstract] OR “e-Health*”[Title/Abstract] OR “Digital Technology”[MeSH] OR “digital technolog*”[Title/Abstract] OR “digital health*”[Title/Abstract] OR “digital intervention*”[Title/Abstract]

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Chapter 8
Prevention of Tobacco Use in Relation to Alcohol

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Chapter 8
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Introduction

Despite the substantial progress made in reducing smoking among both youth and adults in the United States, 8.1% of middle and high school students reported using at least 1 tobacco product in the past month in 2024.[1] E-cigarettes are the most used product, with 5.9% of middle and high school students reporting current use.[1] According to the 2023 National Health Interview Survey, 19.5% of adults reported currently using any tobacco product (every day or some days)[2] and 6.5% of adults reported currently using e-cigarettes.[3] Adolescents who use tobacco have an increased risk of also using other substances, including alcohol,[4,5] and similarly, adolescents who consume alcohol have an increased risk of using tobacco.[6] In 2024, 13.3% of underage youth (ages 12–20) reported past-month alcohol use.[7] The epidemiology of tobacco and alcohol use, including variation across demographic groups and trends over time, is described in detail in chapters 2 and 3.

Efforts to prevent substance use and establish healthy behaviors are especially timely as adolescents and young adults transition through important developmental periods involving increased independence, risk-taking, and individuation from parents.[8-10] The effects of tobacco and alcohol use during these stages can disrupt development in critical regions of the brain, such as the prefrontal cortex, which governs learning, memory, and reward,[8,11] and the amygdala, which regulates fear-sensing.[8,12] Adverse health behaviors established in adolescence and young adulthood, such as tobacco and alcohol use, can persist into adulthood, leading to harmful health and social outcomes.[13,14] The risks associated with the use of tobacco and alcohol underscore the importance of identifying effective intervention programs and policy responses to prevent and reduce the use of tobacco and alcohol by adolescents and young adults. Such programs and policies could mitigate the adverse health and social consequences of tobacco and alcohol use and thereby enhance public health.

Because tobacco use and alcohol use are influenced by many of the same risk and protective factors,[15,16] it is possible that programs that target one substance might be effective in preventing use of the other substance. Numerous health education and policy interventions exist to prevent tobacco use, alcohol use, and other substance-related behaviors.[17-19] Health education and prevention interventions must first reach and engage young people to change their behavior.[15] To reach large numbers of adolescents efficiently via multiple channels, tobacco and alcohol prevention programs are typically delivered at multiple levels ranging from the individual to the societal.[16-19] Some interventions focus on preventing use of one substance, whereas others focus on preventing use of multiple substances, as well as preventing other risky health behaviors and/or promoting positive health behaviors and coping skills.

The literature on prevention of tobacco and alcohol use tends to be siloed, with many evaluations focusing on one substance or the other but not both. For example, evaluations of alcohol prevention programs rarely assess effects on tobacco use, and likewise, evaluations of tobacco prevention programs rarely assess effects on alcohol use. Similarly, research on tobacco

and alcohol policies is often siloed. Although similar policies exist for tobacco and alcohol separately (e.g., policies to restrict underage access, laws against possession, policies governing pricing and taxation, bans on flavors that appeal to adolescents and young adults), few evaluations of the effectiveness of alcohol prevention policies have assessed effects on tobacco use, and vice versa. This chapter summarizes evidence from the scientific literature to evaluate the extent to which alcohol-focused and general substance use prevention programs and policies are effective in preventing tobacco use among adolescents and young adults. The secondary goal of this chapter is to evaluate the extent to which tobacco-focused and more general substance use prevention efforts are effective in preventing alcohol use. Special attention is given to programs and policies that have been implemented to prevent initiation and progression of tobacco use among adolescents and young adults and tobacco prevention programs that include a focus on alcohol and other substances.

The 2012 Surgeon General's report, *Preventing Tobacco Use Among Youth and Young Adults*,[20] reviewed the epidemiology of tobacco use and the effectiveness of tobacco prevention programs. That report concluded that most tobacco use begins in adolescence or young adulthood, so prevention programs are needed for both age groups. It also concluded that multiple strategies are effective for preventing tobacco use, including mass media campaigns, comprehensive community programs, comprehensive statewide tobacco control programs, increases in cigarette prices, and specific evidence-based prevention programs in schools. Because the 2012 Surgeon General's report was released before the proliferation of novel tobacco products in the marketplace (e.g., e-cigarettes) and before substantial changes in the policy and media environment, an updated review is needed. This chapter extends beyond the 2012 Surgeon General's report by investigating the effects of tobacco-focused, alcohol-focused, and general or multiple substance use prevention program and policies on both tobacco and alcohol use.

Scope of the Chapter

This chapter reviews the potential of alcohol prevention programs and policies to prevent tobacco use, and vice versa. The programs and policies reviewed in this chapter focus on adolescents and young adults, because adolescence and early adulthood are the developmental periods when tobacco and alcohol initiation and escalation typically occur.[15,20] This chapter examines psychosocial prevention programs and policies delivered at the individual level (e.g., text-based, internet, and video game programs for individuals), group level (e.g., school-based and family-based programs), and population level (e.g., underage access policies, health education messages in traditional or social media) that focus on tobacco, alcohol, and/or polysubstance use prevention and measure tobacco use as an outcome. This chapter also examines some policies that have the prevention of tobacco and alcohol use among young people as a primary goal, specifically the National Minimum Drinking Age Act, Tobacco 21 legislation, and vertical identification card laws, while broader discussion of policies that affect tobacco and alcohol use more generally (e.g., clean indoor air laws, taxation) are covered in chapter 9. Additionally, treatment interventions aimed specifically at cessation, including studies with adolescents, are covered in chapter 7.

This chapter highlights interventions that address knowledge, attitudes, and behaviors to prevent tobacco use, as well as interventions that target upstream social determinants of health, such as access, pricing, and other critical economic influences on the uptake of tobacco and alcohol.

Because this monograph focuses on understanding how addressing tobacco use in the context of alcohol use can improve tobacco control, this chapter is limited to prevention studies that assessed effects on tobacco use or attitudinal precursors to tobacco use, such as intentions and susceptibility (i.e., the lack of a firm commitment not to use). The review of prevention programs is limited to randomized controlled trials (RCTs) conducted in the United States and published in English-language peer-reviewed journals. When possible, meta-analyses and systematic reviews are cited in lieu of individual studies. Systematic reviews and meta-analyses cited in this chapter are summarized in Appendix 8B, and summaries of evidence for effective or promising intervention programs are summarized in Table 8.3.

It is important to note that prevention is a broad term that can refer to preventing initial experimentation with tobacco or alcohol, preventing initiation of regular use, or preventing escalation to higher levels of use. Villanti and colleagues (2019)[21] described a concept called "prevescalation," which generally refers to preventing escalation of substance use during the transition from adolescence through young adulthood. This concept acknowledges that the process of developing substance use patterns and dependence involves a series of transitions that can occur throughout adolescence and young adulthood (e.g., nonsusceptible to susceptible, susceptible to experimentation, experimentation to more regular use, regular use to dependence). Some adolescents progress quickly from first experimentation to regular use and dependence in early adolescence, others initiate use later and reach regular use and dependence in young adulthood, and still others experiment during adolescence and/or young adulthood but then desist from use and never reach regular use or dependence. Prevention programs delivered during adolescence or young adulthood could intervene at any step on this continuum from experimentation to dependence. Because prevention programs are typically targeted to large segments of the population (e.g., all students at a high school or college, the general population via media campaigns), they generally reach people at all stages of the process from first experimentation to regular use or dependence. For example, a prevention program for adolescents or young adults will reach some never users; some experimenters; and some dependent, regular users. The goal of such programs is to prevent future use, regardless of the individual's history of use. An important corollary of this philosophy is that experimentation with tobacco or alcohol does not imply a failure of a prevention program or of an individual; rather, experimentation presents an opportunity to prevent escalation to the next stage in the use process. The concept of prevescalation integrates prevention for adolescents and young adults by focusing on disrupting and reversing the progression from never use to regular, dependent use, meeting the individual wherever they are in the use process. Viewing tobacco and alcohol use and dependence as a gradual process that unfolds over the approximately 2 decades of adolescence and young adulthood presents continuing opportunities to halt and reverse the process at any stage and at any age, allowing prevention programs to be delivered to populations at varying stages of substance use.[21] As this chapter will demonstrate, integration and continuity of prevention strategies across the life course is a strength, not a weakness, of prevention programs.

Literature Search Strategy

Literature searches were conducted to identify published articles about tobacco or alcohol prevention programs among adolescents and young adults, as well as empirical studies of youth-focused policy evaluations of crossover impact on tobacco and alcohol use (see Appendix 8A for search details). Relevant articles are summarized in a narrative format in this chapter. Because the results of tobacco-industry sponsored prevention programs may be biased by conflicts of interest,[22,23] such trials are considered in a separate section of the chapter ("Industry-Sponsored Adolescent and Young Adult Prevention Programs" section).

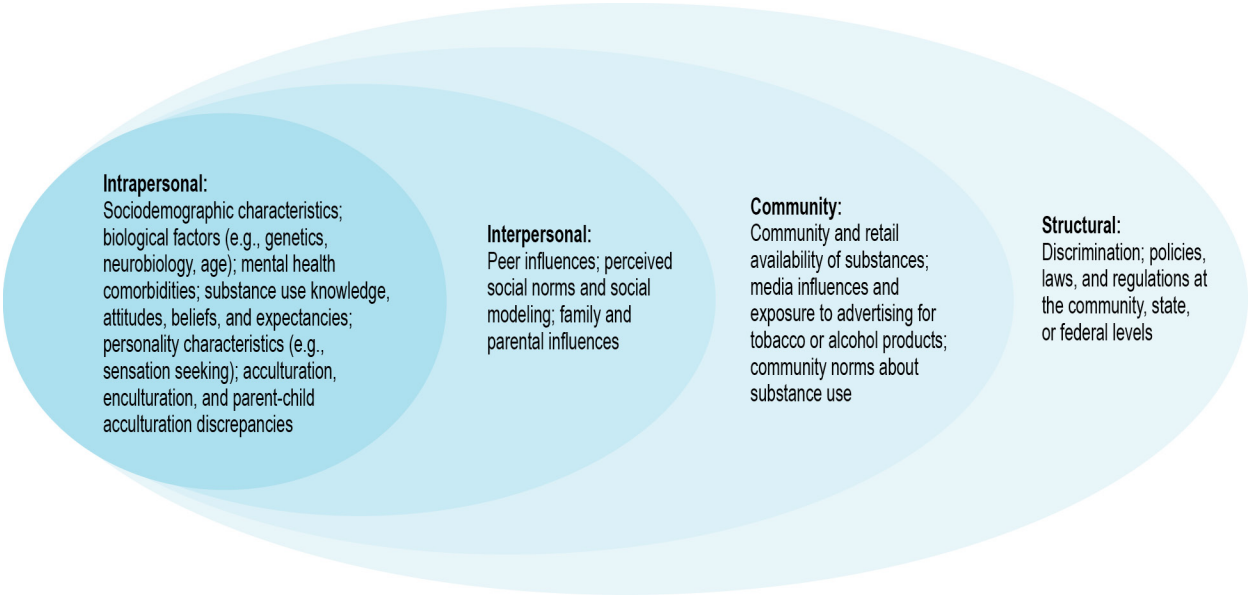
The policies reviewed in this chapter are ones for which the primary intended effect is to prevent adolescent and young adult initiation of tobacco and alcohol use. Policies that are broader in their intended effects (i.e., have prevention effects and effects on reducing prevalence and/or exposure to tobacco use), such as economic, retail, and smokefree policies, are reviewed in chapter 9. Furthermore, this chapter reviews restrictions on tobacco advertising and promotion when they occur in the context of specific prevention efforts; restrictions on advertising and promotion more broadly are reviewed in chapter 9. Exposure to tobacco advertising and promotion is a known risk factor for tobacco use, with the 2012 Surgeon General's report concluding that "advertising and promotional activities by tobacco companies have been shown to cause the onset and continuation of smoking among adolescents and young adults."[20,p.8] Effects of tobacco use and media exposure were also covered extensively in the 2008 NCI Tobacco Control Monograph 19, *The Role of Media in Promoting and Reducing Tobacco Use,* with the report concluding that "the total weight of evidence—from multiple types of studies, conducted by investigators from different disciplines, and using data from many countries—demonstrates a causal relationship between tobacco advertising and promotion and increased tobacco use."[24,p.8-9] Similarly, exposure to alcohol use through media and advertisements is associated with alcohol use.[25-27] The evidence reviewed in previous Surgeon General's reports and NCI Tobacco Control Monographs is not revisited in this chapter; instead, attention is focused on other prevention effects. Although the policies reviewed in this chapter are those that focus primarily on prevention, they may also reduce the toll of tobacco use in other ways, such as by increasing cessation or reducing secondhand smoke exposure.[28]

Common Modifiable Risk and Protective Factors for Tobacco and Alcohol Use

It is important to understand the modifiable risk and protective factors for tobacco and alcohol use, because these factors are the targets of effective prevention programs.[29,30] Prevention programs can be effective only to the extent that they target the antecedents of substance use in a population.[15,31] For example, programs based on teaching refusal skills will be effective only if adolescents or young adults want to refuse to use a substance. If adolescents or young adults perceive that substance use will allow them to fit in with a social group, to self-medicate a mental health problem, or to rebel against their parents, a program that centers only on the refusal skills approach is unlikely to be effective. A thorough understanding of the risk and protective factors for substance use can guide prevention program development and ensure that prevention programs actually modify the most salient intrapersonal, interpersonal, community, and structural antecedents of substance use.

Figure 8.1 shows a conceptual model of the risk and protective factors for tobacco and alcohol use, which are complex and exist at multiple levels. This conceptual model is a variation of the social-ecological model,[32-35] which identifies interactions among individuals, groups/communities, and their broader environment as affecting health. These varied factors include genetic influences; mental health comorbidities; individual knowledge, attitudes, and beliefs; peer and parental influences; perceived social norms and social modeling; access to substances; media influences; community-, state-, or federal-level policies; social determinants of health (e.g., poverty, discrimination based on race, ethnicity, or sex); and numerous other factors.[36-38] More comprehensive lists of risk and protective factors are published elsewhere.[37,39] Decades of research have identified numerous factors that affect the likelihood of adolescent and young adult substance use, substance use patterns, and health outcomes associated with substance use.[37,40-44] Contributing factors at the intrapersonal, interpersonal, community, and structural levels can be considered to understand these different behaviors and health outcomes.

Figure 8.1 Conceptual Model of Selected Risk and Protective Factors for Tobacco and Alcohol Use



Note. This conceptual model is a variation of the social-ecological model,[32-35] highlighting specific factors that influence youth tobacco and alcohol use at the intrapersonal, interpersonal, community, and structural levels.

Intrapersonal factors include substance use knowledge and expectancies,[45,46] personality characteristics such as sensation seeking and impulsivity,[47] acculturation (the process by which individuals adapt their own cultural beliefs, values, and practices to another culture[48]), and mental health conditions such as depression and attention-deficit/hyperactivity disorder.[49] Family and parental factors include substance use of parents and siblings,[50,51] parent-child communication,[52] parental monitoring and support,[51,53] family conflict,[51] and parental attitudes and rules about substance use.[51,53] Peer factors include substance use by friends and close others in one's social network,[54-56] substance use offers from friends,[57] and perceptions of substance use norms.[58-60] These peer factors are consistent with social cognitive theory,[61] which states that people are motivated to imitate a peer's behavior if they witness the peer receiving tangible

or social reinforcement for performing the behavior. Community-level factors include community and retail availability of substances (see chapter 9), community norms about substance use,[62] and exposure to advertising for tobacco or alcohol products.[63,64] Structural-level factors including policies and regulations are discussed in chapter 9 and in this chapter in the section "Policy Influences on Tobacco and Alcohol Use."

Additional factors that influence the risk of substance use among subpopulations of adolescents and young adults can be grouped along the continuum of influences described in Figure 8.1. Individual level factors such as acculturation to the U.S. culture (absent retention of the heritage culture) is a risk factor,[65] whereas enculturation (learning about or connecting to the heritage culture) is protective.[66-68] Family and parental factors such as parent-child acculturation discrepancies (i.e., greater adolescent orientation to U.S. culture versus greater parental orientation to heritage culture) increase the risk of substance use by impairing family functioning.[69,70] A more expansive view of community- and structural-level influences includes stressors that are unique to immigrant and racial and/or ethnic minority adolescents, including discrimination, bicultural stress, and perceived negative context of reception in the United States, all of which are also risk factors for substance use.[71] Cultural socialization (i.e., the process by which parents convey cultural heritage, pride, values, and practices to their children)[72] and a positive sense of one's racial and ethnic identity may protect against adolescent substance use.[73] The disparities in tobacco and alcohol use (see chapter 3) and their associated health outcomes among certain populations suggest that it may be beneficial to tailor prevention programs for these groups.[28,74,75] For example, the Next Legends prevention campaign from the U.S. Food and Drug Administration's (FDA) Center for Tobacco Products,[76-78] was developed to address e-cigarette use among American Indian/Alaska Native (AI/AN) adolescents; consequently, the campaign messaging was designed to be congruent with the cultural considerations identified through formative research with AI/AN adolescents.[76]

History of Tobacco and Alcohol Prevention Programs for Adolescents and Young Adults

For much of the 20th century, use of tobacco and alcohol was widespread in the United States. Contributing factors included the repeal of alcohol prohibition in 1933, promotion and distribution of cigarettes to soldiers during the first and second World Wars, and the ubiquitous promotion of tobacco products in the media.[79,80] Smoking prevention was recognized as an important public health challenge after the landmark 1964 Surgeon General's report, *Smoking and Health: Report of the Advisory Committee of the Surgeon General of the Public Health Service,*[81] documented the causal relationship between cigarette smoking and lung cancer, and called for appropriate remedial action. Early studies determined that tobacco and alcohol use typically co-occur[82] and identified common psychological and social risk factors for tobacco and alcohol use that were consistent with existing and subsequent behavioral theories (see Table 8.1).[83]

Table 8.1 Psychological and Behavioral Theories Relevant to Understanding Tobacco and Alcohol Prevention

Theoretical framework	Brief description	Key concepts	Examples of use in tobacco and alcohol use prevention programs
Cognitive Dissonance Theory[84]	People are motivated to act in ways that are consistent with their previous statements. Dissonance between cognitions or between cognitions and behavior is psychologically uncomfortable, and individuals are motivated to reduce this dissonance, such as by avoiding dissonant information or changing behaviors and cognitions to be consonant.	**Consonant relationship:** Cognitions that are relevant to and consistent with one another **Dissonant relationship:** Cognitions that are relevant to but inconsistent with one another **Dissonance magnitude:** Number and importance of cognitions that are consonant versus dissonant **Dissonance reduction:** Removing/reducing importance of dissonant cognitions or adding/increasing importance of consonant cognitions	When applied to prevention programs, having adolescents make a public commitment to not use tobacco or alcohol should motivate them to subsequently act in accord with these previous statements.
Common Liability to Addiction Model[85]	Dependence on more than one substance is caused by a common propensity for substance use, which consists of a complex array of genetic, physiological, intrapersonal, social, and environmental factors.	**Liability:** Extent to which an individual would be affected by or resilient to developing a substance use disorder	When targeting antecedents to tobacco and alcohol use in prevention programs, it should be recognized that these targets can be leveraged to decrease likelihood of use for both tobacco and alcohol, given the shared liability for substance addition that underlies use disorders for both substances.
Elaboration Likelihood Model[86]	A dual-process model proposing two routes for information processing in relation to attitude change: a central route and a peripheral route. When individuals have the motivation, opportunity, and capability to process information presented, they will engage in more deliberate, thoughtful elaboration of the information (through this central route), and their resultant attitudes will be more persistent.	**Central route:** Engagement of this route is available with sufficient motivation, opportunity, and capability. Persuasion results from engaging in a high degree of thought/elaboration on the presented information; resultant changes in attitudes will be relatively more persistent and more likely to influence behavior. **Peripheral route:** Engagement of this route is not dependent on having sufficient motivation, opportunity, and capability. Persuasion is based on simple inferences from positive or negative associations with the presented information, including aspects unrelated to informational message content (e.g., who delivers the message, how professional a message looks).	When applied to prevention programs, content may be tailored to the situation. In situations where individuals may have the motivation, opportunity, and capability to engage in attitude change (e.g., in a health class in school), well-reasoned, justified, and defensible arguments will be more persuasive. In situations where individuals may have limited motivation, opportunity, or capability to engage in a high degree of thought about a message (e.g., viewing an advertisement on TV, hearing an advertisement on the radio), using messages that have clear negative or positive connotations to direct the recipient to a positive or negative position will be useful (e.g., presenting smoking with images of diseased lungs).

Table 8.1 (continued)

Theoretical framework	Brief description	Key concepts	Examples of use in tobacco and alcohol use prevention programs
Problem Behavior Theory[87]	Health-compromising behaviors tend to co-occur in individuals, presumably because similar risk and protective factors associated with the behaviors increase or decrease the likelihood of engaging in these behaviors.	**Risk factor:** Increases the likelihood of an individual engaging in risky or negative health behaviors **Protective factor:** Decreases the likelihood of an individual engaging in health-compromising behaviors	Recognizing that tobacco and alcohol use co-occur because of shared risk and protective factors, prevention programs can work to address use of both substances and to identify the most important shared risk and protective factors to address.
Reasoned Action Approach[32,34,88-90]	Originally proposed as the Theory of Reasoned Action, this approach posits that attitudes and perceived norms influence intentions, which then influence behavior. It was further modified into the Theory of Planned Behavior with the inclusion of perceived behavioral control on intentions and behaviors. It was again modified into the Reasoned Action Approach by including indirect effects on behavior (such as perceived norms and antecedents predicting attitudes) and by including feedback effects from behavior to antecedents.	**Attitudes:** Favorable or unfavorable beliefs about a behavior, based on anticipated outcomes and past experiences **Perceived norms:** Whether the behavior should or ought to be done (injunctive norm) and whether others engage in the behavior (descriptive norm) inform perceived social pressure to engage in the behavior **Perceived behavioral control:** The extent to which individuals believe they can engage in a behavior **Intentions:** An individual's perception of their likelihood of engaging in a behavior	When applied to tobacco and alcohol use, prevention programs may focus on creating more positive attitudes about not using substances (or negative attitudes about using substances), changing perceived norms around use, enhancing perceived behavioral control over choosing not to use a substance, or forming intentions not to use a substance.
Self-Determination Theory[91]	Individuals are driven to fulfill the psychological needs of autonomy, competence, and relatedness, and these drives motivate behavior. Fulfillment of these needs supports autonomous or intrinsic motivation.	**Autonomy:** Reeling in control over one's behaviors and outcomes **Competence:** Having the necessary skills to engage in a behavior **Relatedness:** Having close relationships with others and experiencing belonging **Intrinsic motivation:** Engaging in a behavior for outcomes inherent to behavioral engagement itself (e.g., a feeling of mastery, enjoying the activity); enhanced by fulfilling autonomy, competence, and relatedness **Extrinsic motivation:** Engaging in a behavior for outcomes external to the individual (e.g., social positioning, financial costs)	Prevention programs that support autonomy (e.g., by providing options, recognizing adolescents' perspectives), competence (e.g., by teaching coping strategies or skills to not use tobacco or alcohol), and relatedness (e.g., engaging in program activities with close others) will result in greater intrinsic motivation not to use tobacco or alcohol. Prevention programs that impose external costs only may be effective in the short-term, but once the costs are removed, participants will no longer have the extrinsic motivation not to use tobacco or alcohol.

Table 8.1 (continued)

Theoretical framework	Brief description	Key concepts	Examples of use in tobacco and alcohol use prevention programs
Social-Ecological Model[32,92-94]	The social-ecological model considers that a complex interplay between multiple levels of protective and risk factors contribute to the development and maintenance of substance use and dependence. This approach acknowledges the interconnectedness of individual choices and broader environmental influences in shaping substance-use–related behaviors and outcomes.	**Individual level:** Biological (e.g., genetics, neurobiology, and age); psychological (e.g., psychiatric comorbidities and trauma); and behavioral factors (e.g., frequency and quantity of substance use, decision-making, and motivation to quit or reduce substance use) substance use knowledge, attitudes, beliefs, expectancies; personality characteristics (e.g., sensation seeking); acculturation, enculturation, and parent-child acculturation discrepancies **Interpersonal level:** Family dynamics (e.g., history of addiction, parenting styles, and family support/conflict); peer influence (e.g., social network and peer pressure); and relationships (e.g., quality of personal relationships, including romantic, friendship, and social interactions) **Community level:** Social norms (e.g., community and cultural norms surrounding substance use); access to substances (e.g., availability and accessibility); community support (e.g., access to substance use treatment and mental health services); and cultural and media influences (e.g., media portrayal of substance use via advertisement and entertainment) **Structural level:** Policies and laws pertaining to substance use prevention (federal, state, and local levels), discrimination, influence of industries producing and marketing substances (e.g., tobacco and alcohol)	The social-ecological model recognizes that prevention programs must consider addressing multiple levels simultaneously. Prevention efforts should consider not only individual factors but also the broader context in which substance use occurs. Effective strategies often involve a combination of education, policy changes, access to treatment, and support at various levels to reduce the prevalence and impact of substance use in society.
Social Cognitive Theory[32,34,61,88]	Originally proposed as social learning theory, it posits that individuals learn new behaviors by observing behaviors and their consequences in others and imitating them. It was extended into social cognitive theory with the inclusion of cognitive processes that mediate learning and the inclusion of self-efficacy.	**Observational learning:** Acquiring new skills or behaviors by watching the behavior of others, both in person and through other mediums (e.g., a video) **Vicarious reinforcement:** Increasing (or decreasing) the likelihood of engaging in a behavior by watching another person who has engaged in that behavior receive reinforcement; learning behavior through observing others receive reinforcements or punishments for that behavior **Reciprocal determinism:** Cognitions, behaviors, and the environment all influence each other **Self-efficacy:** Belief in one's abilities to successfully engage in a behavior	When applied to tobacco and alcohol use, prevention programs may focus on modeling the desired behavior with reinforcements, or the undesired behavior with punishments, and on enhancing confidence in one's abilities not to use a substance. For example, prevention programs may include role-playing activities in which students watch a facilitator refuse an offer of tobacco or alcohol from a peer and then practice refusing an offer themselves.

Table 8.1 (continued)

Theoretical framework	Brief description	Key concepts	Examples of use in tobacco and alcohol use prevention programs
Social Influence Theory[88,89,95,96]	Individuals' emotions, cognitions, and behaviors are influenced by others through informational and normative influence processes, which operate by eliciting compliance, identification, and/or internalization.	**Compliance:** Behaving as others expect or request out of a desire for a positive response from others **Identification:** Accepting a belief/behavior out of a desire to create or maintain a relationship with others or a group **Internalization:** Accepting information and behaving accordingly because its content is aligned with one's existing beliefs **Informational social influence:** Accepting information from others as accurate or truthful; can lead to re-evaluation of one's thoughts and behaviors, or internalization of that information, or acceptance of that information via identification **Normative social influence:** Conforming to others' expectations; leads to behaviors consistent with those expectations regardless of one's beliefs, or compliance	Prevention programs can use social influence to promote anti-tobacco and anti-alcohol norms, which will elicit identification and compliance. Prevention programs can also frame information in self-relevant values to enhance internalization of that information.
Social Inoculation Theory[97,98]	Attitudes can be resistant to change from counterattitudinal messages (e.g., advertising, peer pressure) through exposure to counterarguments paired with successful refutation, making those attitudes more resistant to future persuasion attempts.	**Threat:** Message content that creates a perception that one's current attitudes are vulnerable. When activated, threat motivates individuals to protect those attitudes. **Preemptive refutation:** Message content that allows one to strengthen their attitudes and make them more resistant to change, typically by raising and refuting counterarguments.	People can learn to counterargue against misleading tobacco and alcohol advertising messages; therefore, prevention programs may contain media literacy components.

Note. Theories are listed in alphabetical order.

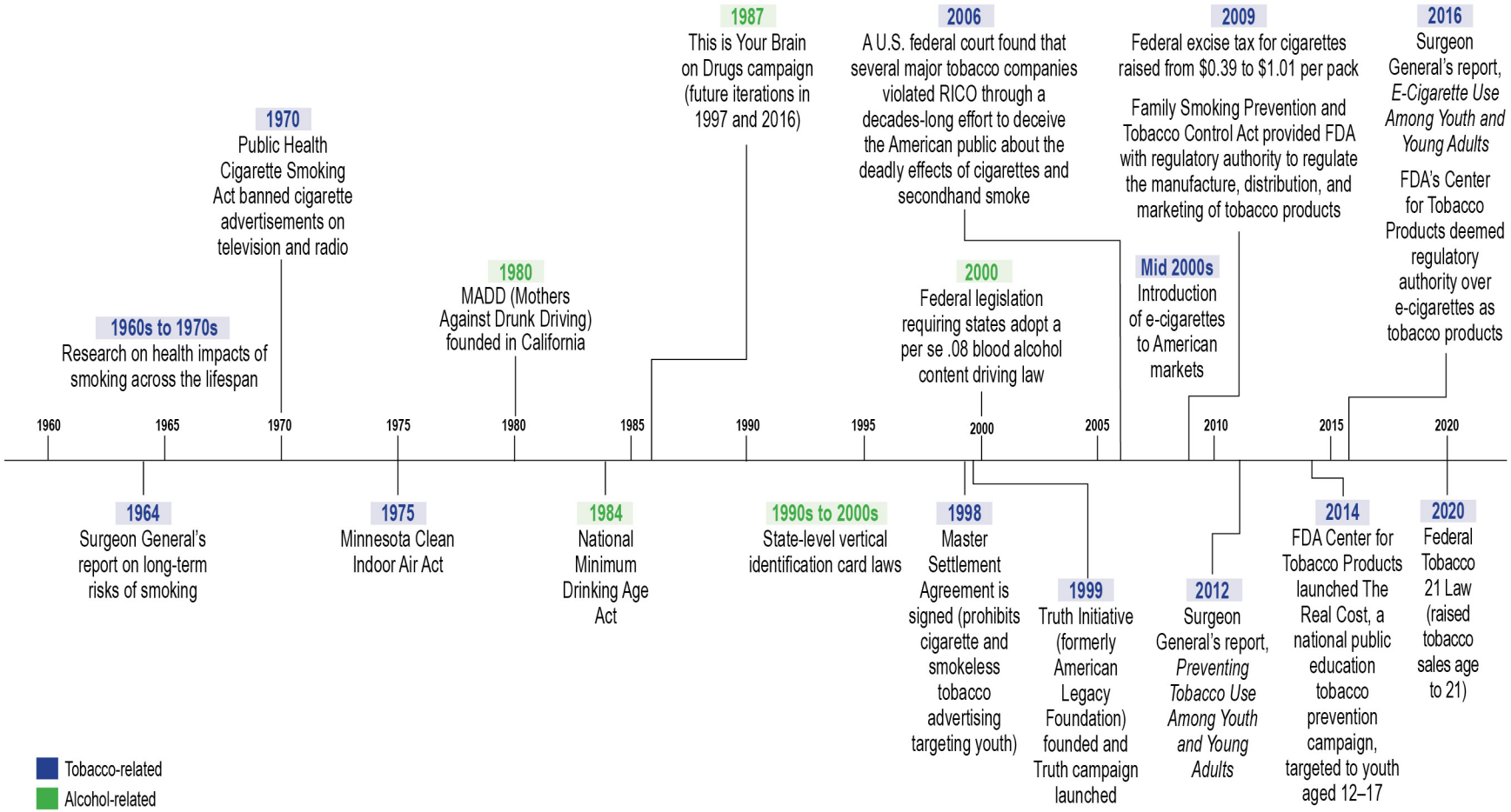
The earliest tobacco prevention programs in the 1960s used fear appeals and information about the health consequences of tobacco use, with the expectation that adolescents who understood the risks of tobacco would make the logical decision not to use the substance.[99,100] However, these programs were not effective at preventing tobacco use because knowledge alone is rarely sufficient to change behavior, and these early approaches focused on future health consequences rather than more salient immediate consequence of substance use.[99,100]

Subsequently, in the 1970s, the National Institute on Drug Abuse provided nationwide technical assistance, planning, and funding for primary prevention of substance use, including use of tobacco, alcohol, and other drugs, among school-age adolescents.[89] This initiative funded a series of school-based substance use prevention education studies to improve adolescents' development, strengthen self-esteem, clarify positive values, and provide alternative activities to substance use. However, most of these early trials had substandard experimental designs and did not provide a strong evidence base for effectiveness.[89]

In the 1980s, prevention research brought knowledge from epidemiology, social psychology, and biology to bear on prevention topics and identified common risk and protective factors for the use of tobacco and alcohol across multiple domains, including individual, peer, family, and community.[89] Interventions based on psychological theories (Table 8.1) included role-playing activities to build refusal skills, education to correct misperceptions that substance use is normative and approved by peers, media literacy, and requests asking students to publicly commit to their peers to abstain from substance use. This convergence of scientific research from multiple fields produced more effective intervention approaches, as well as evaluation methodologies such as group-randomized trials, assessments of the fidelity of program delivery, and common measures of substance use and related constructs.[89] For example, Albert Bandura's social learning theory[61] postulated that people are influenced by the modeled behavior of their peers, especially when the peer modeling behavior receives reinforcement and/or if the peer has high social status. Therefore, prevention programs often included role-playing activities in which students watch a facilitator refuse an offer of tobacco or alcohol from a peer and then practice refusing an offer themselves.[89] Studies also found that adolescents and young adults commonly overestimate the prevalence of substance use among their peers,[101] leading them to use substances in an attempt to conform to their distorted perception of peer norms. Therefore, interventions were developed that aimed to correct adolescents' misperceptions that substance use is normative and to demonstrate that most of their peers, in fact, do not use substances and do not approve of substance use.[89] William J. McGuire's social inoculation theory[98] posited that people can learn to counterargue against misleading advertising messages, so interventions also focused on media literacy.[89] Because cognitive dissonance theory[84] posited that people are motivated to act in ways that are consistent with their previous statements, interventions encouraged adolescents to publicly commit to their peers not to use substances.[102] Programs also continued to include information about the negative short-term and long-term physical and social consequences of substance use.[89]

Selected notable events that have affected tobacco and alcohol use norms are summarized in Figure 8.2.

Figure 8.2 Timeline of Selected Tobacco and Alcohol Prevention-Related Contextual Milestones



Note. FDA = Food and Drug Administration; RICO = Racketeer Influenced and Corrupt Organizations.
Source: American Lung Association 2024,[103] Bellou and Bhatt 2013,[104] Campaign for Tobacco-Free Kids,[105] Farrelly et al. 2017,[106] Griffin 2021,[107] H.R.6543 — 91st Congress (1969–1970),[108] Mothers Against Drunk Driving 2024,[109] NIAAA,[110] PACE Recovery Center 2017,[111] U.S. Department of Health Education and Welfare 1964,[81] USDHSS 2012,[20] USDHSS 2014,[74] USDHHS 2016,[112] Truth Initiative,[113] White 2016.[114]

Although smoking prevalence began to decrease after the 1964 Surgeon General's report[81] demonstrated the causal relationship between smoking and lung cancer,[115] alcohol use was actually increasing.[116,117] The minimum legal drinking age (MLDA) had been 21 in most states since the end of Prohibition, but in the 1970s, 29 states lowered their alcohol purchase ages to 18, 19, or 20, reasoning that young adults who were old enough to vote and be drafted into the military were also old enough to purchase and use alcohol.[118]

By the mid-20th century, the medical community was aware of the acute and long-term hazards of alcohol use,[119] and alcohol use disorder (previously referred to as alcoholism) was increasingly understood through a disease model of addiction, with contributing genetic, neurological, psychological, and social mechanisms.[120,121]

In the early 1980s, advocacy organizations such as Mothers Against Drunk Driving (MADD) highlighted the deadly effects of alcohol-impaired driving on victims of car crashes.[122] MADD's emphasis on the effect of alcohol-impaired driving on other people (e.g., pedestrians, passengers, and drivers of other cars) was an effective rebuttal to the argument that individuals should have the freedom to engage in behaviors that could damage their health,[122] similar to counterarguments to tobacco use that highlight the adverse effects of secondhand smoke and the right of nonsmokers to breathe smokefree air.[123] In response to grassroots movements to reduce deaths caused by drunk drivers, many of whom were adolescents or young adults, the U.S. Congress passed and President Reagan signed into law the 1984 National Minimum Drinking Age Act (23 U.S.C. § 158). The Act required states to establish a minimal legal purchase age and drinking age of 21 by 1987 or face a decrease in federal highway funding. Faced with a loss of federal funding, states with lower age limits returned their MLDA to age 21 by 1988,[118] where it has remained ever since.[124] Subsequent laws aimed at reducing alcohol-impaired driving have focused on reducing the allowable blood alcohol content for drivers, particularly for individuals under the legal drinking age[125]; making criminal and driving records available to police during traffic stops; eliminating plea bargains for charges of driving under the influence; and instituting mandatory jail sentences, higher minimum fines, liability laws for servers, and highway sobriety checkpoints.[122]

In the latter half of the 20th century, antismoking social norms were increasing, with information about the adverse health consequences of smoking appearing in the media,[115] and tobacco advertisements were banned from radio and television after the Public Health Cigarette Smoking Act was signed into law in 1970.[126] At the same time, television featured numerous alcohol commercials but few anti-alcohol messages, except for those about the dangers of alcohol impaired driving.[126] Advertisements for both tobacco and alcohol were still prevalent on billboards and in magazines.[127,128] The Master Settlement Agreement[129] between 52 state and territory attorneys general and the 4 largest U.S. tobacco companies, signed in 1998, permanently removed tobacco advertising from billboards, transit and outdoor advertisements, and outdoor signs, and some tobacco companies agreed to remove tobacco advertisements from magazines with substantial adolescent readership.[24,130] The contrast between the numerous tobacco prevention efforts that appeared through the end of the 20th century and the fewer alcohol prevention efforts reflects how youth tobacco use tended to be prioritized over youth alcohol use as a public health issue.

The following sections of this chapter describe the interventions that have been implemented at various levels (e.g., individual, group, population), highlighting evidence about whether alcohol-focused and general substance use prevention programs are effective in preventing tobacco use and whether tobacco-focused and general substance use prevention programs are effective in preventing alcohol use.

Individual-Level Interventions

Substance use primary prevention programs have typically been delivered to groups rather than individuals for the sake of efficiency and cost-effectiveness,[15] with the exception of one-to-one counseling by physicians, dentists, or other health professionals.[131] Individual-level interventions to prevent substance use, including those targeted to tobacco and alcohol use, have evolved in parallel with the available technology. Some more recent interventions use technologies such as smartphone apps, educational video games, virtual reality, or text messaging to deliver prevention content to individuals.[133-138] Notably, the timing of the evolution of individual-level prevention interventions toward technology-based delivery has coincided with the increased availability and use of e-cigarettes[112], which has resulted in several interventions focused on preventing e-cigarette use.

A 2017 systematic review of individual substance use prevention interventions delivered via wireless technology (e.g., web-based, text messaging, smartphone applications) identified 12 studies.[132] The interventions were based on diverse theoretical frameworks including the theory of planned behavior, social cognitive theory, self-determination theory, motivational enhancement therapy, the elaboration likelihood model, and dialectical behavioral therapy. Most interventions delivered text messages to enhance motivation to avoid substance use, increase intrinsic motivation, provide social support, or reinforce the participant's commitment not to use substances. Some interventions also provided opportunities for self-monitoring, guidance on planning for situations with a high likelihood of substance use, or feedback comparing the individual's actual substance use behavior with their plan not to use substances. However, among the 12 studies identified, 10 focused on alcohol use, 2 focused on use of other substances, and none studied tobacco use. In addition, rather than focusing on prevention among young people not using substances, the interventions generally targeted adolescents, young adults, and adults who reported already using substances.[132]

A 2019 systematic review and meta-analysis of e-health interventions conducted in schools but delivered at the individual level analyzed 11 studies that focused on changing multiple health behaviors, including improving nutrition, increasing physical activity, and decreasing screen time, smoking, and alcohol use.[134] These interventions used a variety of electronic intervention approaches including electronic self-assessments, online games that simulated decision-making, personalized feedback based on self-reported behavior, and multimedia education including videos and animation. This meta-analysis found that the interventions had no significant effect on smoking or alcohol use, although they did have beneficial effects on physical activity and fruit and vegetable intake. The authors noted that most of the interventions were based on the stages of change model, which is typically used in cessation interventions, rather than social influence theories, which are typically used in substance use prevention programs.[134]

A 2021 systematic review and meta-analysis focused on online interventions that targeted multiple risk behaviors including alcohol use and smoking among adults ages 18 and older (primarily college students).[135] Of the 11 studies reviewed, 3 addressed both alcohol use and smoking, as well as nutrition and physical activity.[140-142] All three studies recruited interested participants by advertising broadly to a population (either college students or community members), rather than randomly assigning all members of a population to an experimental condition. Thus, the results of these studies are generalizable only to people who are motivated to participate in a health-focused intervention and have access to a computer or smartphone. Among the three studies, a study of college undergraduates[140] and a community-based study[142] reported no significant effects on alcohol use or smoking. The other study of undergraduates[141] demonstrated a significant reduction in the prevalence of current smoking but not alcohol use.

A 2022 meta-analysis of prevention programs that focused on both tobacco and alcohol use among adolescents and young adults identified only two individual-level interventions.[136] One of these interventions, RealTeen,[143] recruited a nationwide sample of 13- and 14-year-old girls via Facebook advertisements and randomly assigned them to a 9-session online intervention or a control group. The intervention focused on improving girls' knowledge about drug use and improving their cognitive and behavioral skills for coping with stress, managing moods, making decisions, maintaining a healthy body image, and refusing offers of drug use. This intervention showed significant prevention effects on cigarette smoking and binge drinking. An earlier version of this intervention, delivered to adolescent girls,[144] had significant prevention effects on past-month alcohol use but not past-month cigarette smoking. In a 2023 rapid review of digital substance use interventions for young people, 6 of the studies focused on prevention and incorporated web-based programs, video games, and virtual reality.[137] In addition to the 2018 RCT[143] that demonstrated that RealTeen was effective in preventing cigarette smoking and binge drinking, this review[137] also identified a 2019 study[145] that reported 2- and 3-year follow-up results from the 2018 study. At the 2- and 3-year follow-up points, the RealTeen intervention remained effective in reducing cigarette smoking, but not alcohol use.[145]

Just-in-time adaptive interventions (JITAIs) are individual-level electronic interventions that are specifically targeted to the individual's risk of using substances at a particular moment in time, defined by the individual's location (e.g., near a tobacco or alcohol retailer) or state of mind (e.g., self-reported craving).[146] A 2022 review of 14 studies that used JITAIs to deliver substance use prevention messages found 7 alcohol prevention studies and 4 tobacco prevention studies, but no studies that addressed both substances.[138] The JITAIs were all delivered to individuals with previous substance use who were attempting to remain abstinent. Although the interventions were too varied to draw conclusions about overall effectiveness, the authors concluded that JITAIs are a promising strategy for substance use prevention. However, given the intensive data collection and analysis necessary to deliver interventions exactly when they are needed,[146] the high participant burden, and the expense of paying participants for adherence to the data collection prompts,[138] it is unlikely that JITAIs can be used broadly to prevent substance use in the general population.

A 2024 systematic review of interventions to prevent e-cigarette use identified 14 studies that delivered content at the individual level.[133] These interventions included three video game interventions that allowed adolescents to role-play decision-making and e-cigarette refusal, and two interventions that sent text messages about the health risks of e-cigarettes and nicotine dependence. The interventions and measures were too variable to conduct a meta-analysis. All of the video game and text messaging interventions produced significant improvements in e-cigarette knowledge and harm perceptions, but they did not produce significant changes in e-cigarette use or intentions to use.[133] Furthermore, none of these studies addressed or measured alcohol use.

Summary of Individual-Level Interventions

Individual-level tobacco and alcohol prevention programs for adolescents and young adults vary widely, from multisession, self-paced curricula to interventions that seek to distract participants from craving tobacco and alcohol by playing a video game. Although more identified individual-level interventions focus on alcohol prevention than on tobacco prevention, all the interventions could potentially be adapted to focus on both substances. The small number of individual-level prevention programs may be attributed to several factors: the relative newness of individually tailored electronic interventions, the difficulty of recruiting and following individuals relative to groups (e.g., schools), and the high participant burden. With the proliferation of smartphones and social media, it is possible that more individual-level interventions will be developed in the future, especially if barriers to recruitment, adherence, and frequent data collection can be overcome.

Group-Level Interventions

Health education and prevention programs for adolescents and young adults typically occur in settings where many individuals can be reached simultaneously, including primary and secondary schools, college campuses, and communities.[15] Some interventions are delivered to families, either to one family at a time or to groups of parents and adolescents. This section reviews school-based, college campus-based, and family interventions that address tobacco and alcohol.

School-Based Interventions in Kindergarten Through Twelfth Grade

Substance use prevention programs for elementary school students (typically ages 5–10), middle school students (typically ages 11–13), and high school students (typically ages 14–18) often involve multiple classroom sessions with group activities to help students learn the short- and long-term physical and social consequences of substance use, practice skills to refuse substance use offers, and restructure normative beliefs about the prevalence and acceptability of substance use.[15,20] Prevention curricula have also focused on increasing various personal and social competencies including self-esteem, decision-making, stress management, and effective communication. Numerous programs have been developed to teach these concepts and skills to adolescents and young adults. These strategies are described in detail in the 2012 Surgeon General's report[20]; although much of the research on school-based prevention was conducted prior to 2012, this report's conclusions remain relevant today (see Table 8.2).

However, youth and young adults' tobacco use behaviors have changed in several ways since 2012. First, the average age of cigarette smoking initiation has gradually increased from early adolescence to young adulthood.[147] Second, youth and young adults' tobacco use now reflects the diversity of the tobacco marketplace; since 2014, e-cigarettes have consistently been the most commonly used tobacco product by youth.[1,148,149] Though research on the efficacy of e-cigarette prevention programs is still emerging, the 2016 Surgeon General's report, *E-Cigarette Use Among Youth and Young Adults,*[112] suggested that "over 50 years of research offers a strong body of evidence on the effectiveness of certain tobacco prevention and control measures. Much of this evidence can also be applied to e-cigarettes."[112,p.243] For example, expanding smokefree policies to include e-cigarettes, regulating advertising of e-cigarettes to adolescents, raising taxes on e-cigarette products, and limiting access to e-cigarettes through approaches such as restrictions on retail licensure for the sale of e-cigarettes may be effective. However, the bulk of existing tobacco prevention programs, including most of those reviewed in this section, have not been directly tested for efficacy at preventing e-cigarette use.

Table 8.2 Selected Conclusions From Reports of the Surgeon General and the National Academies of Science, Engineering, and Medicine Related to Tobacco Use Among Adolescents and Young Adults

2012 Surgeon General's Report, *Preventing Tobacco Use Among Youth and Young Adults*

- Cigarette smoking by youth and young adults has immediate adverse health consequences, including addiction, and accelerates the development of chronic diseases across the full life course.
- Prevention efforts must focus on both adolescents and young adults because among adults who become daily smokers; nearly all first use of cigarettes occurs by 18 years of age (88%), with 99% of first use by 26 years of age.
- Advertising and promotional activities by tobacco companies have been shown to cause the onset and continuation of smoking among adolescents and young adults.
- After years of steady progress, declines in the use of tobacco by youth and young adults have slowed for cigarette smoking and stalled for smokeless tobacco use.
- Coordinated, multicomponent interventions that combine mass media campaigns, price increases including those that result from tax increases, school-based policies and programs, and statewide or community-wide changes in smokefree policies and norms are effective in reducing the initiation, prevalence, and intensity of smoking among youth and young adults.

2016 Surgeon General's Report, *E-Cigarette Use Among Youth and Young Adults*

- E-cigarette use among youth and young adults has become a public health concern. In 2014, current use of e-cigarettes by young adults 18–24 years of age surpassed that of adults 25 years of age and older.
- E-cigarettes are now the most commonly used tobacco product among youth, surpassing conventional cigarettes in 2014. E-cigarette use is strongly associated with the use of other tobacco products among youth and young adults, including combustible tobacco products.
- The use of products containing nicotine poses dangers to youth, pregnant women, and fetuses. The use of products containing nicotine in any form among youth, including in e-cigarettes, is unsafe.
- E-cigarette aerosol is not harmless. It can contain harmful and potentially harmful constituents, including nicotine. Nicotine exposure during adolescence can cause addiction and can harm the developing adolescent brain.
- E-cigarettes are marketed by promoting flavors and using a wide variety of media channels and approaches that have been used in the past for marketing conventional tobacco products to youth and young adults.

2018 NASEM Report, *Public Health Consequences of E-Cigarettes*

- There is substantial evidence that e-cigarette use increases risk of ever using combustible tobacco cigarettes among youth and young adults.
- Among youth and young adult e-cigarette users who ever use combustible tobacco cigarettes, there is moderate evidence that e-cigarette use increases the frequency and intensity of subsequent combustible tobacco cigarette smoking.
- Among youth and young adult e-cigarette users who ever use combustible tobacco cigarettes, there is limited evidence that e-cigarette use increases, in the near term, the duration of subsequent combustible tobacco cigarette smoking.

Note. NASEM = National Academies of Science, Engineering, and Medicine.
Source: Quoted from USDHHS 2012,[20] USDHHS 2016,[112] NASEM 2018.[150]

A 1988 meta-analysis of 47 school-based smoking prevention programs and 29 alcohol prevention programs published in the 1970s and 1980s found small but significant prevention effects for both tobacco and alcohol use.[151] This analysis found that most interventions also increased tobacco and alcohol knowledge and in the short-term (but not long-term) improved attitudes. The authors recognized that the greater effect sizes observed in the smoking prevention studies may have been partially attributable to anti-smoking messages in the media and the emergence of smoking regulations.[151]

After the 1980s, the literature on school-based tobacco prevention programs began to diverge from the literature on alcohol prevention programs. Whereas earlier reviews were likely to report effects on both tobacco and alcohol use, subsequent reviews began to focus on one substance or the other, making comparisons across substances difficult. It is also important to view these early studies in their social context. These studies coincided with an increase in antismoking norms and media containing information about the adverse health consequences of smoking, especially after the Master Settlement Agreement in 1998.[115,129] In contrast, alcohol advertising and promotion in the media, including television, remained prevalent and subject to limited federal regulation.[152-157] In the next two sections, school-based tobacco use and alcohol use prevention programs are discussed separately.

School-Based Tobacco Prevention Programs

By 1993, there were 94 published evidence-based school-based interventions for smoking prevention with rigorous evaluations.[158] A 1993 meta-analysis divided these interventions into four categories: rational (provide information about substances and their effects), developmental (enhance social-emotional constructs such as self-esteem, self-reliance, decision-making, and interpersonal skills), social norms (correct overestimations of prevalence and acceptability), and social reinforcement (teach adolescents to recognize and resist peer pressure to use).[158] This meta-analysis found that although interventions in all four categories produced significant improvements in smoking behaviors in early follow-ups, programs with a social reinforcement orientation produced the largest effects on smoking prevention, followed by developmental and social norms programs; traditional programs with a rational orientation showed the smallest effect.[158] A 1998 meta-analysis of 120 school-based substance use prevention programs found that programs with interactive activities involving face-to-face peer interactions were more effective than traditional didactic programs.[159]

A series of Cochrane reviews illustrates the evolving knowledge of what works in school-based smoking prevention programs.[160-162] Thomas and colleagues produced Cochrane reviews of the effectiveness of school-based smoking prevention programs in 2002,[160] 2006,[161] and 2013.[162] The first review found inconclusive evidence for effectiveness: half of the highest quality studies reported beneficial effects on smoking, but half did not.[160] The second review found some evidence that social influences-based programs were effective in preventing smoking, but many studies produced null effects.[161] The authors called for more rigorous evaluations of programs that used an approach that combined social influence and social competence interventions. The 2013 review of 134 studies concluded that there was no significant overall short-term prevention effect (1 year or less), but the studies with longer follow-up times showed significant prevention effects.[162] Furthermore, interventions that combined social influences and social competence curricula had a beneficial effect, whereas programs that emphasized information only or social influences only were not effective. A 2015 systematic review and meta-analysis by the same researchers focused on 50 RCTs.[163] This meta-analysis reached conclusions similar to those of the 2013 Cochrane review; studies with more than 1 year of follow-up showed significant smoking prevention effects, whereas studies with shorter follow-up did not. Among the studies with long-term follow-up, the average reduction in smoking initiation among participants who never smoked was 12%. Importantly, this evidence excludes programs that are unable to conduct longer-term follow-up studies due to lack of institutional support. It is unlikely that long-term follow-up data collection alone made these programs more effective. Instead, it is likely that programs that had the resources to conduct long-term follow-up were better resourced and had institutional and community support. This review also supported the superiority of interventions that emphasized both social competence and social influences over those that emphasized information only or social influences only. Furthermore, this analysis also found that booster sessions and peer-led activities did not enhance program effects with 1 year of follow-up or less.

Overall, the evidence suggests that school-based tobacco prevention programs that emphasize social competence and social influences are effective in preventing smoking initiation in adolescents, although the effect sizes are small, and some studies showed null effects. It is important to note that these trials were conducted before the rapid increase in prevalence of e-cigarette use among youth in the early 2010s.[1,148,149] Most identified evidence-based tobacco prevention programs focused on cigarettes (the most commonly used tobacco product by youth prior to 2014)[1,148,149] and targeted adolescents (i.e., ages 12–18), rather than young adults (i.e., ages 18–25). All of these reviews focused on tobacco use; none reported program effects on alcohol use.

Several school-based interventions have been developed or adapted to focus on e-cigarette prevention among adolescents. These include CATCH My Breath[164] and Youth Engaged Strategies for Changing Adolescent Norms! (YES-CAN!).[165] These interventions have shown short-term effects on e-cigarette-related knowledge, attitudes, and beliefs.[165-169] A meta-analysis of nine e-cigarette prevention programs for adolescents conducted between 2014 and 2020 found that most interventions also focused on other tobacco products and were based on behavioral theories such as social cognitive theory, social influences, and the theory of planned behavior.[170] Among the 5 studies that reported e-cigarette prevention outcomes, only 3 found significant effects of the intervention (versus control) in reducing e-cigarette use at follow-up (6 to 36 months), 1 found significant increases in e-cigarette use in the intervention condition relative

to control at follow-up, and 1 found no significant difference in e-cigarette use, leading to an overall nonsignificant effect of the interventions on e-cigarette use. The most effective interventions were those that included multiple components such as classroom education, teacher training, and parent education. The effective interventions were the United States-based CATCH My Breath,[164] the Sweden Tobacco-Free Duo,[171] and the England Learning Together programs.[172] However, these studies did not assess intervention effects on alcohol use.

The Stanford Tobacco Prevention Toolkit is an online educational resource to prevent use of all tobacco products among adolescents.[173] The toolkit includes lessons and activities that can be delivered in group settings such as schools or after-school programs. It includes modules about e-cigarettes but does not address alcohol use. This toolkit is unique in that it was developed using a community-based participatory research framework with input from students and school personnel.[174] The toolkit includes curricula, lessons, activities, quizzes, materials, and resources, all available for free online. As of 2020, the online toolkit had been accessed by more than 186,000 users,[174] but an RCT of its effectiveness has not yet been published as of 2026. A small evaluation study conducted without a control group in one Kentucky high school found that exposure to the toolkit produced short-term (same day) improvements in knowledge about e-cigarettes, perceived harms and benefits of e-cigarettes, perceptions of addictiveness, refusal skills, and intentions to use, as well as decreased agreement that tobacco companies are truthful regarding their products' addictiveness and negative health effects.[175] However, this single-day study could not assess changes in e-cigarette use. A small study in Alabama using a pre-post design found improvements in e-cigarette knowledge and perceived addictiveness and decreased intent to try e-cigarettes between pre-test and post-test following a five-session in-person e-cigarette prevention curriculum based on the Standford Tobacco Prevention Toolkit.[176]

School-Based Alcohol Prevention Programs

School-based alcohol prevention programs evolved in parallel with school-based smoking prevention programs and used many of the same theoretical perspectives and intervention strategies, including correcting students' overestimation of alcohol use prevalence, modeling skills to resist an alcohol offer, and encouraging students to make a public commitment not to use alcohol.[177] They also used traditional strategies such as providing information about the consequences of alcohol use, fostering self-esteem, and offering life skills training.[177] Whereas many smoking prevention programs focused only on tobacco, most alcohol prevention programs focused on other substances in addition to alcohol.[177] Even some programs that advertised themselves as alcohol prevention programs (e.g., the Adolescent Alcohol Prevention Trial[178]) also addressed tobacco and other substances. A small number of programs (e.g., the Alcohol Misuse Prevention Study[179]) focused solely on alcohol use and did not assess effects on tobacco. A 2012 Cochrane review of prevention programs identified 11 school-based studies that evaluated alcohol-specific interventions[180]; 6 of these studies found significant prevention effects on alcohol use. This Cochrane review also evaluated 39 trials that focused on substance use in general, not just alcohol. Among these trials, 14 (36%) found significant prevention effects on alcohol use. A 2010 meta-analysis of 6 alcohol prevention curricula for younger adolescents (ages 10–15) found significant prevention effects against both alcohol and cannabis use for programs that used a comprehensive approach, integrating refusal skills, self-management skills, and social skills, rather than an information-only approach.[181] A 2016 systematic review of 70 studies, representing 40 alcohol prevention programs, concluded that the interventions with

the most evidence for effectiveness across multiple trials were the Climate Management and Treatment Education (CLIMATE) Schools program in Australia and the Project Adolescent Learning Experiences in Resistance Training (ALERT) and All Stars programs in the United States.[182] A 2015 meta-analysis of 17 studies examined the effectiveness of school-based brief alcohol interventions (i.e., those that provided <5 hours of programming).[183] These brief interventions differ from school-based tobacco prevention curricula in that they emphasize cognitive decision-making processes rather than other constructs that have been demonstrated successful for tobacco prevention. Hennessy and Tanner-Smith (2015)[183] found that these brief school-based interventions effectively prevent or reduce alcohol use, specifically when delivered individually rather than in a group setting, or when programs used motivational enhancement components.

A 2011 qualitative review of reviews concluded that effective alcohol prevention programs, primarily delivered to middle school students but sometimes to high school or elementary school students, had several key characteristics.[184] Effective programs (1) are theory driven, based on the social influences model; (2) address social norms and reinforce that alcohol use is not common or acceptable among adolescents; (3) build skills to resist pressure to use alcohol; (4) use interactive teaching techniques; (5) use peer leaders to facilitate program delivery; (6) involve parents and other members of the community; (7) are delivered across multiple sessions and multiple years; (8) provide adequate training for facilitators; and (9) are culturally and developmentally appropriate for the students. However, all of these reviews focused on program effects on alcohol use without considering effects on tobacco use.

School-Based Programs That Addressed Multiple Substances

Numerous studies have implemented school-based programs to prevent the use of multiple substances including tobacco and alcohol, as well as influence other health-related behaviors. Because these programs have been reviewed extensively,[136,185,186] this section describes the findings from several meta-analyses and Cochrane reviews. This section also describes several programs that have been used extensively in school settings and several programs with demonstrated effectiveness in preventing tobacco use (see Table 8.3).

A 2018 Cochrane review[185] examined individual-, family-, and school-level interventions for adolescents that targeted risk behaviors from at least two of the following categories: substance use (including alcohol, tobacco, cannabis, and other substances), risky sexual behaviors, antisocial behaviors, vehicle-related risk behaviors, self-harm, gambling, and physical activity/diet-related behaviors. This review included 70 studies; 28 (40%) were universal (i.e., delivered to all students, not just students who used substances) school-based studies. Behaviors that were most frequently addressed included alcohol use, drug use, antisocial behavior, and tobacco use. Most of the effective school-based programs were comprehensive; they included multiple years of prevention programming beginning in elementary or middle school and used multiple components, including social skills training, parent education, and changes to the school environment. This review concluded that universal school-based interventions are effective in preventing tobacco and alcohol use, especially when they are reinforced by interventions in the family and/or community.[185]

The findings from MacArthur and colleagues' 2018 review[185] of interventions aimed at multiple risk behaviors contrast with findings from a more focused 2022 meta-analysis of interventions that addressed use of at least two substances (including tobacco, alcohol, cannabis, and other substances) but no other risk behaviors.[136] Tinner and colleagues' 2022 meta-analysis[136] of interventions among young people ages 8–25 found moderate quality evidence that the 41 universal school-based interventions had little or no short-term benefit (up to 12 months) in reducing alcohol, tobacco, cannabis, or other drug use. However, it is important to note that the wide age range of the participants in the included studies is a limitation in interpreting and drawing conclusions from these findings because the needs of younger and older adolescents differ according to developmental stage.

A 2020 systematic review took a slightly broader approach to assessing school-based substance use prevention programs, including prevention programs that addressed at least one substance (e.g., alcohol, cannabis, or other substances).[186] Interventions could address tobacco use in addition to use of other substances, but interventions that addressed only tobacco use were not included because the authors did not classify tobacco as a mind-altering substance.[186] The 90 identified studies represented 16 different prevention programs. Among these 16 programs, 4 targeted alcohol alone, 4 targeted alcohol and nicotine (with or without other targeted substances), 4 targeted all psychoactive substances, 2 targeted all abused substances, and 2 targeted alcohol and non-tobacco drugs. The evaluated programs with the largest combined sample sizes were the Drug Abuse Resistance Education (DARE) program, Project ALERT, Life Skills Training, Adolescent Alcohol Prevention Trial, and Project Choice. The programs that focused on alcohol use only included the Alcohol Misuse Prevention Study, Protecting You/Protecting Me, the School Health and Alcohol Harm Reduction Project (SHAHRP), and the Seventh Generation Program; none of the evaluations of the alcohol-focused programs measured tobacco use outcomes. Among the programs that targeted multiple substances, the following reported significant effects on tobacco use in RCTs in the United States: the Adolescent Alcohol Prevention Trial,[187,188] Life Skills Training,[189-194] and Project ALERT.[195,196] Several of the widely used interventions are reviewed in this section.

Some school-based prevention programs that originally targeted one substance have been expanded to incorporate other substances. For example, Project Towards No Tobacco Use (TNT) originally focused on preventing cigarette smoking among middle school students.[197,199] TNT was one of the first school-based tobacco prevention programs to be evaluated rigorously in group-randomized trials. Sussman and colleagues (1993)[199] and Dent and colleagues (1995)[197] evaluated the 1- and 2-year effectiveness of 3 prevention strategies: teaching refusal skills, building awareness of social value misperceptions, and providing information on the physical consequences of tobacco use. In a large RCT of 48 middle schools, smoking prevention effects were strongest in schools that were assigned a combination of all 3 strategies.[197,199] These studies provided more evidence that comprehensive, multicomponent strategies prevent adolescent tobacco use.

TNT was later adapted to address multiple substances and became Towards No Drug Abuse (TND).[200] The program was adapted for implementation in alternative high schools, which typically have a higher prevalence of substance use than traditional high schools, and students may be diverted to these schools because of substance use, failing grades, or previous disciplinary actions. TND focuses on preventing use of tobacco, alcohol, cannabis, and other

drugs, and the program follows the same basic structure and uses the same prevention strategies as TNT (e.g., motivation, skills training, decision-making). Although TND was developed from an effective tobacco prevention program (TNT), the initial evaluation of TND produced significant prevention effects on alcohol use but not on tobacco use.[198] At 5-year follow-up, significant prevention effects persisted for use of cocaine, hallucinogens, stimulants, inhalants, and other drugs but not for tobacco or alcohol use.[201] A replication of this study in traditional high schools found prevention effects for alcohol and other drug use but not for use of cigarettes or cannabis.[202] A third evaluation in continuation high schools (alternative high school programs for students who are at risk of not graduating) found significant prevention effects for all substances, but only when the curriculum was delivered in a group setting by trained health educators.[202] TNT has since been updated to include content about e-cigarettes. This new program, Towards No Nicotine, is being evaluated in an after-school setting,[203] but as of 2026 results are not yet available.

Project ALERT. Project ALERT is a substance use prevention curriculum delivered in middle schools.[195,196] It is based on several theories, including the health belief model, which emphasizes cognitive factors that motivate behavior; social learning theory, which emphasizes social norms and social influences; and social cognitive theory, which emphasizes self-efficacy.[196] Like other prevention curricula, Project ALERT focuses on the physical and social consequences of using substances, correcting overestimates of substance use prevalence, and building self-efficacy to refuse substance use offers.[195] The program focuses on cigarettes, alcohol, and cannabis.[204] Tremblay and colleagues (2020)[186] reviewed 12 evaluations of Project ALERT. Among these studies, one[196] demonstrated prevention effects on alcohol, and two[195,196] demonstrated prevention effects on tobacco. However, one study reported that Project ALERT decreased tobacco use among students who experimented with tobacco but increased tobacco use among those who used tobacco regularly.[204] The other eight studies reported no significant effects on tobacco or alcohol use, or did not measure either tobacco or alcohol use.

DARE. DARE is a widely disseminated substance use prevention program for elementary, middle, and high school students that is delivered by police officers.[205] DARE was developed in 1983 to prevent tobacco, alcohol, and other drug use, and quickly spread to school districts nationwide. In 1994, a review of DARE programs reported that 52% of U.S. school districts had adopted DARE,[206] and in 2007, approximately 75% of school districts were implementing DARE.[205] Like many other substance use prevention programs, DARE focuses on enhancing psychosocial skills to reduce substance use, including social skills (e.g., peer-pressure resistance), building self-esteem, informing attitudes toward drug use and attitudes toward police, and improving family bonding. DARE was controversial because it was disseminated widely in the absence of RCTs demonstrating its effectiveness and because some communities were not comfortable with police officers delivering a substance use prevention program.[207] A 1994 early meta-analysis of eight evaluation studies of DARE found a significant effect for tobacco use but not for alcohol or cannabis use, and effect sizes were substantially lower than those of other more interactive classroom-based social influences programs.[208] The largest effects were on knowledge, social skills, and positive attitudes toward police officers. A 2009 meta-analysis of 20 evaluations of DARE,[205] which updated previous meta-analyses,[208] found very small effect sizes for the prevention of tobacco, alcohol, or other drug use. In both meta-analyses, the authors concluded that DARE is largely ineffective for preventing tobacco, alcohol, and other drug use, despite its widespread use.[205,208]

In 2018, DARE partnered with an evidence-based prevention program, *keepin' it REAL*, to adapt *keepin' it REAL* to be delivered by DARE officers to 5th grade students.[209] The adapted program produced statistically significant reductions at 1-year follow-up in the prevalence of past-month alcohol use, drunkenness, and e-cigarette use when compared with virtual controls (i.e., expected substance use prevalence data derived from baseline age and psychosocial predictor percentiles, and anticipated trajectories).[209] However, effects on cigarette smoking were not analyzed because the prevalence of cigarette smoking among participants was too low.

College Campus Tobacco and Alcohol Prevention Programs and Policies

As of 2022, approximately 39% of young adults ages 18–24 were enrolled in college.[210] Compared with young adults who are not in college, college students report a higher prevalence of heavy alcohol consumption, including binge drinking,[211] but a lower prevalence of tobacco use.[212] Most middle school or high school prevention programs have focused only on tobacco use or on use of multiple substances; in contrast, on college campuses, there has been an abundance of alcohol prevention programs but fewer tobacco prevention programs.[213] Before passage in 2019 of legislation that raised the federal minimum age of sale of tobacco products from 18 to 21 years of age, college students in many states were old enough to purchase tobacco legally. Therefore, colleges may not have had legal justification for preventing students from purchasing or possessing tobacco. Colleges could, however, implement smokefree policies (described in detail in the section "Policy Influences on Tobacco and Alcohol Use"), though the efficacy of those policies depended on effective enforcement, which could be difficult.[214,215] In addition, college students (and other young adults) do not often experience the serious adverse health effects of tobacco use, because these effects typically occur after decades of tobacco use.[74] In contrast, alcohol use on college campus often produces immediate hazards both to students who use alcohol and others around them; campus alcohol use often manifests as binge drinking, which has been linked with unintentional injury, physical and sexual assault, and driving under the influence of alcohol.[216] In addition, campus alcohol use poses negative consequences for the schools themselves, such as increased costs for health care and security, campus vandalism, and damage to a school's reputation.[217]

College Campus Tobacco and Alcohol Prevention Programs

Many college campuses implement programs to prevent or reduce alcohol use by students,[218] most of whom are below the legal age to purchase alcohol, which has been age 21 since enactment of the 1984 National Minimum Drinking Age Act.[219] A 2007 meta-analysis of 62 campus-based interventions identified a wide variety of strategies including alcohol education, decisional balance, expectancy challenge, feedback on consumption, feedback on expectancies or motives, feedback on problems, goal setting, identification of high-risk situations, educational materials, motivational interviewing, moderation strategies, normative comparisons, skills training, and values clarification (most campus-based programs used multiple strategies).[220] These interventions produced reductions in alcohol use quantity, frequency, and problems at short-term (4–13 weeks post-intervention) and intermediate-term (14–26 weeks post-intervention) follow-ups, and effects on alcohol use frequency and alcohol-related problems persisted at long-term follow-up (more than 27 weeks). This review concluded that the most effective interventions included motivational interviewing, normative feedback, feedback on

expectancies and/or motives for drinking alcohol, and/or used decisional balance exercises. However, these interventions were less effective among students who drank heavily than among students who drank occasionally. This review and a subsequent 2012 review by the same authors[221] both concluded that in-person or face-to-face interventions were more effective than computer-delivered interventions. However, these studies did not examine tobacco use as an outcome.

Many colleges implement substance use prevention programs as part of larger student wellness initiatives. A 2019 review of 88 college campus substance use prevention programs found that 70 programs focused on alcohol use; only 9 programs focused on tobacco use, and the rest focused on use of other drugs.[213] This analysis found support for the effectiveness of the alcohol prevention programs on multiple alcohol-related outcomes (e.g., alcohol consumption, blood alcohol content, negative consequences related to alcohol consumption, binge drinking). However, the effectiveness of tobacco prevention programs was mixed, with only 4 (44%) of the studies reporting a reduction in tobacco use frequency. The review included studies that measured tobacco use frequency, but it did not evaluate complete abstinence from tobacco, which is often the main outcome variable in tobacco cessation studies. The relative lack of tobacco prevention programs on college campuses is a missed opportunity to prevent disease caused by tobacco, especially because between 2002 and 2018, the average age of transition from smoking initiation to daily smoking shifted closer to young adulthood from adolescence.[147]

Some alcohol prevention programs on college campuses focus on students who have violated campus alcohol policies, for example, by possessing alcohol or being intoxicated on campus. These programs include mandatory participation in alcohol education groups, parental notification, individual counseling including motivational interviewing, and computer-delivered alcohol education.[222] A 2016 meta-analysis of 31 mandated alcohol prevention programs found that the interventions decreased alcohol consumption (e.g., drinks per week/month, drinks per drinking interval, peak consumption, frequency of drinking days and heavy drinking, typical and peak blood alcohol content) and alcohol-related problems in the short term (within 3 months), but these effects diminished over time.[223] None of these studies assessed tobacco use as an outcome.

College Campus Tobacco and Alcohol Policies

Many college campuses have implemented tobacco-free and/or alcohol-free campus policies alone or in conjunction with health education programs. A 2015 meta-analysis of 19 studies of the effectiveness of tobacco-free college campus policies through 2013 found that the majority of students and faculty supported these policies.[224] However, only two of the studies assessed policy effects on student tobacco use[225,226]; both studies found statistically significant reductions in student tobacco use, and Seo and colleagues (2011)[226] also found a decrease in students' perception that smoking is normative among their peers. A 2017 publication reviewed 11 evaluations of tobacco-free college campus policies that assessed student tobacco use.[227] However, only three of these studies[225,226,228] were longitudinal; the rest reported student tobacco use outcomes after a campus tobacco-free policy was instituted, compared colleges with and without tobacco-free policies, or compared colleges with comprehensive versus partial tobacco-free policies. This review concluded that more comprehensive tobacco policies (i.e., broad tobacco-free campus policies rather than policies solely restricting tobacco use in or near

buildings) are more successful in reducing student tobacco use and increasing smoking cessation attempts. This review noted several threats to validity, including reliance on students' self-reports of tobacco use, varying measures of tobacco use across studies, and study populations of primarily White students. Interestingly, campuses that instituted designated smoking areas as an anti-tobacco policy had a higher smoking prevalence than campuses that implemented comprehensive tobacco-free policies, perhaps because designated smoking areas offer students who smoke a place to socialize with fellow students, which reinforces the smoking behavior. The social aspect of smoking is likely particularly relevant in the college context. None of the studies in this 2017 review focused on e-cigarette use.[227]

A 2024 review identified 54 studies of smokefree or tobacco-free policies on college campuses published from 2005 to 2022.[229] A total of 15 studies found that smokefree or tobacco-free campus policies were significantly associated with reduced smoking prevalence over time. The review included 6 studies that measured e-cigarette use outcomes. Five of the six studies found that smokefree or tobacco-free campus policies were associated with increased initiation of e-cigarette use or e-cigarette use prevalence among students, with the remaining study finding no effect.[230-234] However, the studies examining e-cigarette outcomes were conducted from 2012 to 2019,[230-234] a period that coincided with a rapidly increasing prevalence of e-cigarette use overall.[235] As a result, it is unclear whether the increase in e-cigarette use was due to the overall increase in e-cigarette use occurring at that time, to students switching from cigarettes to easier-to-conceal e-cigarettes in response to the tobacco-free campus policy, or to a combination of these and other factors. None of these studies assessed the impact of campus tobacco policies on alcohol use, although one study found that frequent alcohol consumption had a negative effect on compliance with policies.[236]

Very few studies have focused on the effects of tobacco-free policies on alcohol use. Two small studies found that new smokefree bar policies in communities surrounding college campuses did not affect college students' alcohol use[237] or intention to go to bars that served alcohol.[238]

Alcohol-free college campuses are uncommon, and few studies have addressed the effectiveness of campus alcohol-free policies.[239,240] One study found that the prevalence of student alcohol use was lower on campuses with alcohol-free policies,[240] but this might reflect self-selection of alcohol-using students into campuses without alcohol-free policies or underreporting of alcohol use on campuses that prohibit alcohol consumption. A 2005 nationally representative study of college students across 120 universities found associations between self-reported campus alcohol policies and smoking behavior.[241] Adolescents and young adults on campuses where their peers rated ease of alcohol availability as very easy and/or alcohol policy enforcement as low were more likely to report cigarette smoking than those on campuses where alcohol was perceived as less available or alcohol policy enforcement was perceived as greater. A limitation of this study is that the authors measured students' perceptions of alcohol availability and alcohol policy enforcement, rather than the actual availability of alcohol and implementation of specific policies on campuses.

Many colleges ban alcohol use in specific situations or settings, such as sporting and tailgating events, fraternity and sorority houses, and dormitories.[242] A 2007 review of policies listed numerous strategies to restrict alcohol access among underage students, for example, banning alcohol at parties, banning beer kegs and self-service of alcohol at parties, requiring an

identification card for alcohol purchase, and banning alcohol advertising and sponsorship on campus.[243] This review concluded that multicomponent strategies as well as social norms campaigns are promising but need more rigorous evaluation. Research produced through 2017 has been used to inform the College Alcohol Intervention Matrix,[217,244] which evaluates the effectiveness of individual interventions and environmental policies for preventing alcohol-related problems. The College Alcohol Intervention Matrix suggests numerous policies and interventions that are rated as effective based on evidence in published, peer-reviewed studies, such as normative re-education or prohibiting alcohol sale or consumption at college sporting events. None of the studies identified in the literature search for this chapter evaluated the effect of alcohol policies on prevention of tobacco use. The literature on campus tobacco and alcohol policies tends to remain siloed, with studies of campus tobacco policies not measuring alcohol use, and studies of campus alcohol policies not measuring tobacco use.

Family-Based Interventions

Some substance use prevention programs are delivered to families to improve parenting behaviors that are protective against adolescent substance use, such as supportive parent-child relationships, positive discipline methods, rule-setting, monitoring and supervision, family advocacy for children, and information seeking and support for the benefit of children.[245,246] The Community Preventive Services Task Force (known as "the Community Guide") recommends family-based interventions as effective for preventing substance use among youth, including tobacco and alcohol. Their systematic review included studies where the family-based intervention was delivered primarily to families with youth aged 10 to 14 years old. [246] In addition, a systematic review of economic evidence on family-based youth substance use prevention programs suggests that the societal benefits of the programs outweighed societal costs.[247] Interventions with published results include the Family Check Up[248-253] (for youth ages 2–17), RaiseSmoke Free Kids[254] (also known as ONE Step, targeting parent behavior to help children), Parents Who Care[255] (for preadolescents to adolescents), Strengthening Families Program[256-260] (for families and children from birth to age 17), and Familias Unidas[261-266] (for adolescents). The types of interventions that have shown significant prevention effects are parent training (primarily cognitive behavioral parent training), family skills training (including training on parenting, children's social skills, and family relationships), and family therapy (structural, functional, or behavioral family therapy).[246,267] Family interventions can be classified as universal (delivered to the general population of families), selected (delivered to families at high-risk of experiencing problems), or indicated (delivered to families that are already experiencing problems).

Family-Based Tobacco Prevention Programs

A 2015 meta-analysis of family-based smoking prevention interventions identified 27 studies, including 6 that focused on tobacco use alone; 2 that focused on tobacco and alcohol; 3 that focused on tobacco, alcohol, and cannabis; 1 that focused on alcohol use alone; 2 that focused on tobacco and cardiovascular health; 1 that focused on general substance use; 2 that focused on HIV and unsafe sex and associated tobacco use outcomes; and 10 that focused on family function, child development, and modifying adolescent behavior.[268] The analysis found moderate quality evidence that family-based interventions, used alone or with school-based programs, are effective against adolescent smoking initiation. The most effective interventions were those that

encouraged authoritative parenting (high warmth and high demandingness, including interest in the adolescent, care for the adolescent, and rule setting), as compared with authoritarian (low warmth and high demandingness) or permissive (high warmth and low demandingness) parenting styles.[268,269]

Family-Based Alcohol Prevention Programs

Meta-analyses have examined the effectiveness of family-based alcohol prevention programs. Family-based alcohol prevention programs typically involve parenting interventions designed to foster positive parenting, parent-child communication, and parental monitoring of adolescents' activities.[246] Some programs focus on parents, whereas others include activities for parents and adolescents, to complete either separately or together. A 2008 meta-analysis of 18 studies of 9 RCTs found that family-based alcohol prevention interventions were generally effective in preventing adolescent alcohol use initiation, past-month use, and frequent use.[270] The authors noted that family-based alcohol prevention programs are more effective when all families within a given school system are receiving the intervention, potentially because intervention effects are amplified within a school setting as intervention recipients' attitudinal and behavioral changes feed into new norms and peer influences. This result suggests that family-based prevention programming should be part of a comprehensive alcohol prevention package. They also noted that studies varied in how they analyzed families that were lost to attrition. As a result, it is unclear whether prevention effects generalize to families who drop out of family-based interventions. This qualification is important because adolescents in these families are typically at higher risk for substance use.[270]

A 2019 Cochrane review of 46 studies examined the effects of family-based interventions on adolescent alcohol use delivered to adolescents with no known risk factors (universal interventions), those at increased risk of alcohol use (selective interventions), and those already using alcohol (indicated interventions).[271] Most of the interventions focused on prevention of alcohol, tobacco, and other drug use; 10 focused on alcohol use only. This review found no significant pooled effect of family-based intervention as compared with either no intervention or standard of care on prevalence or frequency of alcohol use, and the results were similar for universal and selected or indicated interventions. Interventions had only a very small effect on the volume of alcohol consumed by adolescents.

Family-Based Interventions to Prevent Both Tobacco and Alcohol Use

A 2016 meta-analysis of 116 studies of family-based substance use prevention programs categorized the interventions according to the components of family function that they emphasized, including parental monitoring and behavior management; fostering school success; positive family relationships; substance use knowledge, attitudes and values; self-regulation and stress management; problem solving; resisting peer pressure psychoeducation; ethnic identity; and future orientation.[272] This meta-analysis found that programs that included an emphasis on positive family relations or future orientation had significant effects on adolescent substance use. Effect sizes were similar for use of tobacco, alcohol, and other drugs. The authors also concluded that the most effective family-based programs included activities for both parents and adolescents, not just for parents.[272]

A 2022 meta-analysis of parenting interventions to modify multiple behaviors (including tobacco, alcohol, or other drug use; physical activity; diet; and sedentary behavior) among adolescents included 46 studies of 36 RCTs.[273] These programs typically addressed multiple behaviors, often targeting both tobacco and alcohol use or both diet and physical activity. Among the 36 trials, 20 targeted both tobacco and alcohol use, and 1 targeted use of alcohol and other drugs but not tobacco use. All studies that focused on tobacco and alcohol use included face-to-face group sessions for parents to provide emotional and practical social support. Other parent-based intervention strategies included goal setting, problem solving, feedback and monitoring, parenting skills, information about consequences of use, and behavioral practice of parenting skills. This meta-analysis found that the reviewed programs had no significant prevention effects on adolescent tobacco or alcohol use.[273]

Summary of Group-Level Interventions

School-based interventions for preventing tobacco and alcohol use can be effective, particularly when they contain components to address social influence, social competence, and perceived social norms; are interactive; and more intensive in terms of dose and content, including multiple levels of interventions (e.g., at the family and community levels) and multiple components (e.g., activities to teach students to recognize and resist peer pressure, parental education) that begin early (e.g., in elementary school or middle school) and persist over time. In elementary, middle, and high schools, as well as in colleges, tobacco prevention programs and alcohol prevention programs are often administered and evaluated separately, and there is a lack of literature addressing cross-substance program effects. Family-based interventions are effective for preventing adolescent substance use, including tobacco and alcohol use. Some evidence indicates that effectiveness of family-based prevention interventions may be bolstered by including the school context.

Population-Level Interventions

Some prevention programs have targeted entire communities (variously defined as cities, counties, states, etc.). This section describes population-level interventions, including multilevel programs and mass media campaigns, that have addressed both tobacco and alcohol use.

Multilevel Programs

Multilevel programs deliver prevention messages in multiple settings including schools, communities, and the media. Several reviews have evaluated the effectiveness of multilevel alcohol prevention programs. For example, a 2008 review of school-based, family-based, and community-based interventions aggregated the findings of 25 earlier reviews and synthesized the evidence for effectiveness of 127 different interventions.[191] Of these interventions, 12 met the criteria for most promising evidence of effectiveness against alcohol use, and 29 met the criteria for mixed or emerging evidence. This review noted that the effects of many interventions on alcohol use could not be disentangled from those for use of other substances because many studies reported composite measures of substance use, rather than program effects on the use of specific substances.[191] This section describes some multilevel programs for tobacco and/or alcohol and general substance use prevention that reported cross-substance outcomes, with selected programs rated as effective or promising by Blueprints or the Department of Justice highlighted in Table 8.3.

Examples of Multilevel Programs That Focused on Alcohol Use and Reported Effects on Tobacco Use

Project Northland. One intervention that focused on alcohol but also examined effects on tobacco was Project Northland.[274-276] Project Northland was created specifically to focus on alcohol use because the authors noted that earlier studies of multisubstance prevention programs typically showed significant effects on tobacco use but not on alcohol use, suggesting that alcohol-specific programming was needed.[276] Project Northland was implemented in rural Minnesota, in communities that were predominantly White. This prevention program for 6th through 8th grade adolescents was comprehensive and included school-based curricula, parent education, homework activities for adolescents and parents to complete together, and community-wide task forces mobilized in support of alcohol-related local ordinances and resolutions. The intervention produced a reduction in adolescent alcohol use and tobacco and alcohol co-use (defined as having used alcohol in the past month and having smoked cigarettes on more than one or two occasions), although the effects on tobacco use alone did not reach statistical significance.[276] This study demonstrated the power of a comprehensive, multicomponent, multiyear intervention to reduce both tobacco and alcohol use. However, because all the program components were implemented simultaneously, it is difficult to identify an active ingredient (e.g., school curricula, family activities, legislation promoted by community task forces, or a combination of these interventions). Furthermore, large budgets are required to implement programs in many school districts and to mobilize an entire community to prevent adolescent substance use.

Project Northland was replicated among 6th through 8th grade students in an urban, low-income setting in Chicago; most of the students were African American or Hispanic.[275] The areas included in this study likely had substantially more alcohol advertising and more alcohol retailers than the original Project Northland setting, and parents perceived that other problems, such as gang violence, were much more important than adolescent alcohol use. In a study evaluating the adapted program, tobacco use was not assessed separately but was included in a tobacco and other drug use composite variable. This study reported no significant effects on alcohol, tobacco, or other drug use, although a secondary analysis found that higher participation in the home-based intervention component was significantly associated with lower scores on a composite variable representing any use of any substance. The authors attributed the lack of effect of the intervention in urban Chicago to several factors including higher student attrition, ubiquitous exposure to alcohol retailers and advertising, competition with other challenges facing the community (e.g., drug trafficking, gangs, violence, and housing concerns) low community acceptance of the problem of teen alcohol use, and limited time among residents to volunteer for community action teams.[275] Additionally, this study highlights the importance of replicating the findings of substance use prevention programs in different community settings.

Midwestern Prevention Project. The Midwestern Prevention Project was a comprehensive substance use prevention program implemented in Kansas in the 1980s.[277] The program included mass media programming, a school-based educational curriculum, parent education and organization, community organization, and health policy components, with these components rolled out over a 6-year period. In the first 2 years of the project, 22,500 sixth- and seventh-grade adolescents from 42 schools received mass media programming and a school-based educational program component that included parental involvement in homework.[277] Analyses indicated that

the use prevalence for all three substances (cigarettes, alcohol, and cannabis) was significantly lower at 1-year follow-up in the intervention condition relative to the delayed intervention condition.[277] The success of this project could be because prevention messages were reinforced community-wide with interventions that spanned school curricula, a media campaign, community events, and new health policies. However, the effectiveness of each individual component of the program is difficult to evaluate because all components were delivered simultaneously.

Communities That Care. Communities That Care (CTC) is a comprehensive, community-based program to prevent substance use and other unhealthy behaviors among adolescents.[278] The program involves five phases. In phase 1, key leaders assess community readiness to adopt the CTC approach to prevention. In phase 2, the community forms a prevention board, and in phase 3, the community board uses community-specific epidemiological data to assess community risks and strengths and conduct a resource assessment of prevention programs already occurring in the community. The board then assesses the epidemiology of problem behaviors in their community, identifies risk and protective factors that influence outcomes, and prioritizes the risk and protective factors and outcomes that should be addressed. In phase 4, the community board selects empirically supported prevention programs to address the prioritized risk factors and outcomes. During phase 5, the board supports the implementation of these programs, evaluates their effectiveness in the community, and revises their plans as necessary. The Community Youth Development Study[279,280] tested the effectiveness of the CTC intervention in a community-randomized trial in which 24 communities across the United States were randomized to receive the CTC program or a control condition. The study followed 5th grade students in the communities after the implementation of the program. One study investigated the effect of the intervention on substance use initiation among the students through 7th grade.[279] However, because the authors aggregated all substances (including alcohol, cigarettes, cannabis, inhalants, cocaine, barbiturates, ecstasy, and prescription drugs) into a single substance use variable, it was not possible to separate out the intervention's effects on tobacco and alcohol use. The intervention and control groups did not significantly differ in substance use during 6th and 7th grade, leading the authors to conclude that the intervention was not effective in preventing substance use.[279] However, another study from the same trial found significant prevention effects for both tobacco and alcohol use by 8th grade;[280] over the following 2-year period, 6th graders in the control communities were 60% more likely to initiate alcohol use and 79% more likely to initiate cigarette smoking between 7th and 8th grade than 6th graders in the CTC intervention condition. An 8-year follow-up study of this cohort found that the CTC program significantly prevented both tobacco and alcohol use.[281] A 10-year longitudinal study of this cohort, collected when participants were approximately 21 years old, reported significant prevention effects on any "gateway drug" use (i.e., alcohol, tobacco, or cannabis)[282]; however, the effects on tobacco and alcohol use individually were not significant. Discrepant findings on the effectiveness of this intervention in preventing substance use may be attributed to differences in coding of substance use variables and analytic strategies across the studies. Additionally, the program's effect may diminish over time, so that its effects decrease as students grow older.

Table 8.3 Selected Effective or Promising Tobacco and Alcohol Prevention Interventions

Intervention	Target population	Published effects on tobacco use	Published effects on alcohol use	Evidence rating: DOJ Model Programs Guide	Evidence rating: Blueprints for Healthy Youth Development
Communities That Care[278-282]	Grades 5–9	Among Grade 5 cohort: • By Grade 7: No significant effect on substance use initiation (alcohol, cannabis, cigarettes, or other illicit drugs). • By Grade 8: Initiation of cigarette smoking (aOR = 1.79, 95% CI = 1.09–2.92) and smokeless tobacco use (aOR = 2.34, 95% CI = 1.34–4.09) was significantly higher in the control communities than intervention communities. • By Grade 12: Abstinence from cigarette smoking (aRR = 1.13, 95% CI = 1.01–1.27) was higher in the intervention communities than control communities. • By Age 21 (11 years after initial implementation): No significant main effect on tobacco use initiation. Among male participants only, there was a significant increase in the likelihood of sustained abstinence from tobacco use among the intervention group (*d* = 0.23; adjusted risk ratio = 1.30 [95% CI, 1.07–1.68]).	Among Grade 5 cohort: • By Grade 7: No significant effect on substance use initiation (alcohol, cannabis, cigarettes, or other illicit drugs). • By Grade 8: Initiation of alcohol use (aOR = 1.60, 95% CI = 1.05–2.44) was significantly higher in the control communities than intervention communities. • By Grade 12: Abstinence from alcohol use (aRR = 1.31, 95% CI = 1.09–1.58) was higher in the intervention communities than control communities. By Age 21 (11 years after initial implementation): No significant main effect on alcohol use initiation. Significant intervention effects were observed for any "gateway drug" use (alcohol, tobacco, cannabis) (*d* = 0.26; adjusted risk ratio = 1.49 [95% CI, 1.03–2.16]).	Promising	Promising
Familias Unidas[262]	Grade 8 and Hispanic	From baseline to 30-month follow-up, past-30-day substance use increased from 15% to 25% among intervention participants, but increased significantly more among participants in the control group, from 13% at baseline to 34% at the 30-month follow-up.	From baseline to 30-month follow-up, past-30-day substance use increased from 15% to 25% among intervention participants but increased significantly more among participants in the control group, from 13% at baseline to 34% at the 30-month follow-up.	Effective	Promising
Midwestern Prevention Project[277]	Grades 6–7	1-year follow-up: Past-month cigarette smoking prevalence was significantly lower among participants in the intervention condition (17%) than participants in the delayed intervention condition (24%).	1-year follow-up: Past-month alcohol use prevalence was significantly lower in the intervention condition (11%) than in the delayed intervention condition (16%).	Effective	Not dissemination ready

Table 8.3 (continued)

Intervention	Target population	Published effects on tobacco use	Published effects on alcohol use	Evidence rating: DOJ Model Programs Guide	Evidence rating: Blueprints for Healthy Youth Development
Project Northland[274-276]	Grades 6–8	Implementation in rural Minnesota, 3-year follow-up: • No significant main effect on lifetime cigarette smoking (defined as more than 2 or 3 lifetime occasions of use). • There was a significant difference in cigarette and alcohol co-use (lifetime cigarette smoking and past-month alcohol use) between the intervention and control groups (14.3% versus 19.4%, $p < .03$). Implementation in Chicago, 30-month follow-up: • Tobacco use was not measured independently. • There was no significant main effect on the composite drug use outcome (alcohol, tobacco, and cannabis use).	Implementation in rural Minnesota, 3-year follow-up: • The prevalence of recent alcohol use was significantly lower in the intervention group than in the control group: past week alcohol use, 10.5% versus 14.8%, $p < .05$; past-month alcohol use, 23.6% versus 29.2%, $p < .05$. • There was a significant difference in cigarette and alcohol co-use (lifetime cigarette smoking and past-month alcohol use) between the intervention and control groups (14.3% versus 19.4%, $p < .03$). Implementation in rural Minnesota, 7-year follow-up: There was a significantly smaller increase in past-month alcohol use in the intervention group than in the control group during phase 1 (Grades 6–8) but not phase 2 (Grades 11–12): phase 1, mean growth rate 0.11 vs 0.16, $p < .01$; phase 2, mean growth rate 0.13 vs. 0.20, $p = .07$. There was a significantly smaller increase in binge drinking (5 or more drinks in a row in the past 3 weeks) in the intervention group than in the control group during both phases: phase 1, mean growth rate 0.05 vs 0.08, $p < .01$, phase 2, mean growth rate 0.09 vs 0.18, $p = .02$. Implementation in Chicago, 30-month follow-up: • There was no significant main effect on alcohol use alone. • There was no significant main effect on the composite drug use outcome (alcohol, tobacco, and cannabis use).	Promising	Not dissemination ready
Project Towards No Tobacco (TNT)[197,199]	Grades 6–8	• 1-year follow-up: Combined implementation of 3 intervention strategies had the largest effects on initiation and weekly use of cigarettes and smokeless tobacco. • 2-year follow-up: Combined implementation of 3 intervention strategies had the largest effects on weekly use of cigarettes and smokeless tobacco.	N/A	Promising	Inconclusive evidence

Table 8.3 (continued)

Intervention	Target population	Published effects on tobacco use	Published effects on alcohol use	Evidence rating: DOJ Model Programs Guide	Evidence rating: Blueprints for Healthy Youth Development
Project Towards No Drug Abuse (TND)[198,200,201,202]	Grades 9–11	Continuation high schools, 1st curriculum version, 1-year follow-up: No significant effects on past-30-day cigarette smoking. Continuation high schools, 1st curriculum version, 5-year follow-up: No significant effects on past-30-day cigarette smoking. Traditional high schools, 1st curriculum version, 1-year follow-up: No significant effects on past-30-day cigarette smoking. Continuation high schools, 2nd curriculum version, 1-year follow-up: A significant effect (27% relative reduction in past-30-day cigarette smoking) was observed only for the health educator-led condition.	• Continuation high schools, 1st curriculum version, 1-year follow-up: Past-30-day alcohol use was lower at follow-up among students with higher pretest alcohol use levels. • Continuation high schools, 1st curriculum version, 5-year follow-up: There were no significant effects on past-30-day alcohol use. • Traditional high schools, 1st curriculum version, 1-year follow-up: The intervention resulted in a significant reduction (12%) in past-30-day alcohol use. • Continuation high schools, 2nd curriculum version, 1-year follow-up: A significant effect (9% relative reduction in past-30-day alcohol use) was observed only for the health educator-led condition.	Ineffective	Model
Strengthening Families Program[256-260,257,283]	Grade 6	• 1-year and 2-year follow-ups: Intervention participants were less likely than control participants to progress from nonuse to tobacco or alcohol use, or from use of 1 substance to use of both substances. • At Grade 10 (4-year) follow-up: Significantly reduced tobacco use, $p < .01$. • At age 21 (9-year) follow-up: Significant reductions in polysubstance use ($p < .05$) and marginally significant reductions in cigarette frequency ($p < .10$).	• 1-year follow-up: Lower alcohol initiation in intervention participants relative to control participants (Cohen's f = 0.26, $p < .05$). • 2-year follow-up: Lower alcohol initiation in intervention relative to control (Cohen's f = 0.29, $p < .01$). • At Grade 10 (4-year) follow-up: Significantly reduced alcohol use ($p < .01$). • At age 21 (9-year) follow-up: Significant reductions in polysubstance and drunkenness frequency ($p < .05$).	Promising	Promising

Note. aOR = adjusted odds ratio; aRR = adjusted risk ratio; CI = confidence interval; DOJ = Department of Justice; N/A = not applicable. Programs are listed alphabetically by program name. For DOJ Model Programs Guide, a rating of "promising" indicates that "Programs have some evidence indicating they achieve their intended outcomes," and a rating of "effective" indicates "programs have strong evidence indicating they achieve their intended outcomes when implemented with fidelity."[284] For Blueprints for Healthy Youth Developments, a rating of "promising" indicates that an intervention meets the "minimum standard of effectiveness," and a rating of "model" indicates that an intervention meets a higher standard than "promising" and provides a "greater confidence in the program's capacity to change behavior and targeted outcomes."[285] DOJ and Blueprints ratings reflect ratings as of August 2025.
Additional sources: Blueprints for Healthy Youth Development; U.S. Department of Justice, Office of Justice Programs.

Tobacco and Alcohol Prevention for Different Populations

Sociodemographic subpopulations, such as immigrant populations and/or groups that may experience discrimination, have additional risk and protective factors that must be considered when developing, implementing, or evaluating prevention interventions for tobacco and alcohol use.[286] These factors include acculturation, enculturation, parent-child acculturation discrepancies, discrimination, and cultural stress.[287] It is important to determine whether specific sociodemographic groups would benefit from culturally tailored substance use prevention programs, or whether culturally generic prevention programs would be equally effective across cultural groups. It is possible that culturally tailored programs, which incorporate culturally resonant characters, artwork, and language and address specific protective cultural values, could be even more effective.[288]

Indeed, there is evidence that tailoring prevention program content to the specific norms and values of a cultural group can improve program effectiveness. Programs that are adapted and culturally tailored for a specific population typically produce stronger prevention effects within that population than effects produced using the culturally generic version of the same program. A meta-analysis of 30 RCTs focused on interventions that had been culturally adapted for specific racial and ethnic groups found that the positive effect of culturally tailored interventions on substance use was larger when the control condition was a nontailored, active intervention than when the control condition was nonactive (e.g., waitlist control).[288] More than one-half of the interventions (53%) were culturally adapted for Hispanic adolescents, 23% were adapted for Black adolescents, and 23% were adapted for American Indian adolescents. Among the interventions tailored to Hispanic adolescents, most addressed acculturation issues (e.g., adolescents' bicultural competency, parental knowledge of U.S. cultural norms), cultural values, and ethnic identity, with some coverage of racial and ethnic discrimination and immigration-related challenges. The seven interventions tailored to Black adolescents generally focused on cultural socialization (e.g., building positive racial and ethnic pride and cultural pride) and preparation for racial bias (e.g., strategies to handle discrimination, culturally appropriate coping strategies). Interventions tailored to American Indian adolescents included culturally sensitive components that addressed cultural socialization (e.g., incorporating American Indian cultural symbols, cultural values, knowledge, stories, illustrations, and traditional practices into program content), learning about Tribal-specific histories, and promoting ethnic and cultural identity. This meta-analysis found significant prevention effects for both tobacco and alcohol use for intervention participants relative to control participants. Effects were significant for Hispanic, Black, and American Indian adolescents, and there were no significant differences in prevention effects across racial and ethnic groups in the overall effect size.[288]

Media Literacy Interventions

Some interventions attempt to counter the effects of tobacco and alcohol advertising by teaching media literacy—the ability to view an advertisement and understand its intended audience, the message, the message's producer, and who stands to benefit from the audience viewing the message. For example, individuals who see an advertisement that presents a person who smokes cigarettes or drinks alcohol as attractive would be able to understand that the tobacco or alcohol company producing the advertisement stands to benefit from consumers associating their product with attractiveness but that the product does not actually make people more attractive.

Media literacy interventions teach adolescents and young adults to recognize and understand the tactics used by advertisers to promote products to help the participants develop counterarguments against such advertising. A 2018 meta-analysis of 20 media literacy interventions (10 on smoking, 5 on alcohol use, and 5 on other health behaviors) found an overall significant positive effect on media literacy, attitudes, and behavioral intentions.[289] However, these studies did not assess behavioral outcomes longitudinally. None of the smoking media literacy studies assessed alcohol media literacy, and none of the alcohol media literacy studies assessed smoking media literacy.

Industry-Sponsored Adolescent and Young Adult Prevention Programs

The tobacco industry has sponsored adolescent tobacco prevention programs as part of corporate social responsibility efforts.[20,290-292] The stated purpose of these programs was to decrease tobacco use among adolescent and young adults; however, as noted in NCI Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control*, "the U.S. Surgeon General, the courts, and research studies analyzing previously internal tobacco industry documents have generally concluded that the actual purpose of the industry programs is to discourage legislation, regulation, and programming to effectively reduce youth tobacco use; to improve the tobacco industry's public image; to generate partnerships with youth-serving organizations; and to limit the threat of litigation."[291,p.290] Furthermore, NCI's Monograph 19 concluded that "tobacco industry youth smoking prevention campaigns have been generally ineffective in reducing youth smoking. Moreover, they may even have increased smoking in some subgroups of youth."[24,p.15] Specific strategies used by the tobacco industry, including self-help booklets for families; industry-sponsored, school-based prevention programs; industry/community partnerships; and industry-sponsored antismoking campaigns in the mass media, are described in detail in the 2012 Surgeon General's report[20] and the 2008 World Health Organization's report, *Tobacco Industry Interference with Tobacco Control*.[292]

Similarly, the alcohol industry has sponsored adolescent and young adult alcohol prevention programs. A content analysis of several alcohol prevention programs funded by the alcohol industry found that these programs emphasize themes of personal responsibility, moderate consumption, and individualized problem definitions.[293] Across these programs, with one limited exception, the authors found that, "a striking absence from the discourses is any mention of the role of the alcohol industry and marketing in youth drinking",[293,p.17] which they note stands in contrast to research linking alcohol marketing to underage alcohol consumption. Like tobacco industry prevention programs, alcohol industry prevention programs tend to shift responsibility from the alcohol industry to the individual to drink alcohol in moderation, to parents to prevent their children from accessing alcohol, and to adolescents and young adults to resist peer pressure.[293]

Overall, industry-sponsored tobacco and alcohol prevention programs appear to share several common characteristics. They disseminate programs and messages that are ineffective at changing behavior; they frame use as an individual choice for adults; they attempt to shift responsibility for adolescent and young adult use from the industry to adolescents, young adults, parents, and peers; they do not publish rigorous effectiveness data about their programs; and they serve to position the industry as a responsible actor in the effort to prevent adolescent and young adult use.[20,24,290-293]Although the tobacco and alcohol industries have historically worked

together on occasion to oppose restrictions on the use of both substances (e.g., coordinated opposition to excise taxes, and advertising limitations),[294] the review for this chapter did not find any published studies that investigated coordinated prevention programming, nor did it find studies on the effects of industry-sponsored tobacco prevention programs on alcohol use or vice versa.

Mass Media Prevention Campaigns

Many mass media campaigns are designed to reach large populations and are successful at changing health behaviors, including tobacco and alcohol use.[295] They often focus on determinants of behavior change such as knowledge of the risks of the behavior, attitudes toward the behavior, self-efficacy to change one's behavior, and perceived norms. Mass media campaigns disseminated at the local, state, and national levels have tended to address tobacco and alcohol use separately. Tobacco prevention mass media campaigns that have focused on preventing adolescents and young adults from initiating tobacco use and preventing progression to regular use include the FDA's The Real Cost tobacco prevention campaign[296-298] and the FinishIT campaign from the Truth Initiative (formerly the American Legacy Foundation).[299,300] Culturally tailored campaigns have also been developed, such as FDA's Next Legends campaign,[76-78] which addressed e-cigarette use among American Indian and Alaska Native adolescents ages 12–17, and FDA's This Free Life campaign,[301] which addressed cigarette smoking among sexual minority young adults. Alcohol-centered mass media campaigns have focused on specific alcohol-related risk behaviors, such as alcohol-impaired driving with the Drive Sober or Get Pulled Over campaign,[302] and on specific populations, such as adults who engage in heavy drinking with the Drink Less, Be Your Best campaign.[303] Alcohol-prevention mass media campaigns have also focused on parent education to reduce adolescent and young adult alcohol use, such as the "Talk. They Hear You" campaign,[304] which provides parents and caregivers of youth younger than age 21 with information and resources to discuss underage drinking and substance use. Although these programs may be effective at preventing use of the single substance of focus,[300,301] they are not designed to influence both tobacco and alcohol use, and evaluations of these programs rarely include measures of both tobacco and alcohol use to explore cross-substance effects.

These mass media prevention campaigns are typically delivered to individuals who are also exposed to marketing and mass media campaigns from tobacco and alcohol companies. NCI Tobacco Control Monograph 19 evaluated the role of mass media advertising (e.g., television, billboards), other marketing communications (e.g., merchandising, sponsorship), consumer marketing (e.g., product design, pricing, distribution), and stakeholder marketing (e.g., youth prevention, health warnings, media training) on tobacco use.[24] Based on this evaluation, the monograph concluded that tobacco companies use multiple and complex strategies to make tobacco use appear attractive and normative, which in turn encourages adolescent and young adult tobacco use. Monograph 19[24] also reviewed the literature on the effectiveness of mass media tobacco prevention campaigns and concluded that:

> Mass media campaigns designed to discourage tobacco use can change youth attitudes about tobacco use, curb smoking initiation, and encourage adult cessation. The initiation effect appears greater in controlled field experiments when mass media campaigns are combined with school- and/or community-based programming.[24,p.12]

Furthermore, NCI Tobacco Control Monograph 21 concluded that "well-designed and -implemented anti-tobacco mass media campaigns are effective in improving understanding about the health consequences of tobacco use, building support for tobacco control policies, strengthening social norms against tobacco use, and reducing tobacco consumption among youth and adults."[291,p.303] Neither NCI Monographs 19 nor 21 addressed the effects of anti-tobacco media campaigns on alcohol use.

Several other reviews of the effectiveness of mass media tobacco prevention campaigns have concluded that they are generally effective in improving anti-tobacco attitudes and reducing tobacco use, especially when occurring in the context of other tobacco control programs and policies.[305-307] This is consistent with a major conclusion of the 2012 Surgeon General's report:

> Coordinated, multicomponent interventions that combine mass media campaigns, price increases including those that result from tax increases, school-based policies and programs, and statewide or community-wide changes in smokefree policies and norms are effective in reducing the initiation, prevalence, and intensity of smoking among youth and young adults.[20,p.8]

However, none of these reviews assessed the effectiveness of anti-tobacco media campaigns on alcohol use.

Reviews also have focused on the effectiveness of mass media alcohol prevention campaigns. A 2018 review of 24 studies[308] concluded that mass media alcohol prevention programs are memorable and are effective in improving knowledge about alcohol, but there is insufficient evidence that the programs reduce alcohol use. This review did not assess the effects of alcohol prevention media campaigns on tobacco use. A cross-sectional analysis of Monitoring the Future data found that adolescents who self-reported exposure to anti-tobacco or antidrug media campaigns were more likely to report use of both substances.[309] However, this finding may reflect confounding by overall media use if adolescents who spend more time using media (and are thus more likely to be exposed to anti-tobacco and antidrug campaigns) are also more likely to use tobacco and alcohol. It may also indicate that adolescents who use tobacco and alcohol are more likely to recall exposure to anti-tobacco or antidrug media campaigns. The cross-sectional analysis and use of self-reported media campaign exposure limit causal inferences. This study did not analyze exposure to anti-alcohol media campaigns.[309]

Unfortunately, these evaluations of mass media campaigns all focused on effects on a single substance. No evaluations assessed the effectiveness of tobacco prevention campaigns on alcohol use, and no evaluations assessed the effectiveness of alcohol prevention campaigns on tobacco use. A review of media campaigns to counter tobacco marketing concluded that tobacco countermarketing strategies may be adaptable to media campaigns to counter alcohol marketing,[310] but the review did not identify any studies that evaluated the effectiveness of tobacco-specific or alcohol-specific media campaigns in reducing the use of the other substance.

Other Population-Level Interventions

The NCI Tobacco Control Monograph 21[291] and the 2012 Surgeon General's report[20] highlighted the effectiveness of many population-level interventions, including comprehensive smokefree policies, tobacco excise taxes, policies restricting adolescent and young adult access to tobacco, bans on tobacco advertising, pictorial warning labels on tobacco products, and support for tobacco cessation programs. A 2023 systematic review and meta-analysis of population-level tobacco control interventions evaluated 144 studies for their effects on tobacco-linked adverse health outcomes.[311] The majority of these interventions were policy interventions, such as smokefree legislation (126 studies), tax or price increases (14 studies), multicomponent tobacco control programs (12 studies), and a minimum age law for sale of cigarettes (1 study). Some of these policy interventions are described in detail later in this chapter (e.g., Tobacco 21; see "Policy Influences on Tobacco and Alcohol Use" section), while others, such as tax-related policies, are addressed in chapter 9. Smokefree laws were associated with decreased rates of diseases caused by tobacco,[311] which was likely mediated by decreases in smoking prevalence and decreased exposure to secondhand smoke.[20] This meta-analysis did not report effects of any of these interventions on alcohol use.

Summary of Population-Level Interventions

Mass media campaigns can be an effective population-level prevention strategy for tobacco and alcohol use, particularly when the campaigns are complemented by other school- or community-based programming and additional policy changes. However, these campaigns are generally evaluated for impact on the explicitly targeted substance, whether tobacco or alcohol, and cross-substance effects are not considered. Media literacy interventions for youth, which have been evaluated for impacts on behavioral antecedents such as attitudes or intentions, may reinforce or heighten the effects of mass media campaigns that are designed for public education and to counter tobacco and alcohol industry campaigns.

Policy Influences on Tobacco and Alcohol Use

As of 2026, the literature on whether tobacco control policies prevent alcohol use and/or whether alcohol control policies prevent tobacco use is limited. However, as this monograph describes, tobacco use and alcohol use often occur (or co-occur) within the individual or same social context. For this reason, it is possible that policies aimed at preventing the use of one substance could also decrease the likelihood of the use of the other substance. Notably, studies have applied this approach to understand how laws addressing cannabis use shape other risk behaviors, including tobacco and alcohol use among young people.[312-315]

The following sections address laws that set age restrictions for the sale or purchase of tobacco and/or alcohol and approaches to help enforce those laws. Both sale and purchase laws focus on restricting access to tobacco or alcohol in retail environments; however, minimum legal sales age laws set restrictions on the sale of these products to individuals under a certain age, placing the responsibility on the retailer to ensure a product is not provided to someone underage. By contrast, minimum legal purchase age laws set restrictions on the purchasing of these products by individuals under a certain age, placing the responsibility on those buying the product to not

break the law. For example, the National Minimum Drinking Age Act of 1984 is a purchase law because it requires all states to restrict the age of purchase or public possession of alcohol to age 21, whereas Tobacco 21 is a sale law because it sets the legal age for sale (but not purchase) of tobacco products to age 21. However, numerous states have laws that also penalize the purchase of tobacco by those younger than age 21.[316]

Beyond the sale or purchase of a substance, laws may also address the possession or use of a substance. Possession laws restrict who can possess a substance or the conditions under which one can possess it. In addition to being a purchase law, the National Minimum Drinking Age Act is a possession law because it restricts possession of alcohol to people 21 years of age or older. The National Highway System Designation Act of 1995 is another example of a possession law; it requires that states implement policies categorizing individuals younger than age 21 who are operating a motor vehicle and have a blood alcohol content ≥0.02 (i.e., "internally possess" alcohol) as driving while intoxicated (versus the maximum blood alcohol content limit of 0.08 for those age 21 or older), or risk losing federal highway funds. States and localities may enact further restrictions regarding the sale, purchase, possession, or use of tobacco or alcohol.[317,318]

In the following sections, the impacts of the National Minimum Drinking Age Act, Tobacco 21 legislation, and vertical identification card laws are reviewed. These laws explicitly target the purchase of alcohol by and sale of tobacco to adolescents and young adults younger than 21 years of age and may therefore play a particularly relevant role in prevention. Chapter 9 discusses the effects of other policy-level interventions that influence a broader age range of people who use tobacco and alcohol, such as taxation, restrictions on point of sale, smokefree policies, and policies regarding licensing and distribution (see chapter 9).

Minimum Legal Drinking Age and Tobacco 21 Laws

As of 2026, the minimum legal sales or purchase age for tobacco and alcohol, respectively, in the United States is age 21. This section describes how minimum age purchase laws may affect tobacco use among adolescents and young adults, further highlighting opportunities to coordinate compliance checks and identification policies. The MLDA of 21 was first established in 1984, when the National Minimum Drinking Age Act was signed into law. Prior to its enactment the legal age for purchase of alcoholic beverages varied across states, with some states having already established a minimum purchase age of 21.[124] Tobacco sales age policies have varied over the past 100 years.[319] By 1920, 14 U.S. states restricted tobacco sales to adults age 21 or older. However, in the 1950s and 1960s, laws in some states were changed to allow adolescents as young as 16 to legally purchase tobacco. The Synar Amendment (section 1926) of the 1992 Alcohol, Drug Abuse, and Mental Health Administration Reorganization Act (Public Law 102-321) incentivized states to increase their tobacco sales age to 18 by restricting substance abuse prevention and treatment block grants to states with tobacco sales age of 18 or older. Consequently, all states increased their tobacco sales age to 18 or 19 by 1993. In 2019, Tobacco 21 (T21) provisions in the Further Consolidated Appropriations Act raised the federal minimum age for sale of tobacco products from 18 to 21; to enforce the T21 law, the FDA Center for Tobacco Products conducts compliance inspections of tobacco product retailers.[320] Echoing the policy-making history of MLDA, 19 states and Washington, D.C., had already increased their minimum sales age for tobacco products to age 21 prior to the enactment of the federal T21 law.[316]

The history of MLDA and T21 legislation provides unique contexts to understand the cross-substance effects of tobacco minimum sale age and alcohol minimum purchase age laws. It is important to note that the MLDA predated the federal T21 law by 35 years, so many evaluations of the alcohol policy occurred before the T21 law was implemented, and all evaluations of the T21 law occurred once the alcohol law had been in effect for a generation. Although the literature on cross-substance effects of these laws is generally sparse, several studies have used U.S. data to examine how MLDA laws affected adolescent and young adult smoking. One study using data from the 1977–1992 Monitoring the Future survey found that increasing the MLDA from age 18 to age 21 was associated with reduced smoking prevalence among 12th grade students by an estimated 3% to 5%.[321] In contrast, evaluations using the National Longitudinal Survey of Youth 1997 cohort suggested that reaching (versus being slightly younger than) the MLDA of 21 was not significantly associated with changes in smoking among 19- to 22-year-olds in the United States.[322,323]

Research on the effect of T21 policies on alcohol use is also sparse. One assessment of a T21 policy implemented in a small town in Massachusetts showed a downward trend in cigarette smoking and purchasing among adolescents in the community that was significantly greater than in comparison communities, but it did not observe a comparable trend for alcohol use.[324] A larger state-to-state examination revealed that young adult members (ages 18–20) of the U.S. military were less likely to consume alcohol if they were living in a T21 state.[325] Because these results were derived from 2016–2018 data, opportunities to delve into more recent data may provide a better understanding of the impact of T21 laws on drinking outcomes.

Vertical Identification Card Laws

Although more research is needed to elucidate the cross-substance impact of MLDA and T21 laws, the effectiveness of both policies depend on retailer compliance, monitoring of compliance (often by governments), and enforcement penalties for noncompliance.[326]

To help retailers identify underage individuals who try to purchase tobacco or alcohol and to prevent the use of fake or altered identification cards, between 1994 and 2009, 43 U.S. states changed the design of their state driver's licenses and identification cards to indicate whether a person is a minor or an adult, such as using a horizontal (landscape) layout for adults and a vertical (portrait) layout for minors.[104] This strategy led to state-level reductions in both tobacco and alcohol use by 16-year-old adolescents in a study using 1991–2009 Youth Risk Behavior Surveillance System data.[104] However, reductions were largely confined to the first and second year following the design change. The authors noted that even with sales restrictions in place, underage individuals can still access tobacco and alcohol. Additionally, studies of adolescents and retailers indicate that many underage adolescents are successful at purchasing tobacco or alcohol, suggesting that identification card checks may not be effectively enforced.[327-330] Indeed, adolescents often obtain tobacco and alcohol from social sources (e.g., older friends or relatives) rather than from retail sources,[331] and they may increasingly rely on social sources when vertical identification laws are in place.[332] False identification laws with scanner provisions (policies that

provides incentives to retailers and bar owners who use electronic scanners to ensure that customers are at least 21 years old) are shown to reduce underage drinking and traffic fatalities related to underage drinking and could potentially be adopted to reduce the underage purchase of cigarettes.[333-336] The proximity of tobacco and alcohol outlets and the presence of shared online vendors provide an opportunity to coordinate enforcement of tobacco and alcohol regulations.[337-339]

Challenges in Pursuing Macro-Level Prevention Interventions and Efforts to Overcome Them

In the past decade, few RCTs of substance use prevention programs assessing tobacco and alcohol use have been conducted in the United States. This work has been less common for several possible reasons. First, there may be less perceived need for new programs because multiple evidence-based programs are already available. Second, many schools have already purchased existing prevention curricula.[340] Third, schools could be less welcoming to researchers with programs and evaluations that will take up valuable required instructional time.[341,342] Finally, large-scale RCTs are resource intensive to conduct, requiring a great deal of time, money, and sufficient participation. However, these programs can become outdated if research is discontinued, reducing their effectiveness. Additionally, the emergence of novel tobacco products (e.g., e-cigarettes) and the changing marketplace of alcohol products (e.g., the popularity of alcoholic seltzers) require testing to ensure that existing programs are effective interventions for these new products.

There are practical challenges to conducting RCTs of programs and policies that must be carefully considered when evaluating effectiveness. For example, it is often impossible to randomize individuals to receive a specific policy or program. Therefore, researchers design studies using rigorous methods to account for this challenge. For school-based, family-based, and community-based prevention programs, this limitation can be addressed to some extent by conducting group-randomized trials, in which entire schools, families, or communities are randomly assigned to the treatment or control condition; statistical techniques can then be used to control for the intraclass correlation caused by randomizing groups rather than individuals.[343,344] However, this approach still requires that some schools or families be assigned to the control condition, whereby they provide data but do not receive an intervention. This can make it difficult to recruit schools, families, or communities because potential participants are aware that they may not receive an intervention. It can also be perceived as unethical to withhold interventions from groups that could benefit from them. Delayed intervention designs, in which the control group receives the intervention at the end of the study, can help to alleviate this problem. Additionally, group-randomized studies typically require extensive resources and staff.

Policy evaluations also face several challenges. Most important is that researchers do not control the timing of policy implementation, and consequently, sufficient capacity and timely resources are often needed to quickly conduct a baseline measurement before a new policy is implemented. Because policies are typically implemented at a large scale (e.g., entire countries, states, or cities), it is often difficult to identify an equivalent control group. To address these challenges, researchers may extrapolate from policy effects in another region to understand what may happen in their geographic context if the characteristics of the two regional entities are sufficiently similar. For example, data from Canada's ban on menthol cigarettes was used to model the potential effects of implementation of a similar policy in the United States.[345]

Lastly, much of the research reviewed in this chapter was conducted before 2010, and these studies have already been included in the meta-analyses cited in this chapter. Interestingly, very few RCTs of substance use prevention programs with measured outcomes of tobacco and alcohol use have been published since 2010. This work is continuing robustly outside the United States in countries such as Brazil,[346-348] Mexico,[349-351] Peru,[352] Australia,[353] and Israel.[354] Many of these international studies are collaborations with U.S. researchers who previously conducted similar research in the United States. Some studies test culturally adapted versions of the interventions or strategies that have been implemented in the United States.[355]

Chapter Summary

Because tobacco and alcohol use among adolescents and young adults share many of the same risk and protective factors at the individual, community and structural levels, programs to prevent the use of both products simultaneously might seem like an obvious strategy. Tobacco and alcohol prevention programs have evolved substantially over time in response to better understanding of the drivers of tobacco and alcohol use and associated biological, psychological, and social risk factors. However, tobacco prevention and alcohol prevention programs and research have largely remained siloed.

Modern technologies permit cost-effective dissemination of individual-level prevention programs (e.g., via smartphones) and allow for real time tailoring to accommodate an intervention to an individual's needs and offer real time data collection. Adaptations of tobacco prevention programs also focus on preventing use of e-cigarettes, the most commonly used tobacco product among youth since 2014. To date, these interventions have not assessed the effects of alcohol intervention on tobacco use, or vice versa.

Research has identified effective school-based prevention programs for reducing tobacco use and for reducing alcohol use among adolescents, but studies of the efficacy for programs that target multiple substances are less conclusive about effects on the individual substances. However, examination of qualities that make for effective group-based prevention programs suggests that prevention programs that are theory driven, have more interactive rather than traditional didactic components, and are more extensive (e.g., spread across many sessions), and extend beyond classrooms to involve the broader community, such as parents or media-based messaging, may be more effective than traditional programs. The 2012 Surgeon General's report concludes that multicomponent interventions where school-based programs and policies are implemented alongside other tobacco control measures, such as smokefree laws and mass media educational campaigns, are effective for preventing tobacco use among young people.[20] The effects of addressing alcohol use in that context are understudied.

Many colleges have tobacco- and alcohol-specific policies and have prevention programs for these substances. College campuses represent an important setting for tobacco- and alcohol-prevention and prevescalation (i.e., preventing escalation from experimentation to regular use) programs because the transition from smoking initiation to daily smoking is happening on average later in life, occurring more often in young adulthood than in adolescence, and because the minimum age for alcohol purchase or tobacco sale is 21 years. A review of interventions and

policies specific to college campuses, available through the NIAAA College Alcohol Intervention Matrix, identifies effective alcohol-related policies and programs. This approach and aggregation of policies and evidence for their effectiveness could be expanded to evaluate the cross-substance impacts of these policies and the effectiveness of these policies on tobacco use.

The evidence suggests that strategies that are effective in preventing the use of one substance are likely to be effective in preventing the use of the other substance, and that programs that address both substances are feasible and reasonably effective. Comprehensive prevention programs, which focus on countering social influences to use substances and establishing antisubstance use norms in communities, have effectively prevented tobacco use, alcohol use, or use of both substances. However, relatively few studies measure cross-substance prevention effects.

Given the intense competition for adolescents' and young adults' attention, both in school and in the media, prevention programs that target multiple substances can have an advantage over those that target a single substance. Programs that address multiple substances have the potential to influence use behaviors more efficiently. However, a potential drawback of programs that target multiple substances is that they could dilute the emphasis on each individual substance. In addition, programs that target multiple substances may have difficulty conveying the unique risks and importance of preventing use of both licit and illicit substances without encouraging comparisons of the levels of harm and legal consequences attributed to the different substances. The existing research is not sufficient to draw conclusions about whether a focus on multiple behaviors dilutes the effectiveness of prevention interventions.

Research Needs

Future research could measure the effects of tobacco prevention programs on alcohol use, and vice versa. In general, studies of interventions that focus on preventing tobacco use do not examine effects on alcohol use and vice versa. Even if a program focuses on one substance, researchers can include baseline and follow-up measures of use of other substances to identify cross-substance effects. For example, prevention programs that target use of e-cigarettes could assess effects of the program on alcohol use as well as use of other tobacco products. Further, measurement strategies such as ecological momentary assessment could bolster understanding of the situations in which prevention content is needed for one substance or multiple substances. This prevention content could then be provided with just-in-time adaptive interventions.

Future research studies could also directly compare single-substance prevention programs and multisubstance prevention programs. It is unclear whether a particular prevention program would be more effective or less effective if it focused on multiple substances versus one substance. Research is needed to understand how to achieve the optimal balance between preventing use of multiple substances (or promoting healthy behaviors in general) and preventing use of a single substance. No published studies have compared two programs that varied only on the substance(s) being addressed. Research is needed to test this question directly by comparing the effects of similar prevention strategies in similar populations, varying only the number of substances targeted.

Further efforts are needed to develop and test comprehensive approaches to prevent tobacco and alcohol co-use, such as approaches that address effects of prevention program components across multiple levels and communication channels, and common intrapersonal, interpersonal, community, and structural factors associated with both tobacco and alcohol use. Evidence-based programs for prevention of tobacco and alcohol use are most effective in the context of a comprehensive approach that involves parents and communities in the intervention strategy. In a broader context, comprehensive programs are more successful when embedded in an environment with strong tobacco control measures such as mass media campaigns to address tobacco use, policies to prevent minors' access to tobacco products, approaches to reduce marketing influences, and smokefree laws.

Additional research is needed to determine the extent of tailoring of prevention programs that is necessary to prevent tobacco and alcohol use across population subgroups and to address health disparities. Progress in preventing tobacco use has been uneven with some groups experiencing disproportionate risk of use (e.g., certain racial and/or ethnic groups, low-income individuals, and those with behavioral health conditions). For example, research is needed to understand how prevention programs should address common determinants for tobacco and alcohol use, such as depression, anxiety, or similar mental health conditions. There is some evidence that culturally tailored interventions are effective in preventing substance use, but few studies have compared the effectiveness of culturally tailored tobacco and alcohol prevention programs versus the effectiveness of more general (or non-tailored) programs. More research is needed to achieve an optimal balance between providing tailored prevention programs that address both tobacco and alcohol use for individual subgroups and providing general programs that are effective across the overall population.

Further monitoring of the effects of prevention programs and policies on tobacco and alcohol use would be beneficial. The impacts of both substance-specific and more general prevention programs on tobacco and alcohol use should be evaluated. Monitoring the use of multiple substances before and after prevention programs and policies are implemented can help researchers identify changes in product use over time; doing so requires study-specific surveys and broader surveillance research to assess use of alcohol, tobacco, and other substances. In addition, programs integrating tobacco and alcohol use prevention across both adolescence and young adulthood are needed.

Conclusions

1. **Interventions to prevent tobacco use and/or alcohol use employ approaches that are delivered at multiple levels including individual, group, community, mass media, and policy levels, as well as combinations of these levels.** Some large-scale interventions include prevention approaches at multiple levels (e.g., school and family, community, and media) and have been found effective for preventing tobacco and/or alcohol use. There is limited research to show whether multilevel interventions prevent tobacco and alcohol co-use because the outcomes are often aggregated and not reported separately.

2. **Studies of interventions that focus on preventing use of one substance have not typically examined effects on other substances.** Studies of single substance prevention programs may benefit from determining whether effects generalize to other substances.
3. **The evidence suggests that prevention programs that address tobacco and alcohol co-use are feasible, and some have shown effectiveness for preventing use of both substances.** However, many studies evaluating multisubstance or general substance use prevention programs have often only examined their effectiveness for preventing either tobacco or alcohol use, but not for preventing both tobacco and alcohol use.
4. **Prevention programs that specifically focus on tobacco or alcohol use have been implemented with different populations and with different goals, limiting the potential to assess their effectiveness for preventing co-use.** Tobacco prevention programs have been implemented mainly among children and adolescents and have focused on abstinence as a goal, whereas alcohol prevention programs are more frequently implemented among college student populations and have largely focused on reducing alcohol consumption to prevent or reduce binge drinking.
5. **It is unclear whether prevention programs that focus on a single substance are more effective or less effective than those that focus on multiple substances.** No studies have directly compared single-substance programs and multisubstance programs while holding all other factors constant.
6. **Policy interventions to restrict adolescent and young adult access to tobacco and alcohol, especially minimum legal sales or purchase age laws, have been effective in reducing adolescent and young adult use of these substances.** However, studies rarely assess the effect of alcohol access policies on tobacco use or the effect of tobacco access policies on alcohol use. As of 2026, the minimum legal age for alcohol purchase or tobacco sales is 21; policy evaluations and interventions to increase policy compliance and enforcement should measure their effects on both substances.

Appendix 8A Literature Search Strategy

The following searches were conducted in PubMed and Google Scholar from June to July 2023 to identify tobacco and alcohol prevention programs for adolescents and young adults: (tobacco OR nicotine OR smoking OR e-cigarette OR "electronic cigarette" OR alcohol) AND prevention AND (program OR intervention OR curriculum) AND (children OR adolescents OR "young adults" OR "college students"). Only RCTs, meta-analyses, systematic reviews, and reviews published in English were included. Additionally, studies had to include children (birth to age 18), adolescents (ages 13–18), or young adults (ages 19–24). The titles of these articles were downloaded into Excel and reviewed for relevance, defined as an evaluation of interventions to prevent tobacco and/or alcohol use. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

The following searches were conducted in PubMed and Embase from August to September 2023 to identify empirical studies of policy evaluations of crossover impact of tobacco or alcohol policies on tobacco and alcohol use outcomes. The keyword strings used in PubMed were: "youth" AND "alcohol policy" AND "tobacco use" [all fields] AND "policy" [title/abstract] = 419 results; "youth" AND "tobacco policy" AND "alcohol use" [all fields] AND "policy" [title/abstract]. The keyword strings used in Embase were: (juvenile)/br AND (('alcohol policy')/br) AND (('tobacco use')/br) AND ((policy):ab,ti); (juvenile)/br AND (('tobacco policy')/br) AND (('alcohol consumption')/br) AND ((policy):ab,ti). Limitations on year of publication were not included in the searches. The following inclusion criteria were used to screen articles: 1) conducted in the United States, 2) published in English, 3) peer reviewed (i.e., not commentaries, editorials, conference abstracts, or dissertations), 4) measured exposures of substance-specific national policies, 5) measured outcomes of cross-use behavior (e.g., adolescent smoking comparisons pre- and post-alcohol policy), and 6) included participants who were underage to purchase the product (younger than age 21 for alcohol policies, younger than age 18 or age 21 for tobacco policies, depending on the law at the time of the study). This search was not limited to RCTs because it is rarely possible to evaluate policies using RCTs; policy evaluations typically involve natural experiments or quasi-experimental comparisons of locations where policies were and were not implemented.[356] Ultimately, only six studies[241,321-324,357] met all inclusion criteria in the initial search (see Table 8A). This small number of eligible studies provides a limited evidence base for evaluating policies that address both tobacco and alcohol use outcomes among adolescents and young adults beyond media-based exposure. Therefore, this chapter highlights additional studies that did not meet all inclusion criteria but were deemed relevant to better understand policies that prevent tobacco and alcohol use. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

To further supplement this review of empirical research, individual tobacco and alcohol policies were identified from the Alcohol Policy Information System (APIS) maintained by the National Institute on Alcohol Abuse and Alcoholism (NIAAA),[358] the 2020 State Performance & Best Practices for the Prevention and Reduction of Underage Drinking Report by the Substance Abuse and Mental Health Services Administration (SAMHSA),[359] and the Tobacco Control Milestones maintained by the American Lung Association (ALA).[360] Because a database tool for

tobacco policies comparable to the APIS was not identified, ALA's webpage tracking feature was used to identify events relevant to tobacco control, such as new initiatives, laws, or judicial rulings, and specifically, policies expected to affect adolescent and young adult behavior (e.g., tobacco 21 legislation). Using Google Scholar, identified policies were searched by name to increase coverage of the literature base for this chapter.

Table 8A Studies Meeting Initial Inclusion Criteria Regarding Cross-Substance Effects of Tobacco and Alcohol Policies

Study	Data source	Time period	Behavioral measures	Policy measures	Cross-substance effects
Dee 1999[321]	Monitoring the Future; cross-sectional surveys of high school seniors in the United States	1977–1992	**Tobacco**: Cigarette smoking in past 30 days **Alcohol**: Drinking in past 30 days Heavy drinking (5+ drinks in a row), past 2 weeks	**Tobacco**: Cigarette tax (state and federal excise taxes) **Alcohol**: Beer tax (state and federal excise taxes); Minimum legal drinking age (18, 19, or 20)	A minimum legal drinking age of 18 (versus 21) was associated with increased cigarette smoking. A minimum legal drinking age of 19 (versus 21) or 20 (versus 21) generally did not predict cigarette smoking. Effects of beer taxes or cigarette taxes on tobacco or alcohol use varied depending on whether the models included state-fixed effects and the years evaluated (due to limited within-state variation on these taxes).
Kessel Schneider et al. 2016[324]	MetroWest Adolescent Health Survey; biennial survey of high school students in 25 communities west of Boston	2006–2012	**Tobacco**: Cigarette smoking in past 30 days; cigarette purchasing in a store, past 30 days **Alcohol**: Drinking in past 30 days	**Tobacco**: Minimum tobacco purchase age of 21	Changes in past-30-day alcohol use did not differ based on whether a community had a minimum tobacco purchase age in effect.
Weitzman et al. 2005[241]	Harvard School of Public Health College Alcohol Study; U.S. college students from 120 universities	2001	**Tobacco**: Cigarette smoking in past 30 days **Alcohol**: Binge drinking (4+/5+ drinks in a row for women/men), once in the past 2 weeks	**Alcohol**: School-level perceived alcohol availability; school-level strength of alcohol policy enforcement; school-level expectations of parental notification for illegally obtaining alcohol	Students at colleges with greater alcohol availability had greater odds of cigarette smoking. Students at colleges with more strongly enforced alcohol policies and those at colleges with parental notification policies had lower odds of cigarette smoking.
Welsh et al. 2019[357]	National Survey of Drug Use and Health; cross-sectional survey of U.S. adolescents ages 14–17	2004–2012	**Tobacco**: Nicotine dependence in the past year (DSM-IV criteria) **Alcohol**: Alcohol abuse or dependence in the past year (DSM-IV criteria)	**Tobacco and Alcohol**: State-level policies mandating tobacco, alcohol, or illicit drug use prevention programs in high school	States with required prevention programs (versus those that recommended or had no policy on such programs) had significantly lower prevalence of abuse/dependence for alcohol (p = .034) but did not differ on prevalence of nicotine dependence.

Table 8A (continued)

Study	Data source	Time period	Behavioral measures	Policy measures	Cross-substance effects
Yoruk & Yoruk 2011[322]	National Longitudinal Survey of Youth, 1997 Cohort; longitudinal, nationally representative survey of youth ages 12–16 as of December 31, 1996; restricted to those ages 19–22 who had consumed alcohol, smoked cigarettes, or used cannabis since the date of the last interview	2000–2006	**Tobacco**: (past 30 days) • Cigarette smoking • Number of smoking days • Average number of cigarettes per day • Average number of cigarettes per smoking day **Alcohol**: (past 30 days) • Alcohol use • Binge drinking • Number of drinking days • Number of binge drinking days • Average number of drinks per day • Average number of drinks per drinking day	**Alcohol**: Minimum legal drinking age of 21	Discontinuity analyses, splitting over the age of 21, did not indicate statistically significant changes in tobacco use when an individual reaches age 21.
Yoruk & Yoruk 2013[323]	National Longitudinal Survey of Youth, 1997 Cohort; longitudinal, nationally representative survey of youth ages 12–16 as of December 31, 1996; restricted to those ages 19–22, any alcohol use status	2000–2006	**Tobacco**: (past 30 days) • Cigarette smoking • Number of smoking days • Average number of cigarettes per day • Average number of cigarettes per smoking day **Alcohol**: (past 30 days) • Alcohol use • Binge drinking • Number of drinking days • Number of binge drinking days • Average number of drinks per day • Average number of drinks per drinking day	**Alcohol**: Minimum legal drinking age of 21	Discontinuity analyses, splitting over the age of 21, did not indicate statistically significant changes in tobacco use when an individual reaches age 21.

Note. DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.

Appendix 8B Summary of Meta-Analyses and Systematic Reviews Described in This Chapter

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Multiple types of interventions				
Foxcroft & Tsertsvadze 2012[180] Systematic review 85 studies School-based programs: *N* = 142,738 Family-based programs: *N* = 14,595 Multicomponent programs: *N* = 57,545	Through July 2010	**Language:** Any **Publication:** Any **Population:** Adolescents ages 18 or younger **Study design:** RCTs **Intervention:** Universal alcohol prevention programs in school, family, or multiple settings **Outcomes:** Self-reported or objective measures of alcohol consumption	**Outcomes:** Studies that assessed only psychological perceptions/attitudes or awareness (not alcohol use)	**Tobacco use:** Tobacco use outcomes were not reported. **Alcohol use:** Studies could not be pooled due to heterogeneity. *School-based interventions*: Out of 11 studies of alcohol-specific interventions, 5 did not find statistically significant effects on alcohol use, and 6 found significant beneficial effects. Out of 39 studies of general substance use interventions, 25 did not find significant effects on alcohol use. *Family-based interventions*: Out of 12 studies, 9 found statistically significant short- and long-term beneficial effects on alcohol use. *Multicomponent interventions*: Out of 20 studies, 12 found statistically significant beneficial effects on alcohol use.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
MacArthur et al.2018[185] Meta-analysis 70 studies Total *N* not available	Through November 14, 2016	**Language**: Any **Publication type:** Any including abstracts, conference proceedings, and grey literature **Population**: Children and adolescents up to age 18 and/or parents, guardians, or caregivers **Study design:** RCTs and cluster RCTs; combined intervention and follow-up period of 6 months or longer **Intervention:** Individual, family, and school-level interventions addressing at least 2 risk behaviors (alcohol use, tobacco use, cannabis or other drug use, risky sexual behaviors, antisocial behavior and offending, vehicle-related risk behaviors, self-harm, gambling, unhealthy diet, sedentary behavior, low levels of physical activity) **Outcomes:** Outcomes collected from those ages 8–25; combined intervention and follow-up period of 6 months or longer; primary outcomes in the risk behavior areas of interest	**Intervention**: Interventions in only 1 domain: healthy eating/physical activity or substance use (i.e., interventions in which the 2 risk behaviors addressed were only unhealthy diet, low levels of physical activity, and high levels of sedentary behavior, or only tobacco use, alcohol consumption, and/or drug use); community and population-level interventions. Interventions aimed at individuals with clinically diagnosed disorders or clinical interventions.	Compared with participants in comparator groups, participants in universal school-based interventions had significantly lower tobacco use and alcohol use. **Tobacco use**: *Universal school-based interventions*: OR: 0.77 (95% CI, 0.60–0.97) *Targeted individual-level interventions*: OR: 1.28 (95% CI, 0.75–2.19) *Universal individual-level interventions*: OR: 1.03 (95% CI, 0.32–3.27) *Targeted family-level interventions*: OR: 0.78 (95% CI, 0.40–1.53) **Alcohol use**: *Universal school-based interventions*: OR: 0.72 (95% CI, 0.56–0.92) *Targeted individual-level interventions*: OR: 1.02 (95% CI, 0.80–1.31) *Universal individual-level interventions*: OR: 0.80 (95% CI, 0.58–1.11) Targeted family-level interventions: OR: 0.83 (95% CI, 0.47–1.46)

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Spoth 2008[191] Literature review 25 reviews of 127 interventions Total *N* not available	Not specified	**Language**: Any **Publication type:** Systematic reviews; literature reviews; meta-analyses; summary reports of evidence-based, peer-reviewed journal articles; books and book chapters; internet sources **Population**: Children and youth up to age 20, including youth not in college. **Study design:** Any intervention; universal, selected, and indicated prevention interventions targeting drinking behavior among youth **Outcomes**: Alcohol use among youth ages 10 or older; risk factors for alcohol use among children younger than age 10; or policy, law, or environmental changes that measured actions due to alcohol use (e.g., alcohol-related traffic incidents among adolescents)	**Population**: College-attending populations; youth with alcohol use disorders **Intervention**: Interventions targeting drug use, smoking, sexuality, or general health promotion that did not include measures of alcohol use	**Tobacco use**: Tobacco use outcomes not reported **Alcohol use**: 12 interventions met criteria for "most promising" evidence of effectiveness against alcohol use, and 29 met criteria for "mixed or emerging" evidence.
Tinner et al. 2022[136] Systematic review and meta-analysis 66 studies Studies of tobacco use: studies of individual-level interventions: *N* = 1,024. studies of universal school-based interventions: *N* = 21,762. studies of universal, combined setting interventions: *N* = 2,202.	Through October 2019	**Language**: Any **Publication type:** Any **Population:** Children and youth ages 8–25, or interventions for parents, caregivers, peers, teachers, or others intending to address behaviors among children and young people up to age 18 at study baseline. **Study design:** RCTs, including cluster RCTs, with follow-up period of 6 months or more from intervention initiation **Intervention**: Interventions addressing at least 2 substance use behaviors (tobacco, smoking, alcohol, cannabis, other drugs)	**Population**: Studies of individuals with clinically diagnosed disorders; studies where the majority of participants were age 18 at baseline assessment **Intervention**: Interventions addressing behaviors other than substance use; community- or population-level interventions such as media campaigns or policy, regulatory, or legislative measures	**Tobacco use**: There were no significant short-term effects (up to 12 months) on tobacco use for universal school-based interventions (OR = 0.98, 95% CI = 0.83–1.15), individual-level interventions (OR = 0.82, 95% CI = 0.55–1.23), or combined setting interventions (OR = 0.85, 95% CI = 0.46–1.59). **Alcohol use outcomes**: Universal school-based interventions (OR = 0.94, 95% CI = 0.84–1.04) and targeted school-based interventions (OR = 0.90, 95% CI = 0.74–1.09) had no significant short-term effects (up to 12 months) on alcohol use. Universal family interventions (OR = 0.69, 95% CI = 0.54–0.89) and universal individual interventions (OR = 0.75, 95% CI = 0.59–0.95) had significant prevention effects.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Individual-level interventions				
Champion et al. 2019[134] Qualitative synthesis and meta-analysis 22 studies (11 included in meta-analysis) Studies that assessed smoking prevalence: *N* = 2,891 intervention, *N* = 2,884 control Studies that assessed alcohol use prevalence: *N* = 2,069 intervention. *N* = 2,016 control	January 1, 2000–March 14, 2019	**Language**: Any **Publication type:** Any **Populatio**n: Students ages 11–18 **Study design:** RCTs **Intervention**: Universal school-based prevention programs targeting 2 or more health behaviors (alcohol use, smoking, diet, physical activity, sedentary behavior, and sleep) and delivered via eHealth methods (internet, computer, tablets, mobile technology, telehealth)	11 studies were excluded from meta-analysis because data could not be pooled.	**Tobacco use**: 3 studies included in meta-analysis; no significant intervention effects on smoking prevalence (OR = 1.23, 95% CI = 0.95–1.59; *p* = .12) **Alcohol use**: 2 studies included in meta-analysis; no significant intervention effects on alcohol use prevalence (OR = 1.13, 95% CI = 0.95–1.36; *p* = .17)
Kazemi et al. 2017[132] Systematic review 12 studies Total *N* not available	2005–2015	**Language**: English **Publication type:** Peer-reviewed original research **Population**: Adolescents or adults who reported using substances **Study design:** Studies with an intervention component **Intervention**: Interventions that used mobile communication (mHealth) and addressed substance use	Computer web-based interventions	**Tobacco use**: None of the included studies primarily addressed tobacco use. **Alcohol use**: There were mixed results in terms of intervention efficacy.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Monarque et al. 2023[137] Rapid review 43 studies Total *N* not available	January 2015–December 2021	**Language**: English or French **Publication type:** Any except dissertations **Population**: Youth and young adults ages 12–29, or studies of samples with a mean age of 16–24 and standard deviation <4 **Study design:** Empirical studies using quantitative, qualitative, and mixed methodologies; review articles **Intervention:** Digital health interventions for substance use disorders in youth	**Intervention**: Studies of interventions addressing substance use in combination with nonmental health issues; studies in which participants were not exposed to interventions	The review identified a total of 6 prevention interventions (3 web-based, 1 game-based, 2 virtual reality-based). Only 1 prevention intervention (RealTeen) addressed both tobacco and alcohol use and reported behavioral outcomes. **Tobacco use**: The intervention group in RealTeen reported fewer cigarettes smoked than the control group. **Alcohol use:** The intervention group in RealTeen reported less binge drinking than the control group.
Mylocopos et al. 2024[133] Systematic review 39 studies Total *N* not available	2004–2022	**Language**: Any **Publication type:** Any **Population**: Children and youths younger than age 21 who did not currently use e-cigarettes (vape), never vaped, or did not report vaping in the past 30 days. Studies that included participants ages 21 or older were included if the mean sample age was under 21 or if subgroup analyses of children or youth were reported. Studies with more than 80% of participants not vaping at baseline or that reported a subgroup analysis of participants who did not vape at baseline. **Study design:** RCTs and nonrandomized studies of interventions with a comparison group **Intervention**: Vaping prevention interventions at the individual, school, or community level	**Population**: Studies of participants ages 21 or older; studies including only individuals who vaped in the past 30 days **Intervention**: Studies of vaping policy or regulations at the population level, cessation or reduction strategies, or nonvaping tobacco use interventions	**Tobacco use**: Of 39 included studies, 14 were studies of individual-level interventions, 19 were of school-based interventions, and 6 were of community-based interventions. Of 5 individual-level intervention studies that reported e-cigarette use prevalence, 4 studies were associated with decreased e-cigarette use and 1 had no effect. Of 7 individual-level intervention studies that reported initiation intentions, 3 studies were associated with decreased intentions and 4 had no effect. Of 10 individual-level intervention studies that reported e-cigarette risk knowledge and/or attitudes, 9 studies were associated with improved knowledge/attitudes. Study designs prevented causal inferences. **Alcohol use**: No outcomes reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Perski et al. 2022[138] Systematic review 14 studies Studies of tobacco interventions: *N* = 187 Studies of alcohol interventions: *N* = 120,520	January 2000–January 2020	**Language**: English **Publication type:** Any except for conceptual or methodological papers with no empirical data **Population:** Participants who used tobacco, cannabis, alcohol, cocaine, or heroin **Study design:** Any study type using an intervention, with or without a comparison group **Intervention**: JITAIs to address substance use that were delivered via websites, text messages, apps, or wearables and provided real-time, tailored support repeatedly over time	**Intervention**: Studies with a JIT reminder at time points prespecified by the user	The review identified 4 tobacco prevention studies and 7 alcohol prevention studies, but none that addressed both substances together. **Tobacco**: There were mixed results for smoking outcomes. **Alcohol**: There were mixed results for alcohol use outcomes.
Tzelepis et al. 2021[135] Systematic review and meta-analysis 11 studies Total *N* not available	Before November 18, 2019	**Language**: English **Publication type**: Peer-reviewed article **Population**: Adults ages 18 and older **Study design**: RCTs and cluster RCTs **Intervention**: Internet-based interventions that addressed 2 or more health behaviors (smoking, alcohol use, nutrition, or physical activity)	**Population**: Studies that included only people with chronic conditions or pregnant people; studies that included people under age 18 **Study Design**: No control arm (intervention as a comparison group) **Intervention**: Studies addressing only 1 health behavior; interventions in which the internet-based intervention was part of a multicomponent approach using other modes of support (e.g., face-to-face, telephone) **Outcomes**: Outcomes measures not related to a health behavior of interest; outcomes not measured 6 months or more post recruitment	**Tobacco and alcohol use**: A total of 3 studies addressed both tobacco and alcohol use. A study of undergraduates found no significant intervention effects on current smoking prevalence (11.2% intervention vs. 14.1% control) or mean units of alcohol in the past 7 days (10.4 intervention vs. 11.0 control). Another study of undergraduates found significant intervention effects on current smoking prevalence (8.7% intervention vs. 13.0% control) but not on mean units of alcohol in the past 7 days (13.0 intervention vs. 13.4 control). A community-based study found no effects on number of people who smoked (*n* = 3 intervention vs. *n* = 4 control) or mean units of alcohol per week (6.7 intervention vs. 7.2 control).

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
School-based interventions – General				
Rundall & Bruvold 1988[151] Meta-analysis 76 studies Total *N* not available	1970–1988	**Language**: Not specified **Publication type:** Not specified **Population**: Not specified **Study design**: Evaluations of interventions with a comparison or control group **Intervention**: School-based interventions to reduce, prevent, or quit smoking and/or alcohol consumption **Outcomes**: Behavior, attitudes, and/or knowledge outcomes reported	Studies not meeting inclusion criteria	**Tobacco use**: Behavior change pooled ES: <3 months: 0.15; >3 months: 0.34 Knowledge change pooled ES: <3 months: 0.59; >3 months: 0.56 Attitude change pooled ES: <3 months: 0.05; >3 months: 0.13 **Alcohol use**: Behavior change pooled ES: <3 months: 0.11; >3 months: 0.12 Knowledge change pooled ES: <3 months: 0.52; >3 months: 0.38 Attitude change pooled ES: <3 months: 0.18; >3 months: –0.23
Tremblay et al. 2020[186] Systematic review 90 studies of 16 programs Total *N* not available	Through March 31, 2020	**Language**: English **Publication type:** Any **Population:** School-aged children and adolescents (Grades K–12; ages 5–18) **Study design:** Evaluations of program effectiveness that included a control group **Intervention:** Substance use prevention programs aimed at psychoactive substances and designed for and delivered to the general population (rather than specific groups)	**Intervention**: Programs solely aimed at nicotine prevention; programs designed for or delivered to only specific target groups (e.g., low-income children, clinical populations); programs that targeted parents, teachers, or systems surrounding children; programs for which only 1 study had been conducted	**Tobacco use**: None of the evaluations of alcohol-focused programs (4 total) reported tobacco use outcomes. Of the remaining 12 programs focused on multiple substances, 5 had significant effects on tobacco use in at least 1 study: Adolescent Alcohol Prevention Trial (2 studies), Here's Looking at You (1 study), Life Skills Training (9 studies), Project Alert (3 studies), and Unplugged (1 study). **Alcohol use**: The Life Skills Training program had the most published studies (17 studies); 10 of these studies found significant reductions in alcohol and/or drug use.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
School-based interventions – Tobacco prevention				
Black et al. 1998[159] (based on Tobler and Stratton 1997[361]) Meta-analysis 120 studies Total *N* not available	1978–1990	**Language**: Any **Publication type:** Any **Population:** Adolescents in Grades 5–12 **Study design:** Experimental or quasi-experimental **Intervention**: Universal, school-based adolescent drug prevention programs (addressing use of alcohol, cigarettes, cannabis, and other drugs) conducted in North America	Studies not meeting inclusion criteria	**Tobacco use**: *Interactive programs*: Weighted ES = 0.16 (high-quality studies), ES = 0.18 (all studies) *Noninteractive programs*: Weighted ES = –.02 (high-quality studies), ES = 0.13 (all studies) **Alcohol use**: *Interactive programs*: Weighted ES = 0.19 (high-quality studies), ES = 0.21 (all studies) *Noninteractive programs*: Weighted ES = 0.03 (high-quality studies), ES = 0.07 (all studies)
Bruvold 1993[158] Meta-analysis 84 studies of 94 interventions Total *N* not available	1970s–1980s	**Language:** Any **Publication type:** Any (published or unpublished) **Population**: School-age individuals **Study design:** Experimental design with a no-intervention control or comparison **Intervention:** School-based interventions designed to prevent adolescent smoking	**Intervention**: College-level interventions **Study Design**: Reviews or theoretical papers; surveys or curriculum reports that did not evaluate interventions; control or comparison group received an intervention; no comparison group was included	**Tobacco use**: Behavioral outcome ES were largest for social reinforcement orientation interventions; ES for higher quality studies are presented below. *Rational orientation interventions*: Post-test ES = –0.01; 1st follow-up ES = 0.13 ($p < .05$); 2nd follow-up ES = –0.03; 3rd follow-up ES = –0.1 *Social reinforcement orientation interventions*: Post-test ES = 0.32; 1st follow-up ES = 0.31; 2nd follow-up ES = 0.39; 3rd follow-up ES = 0.27 (all *ps* < .05) *Social norms orientation interventions*: Post-test ES = 0.29;1st follow-up ES = 0.19 ($p < .05$); 2nd follow-up ES = 0.36 ($p < .05$) *Developmental orientation interventions*: Post-test ES = 0.36 ($p < .05$); 1st follow-up ES = 0.08; 2nd follow-up ES = –0.42 ($p < .05$) **Alcohol use**: Alcohol use outcomes not reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Thomas et al. 2013[162] Systematic review and meta-analysis 134 studies *N* = 428,293	1960–October 2012	**Language**: Any **Publication type:** Any **Population**: Children and adolescents ages 5–18 in school settings **Study design:** RCTs where students, classes, schools, or school districts were randomized to intervention group or control group, baseline tobacco use was measured, and participants were followed for at least 6 months **Intervention**: Any curricula used in a school setting to deter tobacco use **Outcomes**: Smoking status of individuals or cohorts who reported no tobacco use at baseline	**Study Design:** Nonrandomized allocation of individuals or groups to intervention and control groups	**Tobacco use**: All "Pure Prevention" cohorts (49 total), up to 1-year follow-up: OR: 0.94 (95% CI, 0.85–1.05, *p* = .26) All "Pure Prevention" cohorts (49 total), > longest follow-up: OR: 0.88 (95% CI, 0.82–0.96, *p* = .002) **Alcohol use**: Alcohol use outcomes not reported
Thomas et al. 2015[163] Systematic review and meta-analysis 50 studies *N* = 143,495	Through January 2014	**Language**: Any **Publication type:** Any **Population**: Students ages 5–18 who had never smoked at baseline **Study design**: RCTs (individual or cluster) **Intervention**: School-based smoking cessation programs **Outcomes**: Smoking status at 6-month follow-up or later	**Study Design**: No control group **Outcomes**: Baseline smoking status not assessed; only smoking attitudes and knowledge (not behavior) assessed	**Tobacco use**: All programs, ≤ 1-year follow-up: OR: 0.91 (95% CI, 0.92–1.01, *p* = .06) All programs, >1-year follow-up: OR: 0.88 (95% CI, 0.82–0.95, *p* = .0007) **Alcohol use:** Alcohol use outcomes not reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
School-based interventions – Alcohol prevention				
Hennessy et al. 2015[183] Meta-analysis 17 studies Total *N* not available	1980 to 2012	**Language**: Any **Publication type:** Any **Population**: Adolescents in middle, high, and secondary schools **Study design**: RCTs or quasi-experimental designs comparing interventions to a no-treatment or treatment-as-usual comparison group **Intervention**: BAI designed to reduce adolescent and young adult alcohol consumption, delivered individually or in groups, delivered in in less than 5 hours contact time, and lasting no more than 4 weeks **Outcomes**: Sufficient information to allow estimation of post-intervention effect sizes between groups on at least 1 alcohol consumption-related outcome	Studies not meeting inclusion criteria	**Tobacco use**: Tobacco use outcomes not reported **Alcohol use**: *All interventions*: Compared to controls, participants in BAIs had significantly reduced alcohol consumption ($\bar{g}$ = 0.34, 95% CI = 0.11–0.56). *Individually-delivered interventions*: Significant intervention effects on reducing alcohol use ($\bar{g}$ = 0.58, 95% CI = 0.23–0.92) *Group-delivered interventions*: No significant intervention effects on reducing alcohol use ($\bar{g}$ = –0.02, 95% CI = -0.17–0.14)
Lee et al. 2016[182] Systematic review 70 studies of 40 programs Total *N* not available	1998–2013	**Language**: English **Publication type:** Published articles **Population**: School-aged children **Study design**: Australian National Health and Medical Research Council criteria for level of evidence I (systematic reviews) to evidence IV (case series or pre-post designs) **Intervention**: School-based programs that target alcohol use **Outcomes**: At least 1 alcohol behavior or knowledge change outcome	**Study Design**: Qualitative studies or case studies focusing on acceptability of programs or process evaluations **Intervention**: School-based programs not focusing on alcohol use; community or family-based programs	**Tobacco use**: Tobacco use outcomes not reported **Alcohol use**: Interventions with the most evidence for effectiveness across multiple trials were CLIMATE Schools in Australia and Project ALERT and All Stars in the United States.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Lemstra et al. 2010[181] Systematic review 6 studies *N* = 11,926	January 1, 1980–December 2, 2007	**Language**: English **Publication**: Any (published or unpublished) **Population**: Children ages 10–15; studies including children outside of this age range could be included as long children ages 10–15 were included in the study. **Study design**: RCTs, prospective cohort studies, longitudinal studies, systematic reviews, meta-analyses; studies with a sample size of at least 300; studies rating with a score of at least 10 out of 14 for methodological evaluation quality **Intervention**: School-based interventions to prevent cannabis and/or alcohol use in adolescents and implemented in Canada, United States, Western Europe, Australia, or New Zealand **Outcomes**: Follow-up period of at least 1 year	**Study Design**: Opinion papers, letters to the editor, case reports, case studies, natural experiments, cross-sectional studies **Intervention**: Comparisons of clinical settings versus schools **Outcomes**: Baseline data not presented or not available upon request	**Tobacco use:** Tobacco use outcomes not reported **Alcohol use**: *Comprehensive programs*: Compared to controls, adolescents who received interventions had a pooled MUR of 0.88 (95% CI, 0.87–0.89), equivalent to a reduction in 12 days of alcohol use per month. *Professional-led knowledge-only programs*: Compared to controls, adolescents who received interventions had a pooled MUR of 0.98 (95% CI, 0.92–1.04), equivalent to a reduction in 2 days of alcohol use per month.
College campus prevention programs				
Bennett et al. 2017[227] Literature review 11 studies Total *N* not available	1990 to June 2016	**Language**: Any **Publication type**: Peer-reviewed **Population**: Young adults ages 18–25 at U.S. college campuses (2- and 4-year colleges and universities) **Study design**: Quantitative methods that directly (e.g., self-report) or indirectly (e.g., count of cigarette butts on premises) assessed young adults' cigarette smoking behaviors **Intervention**: College-level smoking policies	**Study Design**: Case studies, focus groups, interviews	**Tobacco use**: The cross-sectional studies (6 total) found mixed results on college student smoking behavior. Of the 2 longitudinal studies with self-reported smoking behavior identified, 1 found a reduction in proportions of high- and low-frequency smokers, pro-smoking attitudes, and exposure to secondhand smoke before and after the policy was implemented but did not find an effect on smoking prevalence. The other found overall decreased smoking prevalence on campus with smoking policy compared with control campus. **Alcohol use**: Alcohol use outcomes not reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Carey et al. 2007[220] Meta-analysis 62 studies of 98 interventions *N* = 13,750	Through March 9, 2007	**Language**: Any **Publication type**: Published or in press **Population:** College or university students **Study design**: RCTs or quasi-experimental design with a control group **Intervention**: Educational, behavioral, or psychological alcohol interventions **Outcomes:** Drinking behavior outcomes with sufficient information to calculate between-group effect size estimates	**Population**: Noncollege students, or mix of college and noncollege students, and results not separated by college status **Study Design:** No control or comparison condition **Intervention**: Interventions that did not specifically focus on alcohol **Outcomes:** Insufficient statistical information to calculate effect sizes; did not measure any alcohol behavior	**Tobacco use:** Tobacco use outcomes not reported **Alcohol use:** *Quantity of alcohol consumed*: Significant intervention effects at immediate follow-up (*d* = 0.19, 95% CI = 0.07–0.32), short-term follow-up (4–13 weeks; *d* = 0.13, 95% CI = 0.06–0.19), and intermediate follow-up (14–26 weeks; *d* = 0.11, 95% CI = 0.02–0.20) *Frequency of heavy drinking*: Significant intervention effects at immediate follow-up (*d* = 0.17, 95% CI = 0.03–0.31), short-term follow-up (*d* = 0.18, 95% CI = 0.10–0.26), and intermediate follow-up (*d* = 0.11, 95% CI = 0.01–0.22). *Frequency of drinking days*: Significant intervention effects at long-term follow-up (27–195 weeks; *d* = 0.16, 95% CI = 0.03–0.30).
Carey et al. 2016[223] Meta-analysis 31 studies of 68 interventions *N* = 8,621	Through 2012	**Language**: Any **Publication type**: Any **Population**: College students mandated to an alcohol intervention **Study design**: Single-group or independent groups pretest-posttest design **Intervention**: Individual or group-level interventions to reduce alcohol use **Outcomes**: Sufficient statistical information included to calculate effect sizes on alcohol use	**Population**: College students not mandated to an intervention **Study Design:** No intervention/waitlist control conditions **Intervention**: Interventions that did not focus on alcohol use; interventions that included a mass media or structural-level intervention component	**Tobacco use**: Tobacco use outcomes not reported. **Alcohol use**: *Between-group contrasts*: Intervention students reported significantly less drinking per week/month relative to controls (*d* = 0.13, 95% CI = 0.02–0.25) *Within-group contrasts*: Significant intervention effects for all quantity and frequency outcomes at short-term (≤13 weeks; *ds* = 0.14–0.27); significant intervention effects on frequency of heavy drinking days (*d* = 0.14, 95% CI = 0.04–0.23) at intermediate-term follow-up (14–51 weeks); no significant intervention effects on quantity or frequency outcomes at long-term follow-up (≥52 weeks).

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Carey et al. 2012[221] Meta-analysis 48 studies Face-to-face interventions: *N* = 5,237 Computer-delivered interventions: *N* = 32,243	Through December 2010	**Language**: Any **Publication type**: Any **Population**: College students **Study design**: RCTs or quasi-experimental designs with an assessment-only, waitlist, or no treatment control condition **Intervention**: Individual-level alcohol interventions **Outcomes:** Sufficient information to calculate effect sizes	**Population**: Noncollege students **Study Design**: Active control condition (e.g., education only) **Intervention**: Interventions that did not specifically focus on alcohol use; interventions that included a mass media or structural-level intervention component **Outcomes**: Insufficient statistical information to calculate effect sizes	**Tobacco use**: Tobacco use outcomes not reported **Alcohol use**: Participants in face-to-face interventions had reduced alcohol consumption, reduced peak blood alcohol content, and fewer alcohol-related problems than those in computer-delivered interventions (*d*s = 0.12–0.20).
Lupton & Towsend 2015[224] Systematic review and meta-analysis 19 studies Total *N* not available	Through June 2013	**Language**: English **Publication type**: Any **Population**: College or university population **Study design**: Cross-sectional, cluster randomized interventions, or before and after interventions **Intervention:** Complete indoor and outdoor smokefree policies on campus **Outcomes:** Reporting of student and faculty support for smokefree policies, smokefree policy effectiveness at reducing secondhand smoke exposure, cigarette consumption, or smoking prevalence	**Study Design:** Survey methods or effectiveness measures that did not meet quality requirements (e.g., sampling strategy, response rate, or survey questions not reported) **Other:** Articles titles that did not address tobacco use at a university setting	**Tobacco use**: 2 studies reported cigarette smoking prevalence. 1 study reported a significant decrease in smoking, from 16.5% at baseline to 12.8% at follow-up ($p < .001$), while the control campus had a nonsignificant increase in smoking prevalence from 9.5% to 10.1%. Another study found that there was a significant decrease in smoking prevalence from 9.5% at 3 months before the smokefree policy to 7.6% at 1-year follow-up, 6.6% at 2-year follow-up, and 7.0% at 3-year follow-up ($p = .04$). **Alcohol use**: Alcohol outcomes not reported
Plotnikoff et al. 2019[213] Systematic review 88 studies Total *N* not available	January 1970 to October 2016	**Language:** English **Publication type**: Peer-reviewed publications **Population**: Students attending higher education institutions **Study design**: RCTs **Intervention**: Interventions aiming to improve 1 or more health behaviors (alcohol use, drug use, smoking) and implemented in a higher education setting (university, college)	**Publication Type**: Grey literature (e.g., dissertations, theses)	**Tobacco use**: Of 9 studies that reported comparable tobacco use measures, 1 study reported no effect, 4 reported reductions in tobacco use, and 4 reported increased tobacco use. **Alcohol use**: Of 46 studies that reported comparable alcohol consumption measures, 42 reported alcohol consumption decreases and 4 reported alcohol consumption increases.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Toomey et al. 2007[243] Literature review 36 studies Total *N* not available	1999–2006	**Language**: Any **Publication type:** Peer-reviewed, published research **Population**: College students **Study design:** Any **Intervention**: Environmental policies and strategies to reduce drinking among college students	Studies not meeting inclusion criteria	**Tobacco use:** Tobacco use outcomes not reported **Alcohol use**: The authors concluded that environmental policies and strategies can reduce alcohol use and alcohol-related problems among college students, but not all approaches are effective. Many strategies have few or no evaluations.
Family-based interventions – Tobacco				
Thomas et al. 2015[268] Systematic review 27 studies Total *N* not available	Through April 3, 2014	**Language**: Any **Publication type**: Any **Population**: Children (ages 5–12) or adolescents (ages 13–18) and families **Study design**: RCTs **Intervention:** Interventions with children or adolescents and families to deter tobacco use initiation **Outcomes**: Smoking status of children who reported no use of tobacco at baseline, measured at least 6 months from the start of the intervention	**Intervention:** Interventions primarily focused on cessation **Outcomes**: Baseline smoking status not assessed; smoking behaviors not assessed; family-based intervention effects could not be separated from other co-interventions	**Tobacco use**: Compared to no intervention, standalone family interventions can reduce uptake or experimentation with smoking among children and adolescents by 16% to 32% (based on data from 9 trials). Compared to school interventions, combined family and school interventions can reduce uptake of smoking by 4% to 25%. However, effect estimates did not include data from all studies. **Alcohol use**: No alcohol use outcomes reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Family-based interventions – Alcohol				
Gilligan et al. 2019[271] Systematic review 46 studies *N* = 39,822	Through June 30, 2018	**Language**: Any **Publication type**: Any **Population:** Parents or guardians/carers of school-aged children (up to age 18) who were part of the general population **Study design:** RCTs and cluster RCTs of psychosocial or education interventions for parents compared with no intervention or an alternate intervention **Intervention**: Family-based prevention programs to prevent alcohol and problem drinking for children with no known risk factors, at elevated risk for alcohol use, or already consuming alcohol **Outcomes**: Alcohol use (yes/no, quantity, frequency), binge drinking, or incidence of drunkenness among children	**Population:** Adolescents transitioning to college	**Tobacco use**: No tobacco use outcomes reported. **Alcohol use:** Overall, family-based interventions had no significant effects on the prevalence (SMD = 0.00, 95% CI = -0.08–0.08), frequency (SMD = -0.31, 95% CI = -0.83–0.21), or volume of alcohol use (SMD = -0.14, 95% CI = -0.27–0.00) among children younger than age 18.
Smit et al. 2008[270] Meta-analysis 18 studies of 9 trials Total *N* not available	January 1995–September 2006	**Language**: Dutch, English **Publication type**: Published articles and unpublished dissertations **Population**: Parents with children under age 16 **Study design:** RCTs **Intervention**: Family interventions (at least half of the intervention had to target parents directly) conducted in the general population **Outcomes**: Alcohol use behavior outcomes reported for adolescents or school age children	**Intervention**: Interventions conducted in at-risk groups (juvenile offenders, children of adults with alcohol use disorder) **Outcomes**: Interventions part of multicomponent interventions with no separate analyses for family interventions; alcohol use behavior not measured	**Tobacco use**: No tobacco use outcomes reported **Alcohol use**: *Alcohol initiation*: Family interventions reduced alcohol initiation at 18–30 months (OR = 0.71, 95% CI = 0.54–0.94) *Last month alcohol use*: Family interventions had no significant effect on past-month alcohol use (OR = 0.70, 95% CI = 0.46–1.05) *Frequency of alcohol use*: Family interventions significantly reduced frequency of alcohol use in the last month or last year at 36–48 months follow-up (*d* = -0.25, 95% CI = -0.37– -0.12).

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Family-based interventions – Tobacco and alcohol				
Champion et al. 2022[273] Meta-analysis 46 studies of 36 RCTs *N* = 28,322 youth; *N* = 7,385 parents	January 2010–May 2021	**Language**: Any **Publication type:** Any including grey literature (dissertations, clinical trial registries, conference proceedings) **Population**: Parents and adolescents (mean age between 11 and 18 years) **Study design**: RCTs using comparators of no intervention, waitlist control, or an alternate intervention **Intervention**: Parent-based preventive interventions to address 2 or more risk behaviors (alcohol use, tobacco use, diet, physical activity, sedentary behaviors, sleep) **Outcomes**: Outcomes reported for adolescents	**Intervention**: Weight loss interventions targeting overweight or obese youth; interventions with only indirect parental involvement (e.g., receiving a newsletter)	**Tobacco use:** *Ever use:* OR: 1.36 (95% CI, 1.06–1.74) *Past-month tobacco use, short-term (<6 months) follow-up*: OR: 1.21 (95% CI, 0.57–2.54) *Past-month tobacco use, medium-term (6–12 months) follow-up*: OR: 1.27 (95% CI, 0.78–2.08) *Past-month tobacco use, long-term (>12 months) follow-up*: OR: 1.46 (95% CI, 1.12–1.90) **Alcohol use:** No significant intervention effects *Ever use*: OR: 1.01 (95% CI, 0.82–1.24) *Past-month alcohol use, short-term (<6 months) follow-up*: OR: 1.06 (95% CI, 0.36–3.07) *Past-month alcohol use, medium-term (6–12 months) follow-up*: OR: 1.06 (95% CI, 0.66–1.71) *Past-month alcohol use, long-term (>12 months) follow-up:* OR: 0.94 (95% CI, 0.69–1.28)

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Van Ryzin et al. 2016[272] Meta-analysis 116 studies Total *N* not available	Through September 2012	**Language**: Any **Publication type**: Peer-reviewed studies in journals, books, dissertations, theses, and technical reports **Population**: Adolescents and youth ages 11–21 **Study design**: Designs that support inferences about the relative effectiveness of a family-based prevention program compared to a control group or baseline **Intervention:** Family-based universal or selective programs aimed at preventing adolescent substance use that included face-to-face, telephone, or email communication with families **Outcomes**: Adolescent substance use (tobacco, alcohol, cannabis, or other drugs); sufficient information to calculate an effect size	**Intervention**: Interventions targeting those already using substances	Programs that included positive family relations or future orientation had significant effects (overall mean ES *d* = 0.31) on adolescent tobacco and alcohol use. There were no statistically significant differences in effect sizes by type of substance (tobacco, alcohol, and other drugs). **Tobacco use**: Overall ES: *d* = 0.25 **Alcohol use**: Overall ES: *d* = 0.31

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Population-level interventions				
Akter et al. 2023[311] Systematic review and meta-analysis 144 studies Total *N* not available	Through March 2022	**Language**: Any **Publication type**: Peer-reviewed journal articles and grey literature **Population:** No restriction **Study design**: Observational studies (any design including cross-sectional, case-control, cohort studies), quasi-experimental studies (including interrupted time series, before and after studies, controlled before and after studies) **Intervention**: Population-level policies aimed at reducing tobacco use, including policies such as smokefree policies, taxation, and minimum cigarette sales age **Outcomes**: Respiratory symptom disease, cardiovascular disease, cancer, mortality, health care utilization	**Publication Type**: Letters, case studies, systematic or scoping reviews, commentaries, editorials without original research findings **Study Design**: RCTs, simulation or modeling studies	**Tobacco use:** Smokefree legislation (126 studies) was associated with decreased risk of cardiovascular disease events (incidence, prevalence, or mortality; OR = 0.90, 95% CI = 0.86–0.94), respiratory symptom disease events (incidence, prevalence, or mortality; OR = 0.83, 95% CI = 0.72–0.96), and hospitalization due to cardiovascular disease or respiratory symptom disease (OR = 0.91, 95% CI =0.87–0.95). Effects on tobacco use prevalence were not reported. **Alcohol use**: No alcohol use outcomes reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Vahedi et al. 2018[289] Meta-analysis 20 studies Total *N* not available	Through May 2018	**Language**: English **Publication type**: Any **Population**: Participants with a mean age between 11 and 19 **Study design**: Evaluation using pre- and post-measures or a control group **Intervention**: Media literacy interventions (including media skepticism or media deconstruction skills interventions that identified the meaning, source, and intended audience of media images depicting smoking/tobacco use, alcohol use, other substance use, or risky sexual behavior) **Outcomes**: Attitudes, behavioral intentions, and/or behavior toward smoking/using tobacco, drinking alcohol, or using other substances, or engaging in unsafe sexual behavior before and after the intervention	**Study Design**: Cross-sectional studies or surveys	**Tobacco use**: No tobacco use behavioral outcomes reported **Alcohol use**: No alcohol use behavioral outcomes reported
Population-level interventions – Mass media campaigns				
Allen et al. 2015[305] Systematic review 34 studies Total *N* not available	Through March 2014	**Language:** Any **Publication type**: Articles indexed in PubMed and PsycInfo **Population**: Adolescents ages 18 and younger **Study design**: Experimental studies that examined the efficacy of anti-tobacco messages or studies that examine the effectiveness of anti-tobacco mass media campaigns **Intervention:** Mass media campaigns to address youth smoking/tobacco use in terms of cognitions or behavior	Studies that were not quantitative, or did not include results for youth ages 18 or younger	**Tobacco use:** The authors concluded that mass media campaigns can be effective in reducing youth tobacco use behavior, although the size of campaign effects might differ by race and/or ethnicity and by sex. **Alcohol use:** Alcohol use outcomes not reported

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Palmedo et al. 2017[310] Systematic review 14 studies Total *N* not available	January 1998–June 2016	**Language:** English **Publication Type**: Any **Population:** United States and other English-speaking countries **Study design**: Empirical studies of the design, planning, implementation, or evaluation of countermarketing initiatives **Intervention:** Countermarketing initiatives on tobacco, alcohol, or unhealthy food	Studies not meeting inclusion criteria	**Tobacco use**: Aggregated effects on tobacco use behavioral outcomes were not reported. **Alcohol use:** Aggregated effects on alcohol use behavioral outcomes were not reported.
Young et al. 2018[308] Systematic review 24 studies Total *N* not available	Through July 2016	**Language**: English **Publication Type:** Any **Population**: Any **Study design:** Any **Intervention:** Mass media interventions (using channels such as television, radio, cinema, online broadcasting, newspapers, magazines, leaflets/booklets, direct mail, outdoor advertising, email, digital media) targeting alcohol consumption or related behavioral, social cognitive, or clinical outcomes; multicomponent interventions that included assessment of the effects of the mass media component were permitted **Outcomes:** Alcohol consumption; alcohol-related social-cognitive variables (e.g., knowledge, intentions, social norms); exposure outcomes (e.g., campaign awareness); alcohol-related harm; health service usage	**Intervention:** Drunk driving interventions; college campus campaigns	**Tobacco use:** Tobacco use outcomes not reported **Alcohol use:** 13 studies assessed the effects of campaigns on alcohol consumption; however, reducing alcohol consumption was a stated aim of only 6 of the campaigns (the other 7 aimed to address outcomes such as knowledge, attitudes, or beliefs). Of the 6 studies that sought to address alcohol consumption, 5 reported no statistically significant differences in alcohol consumption between the exposed and nonexposed groups. One study of weak quality found significant effects of a TV and mailed letter campaign.

Appendix 8B (continued)

Study Publication type Number of studies included Total *N*	Dates of studies included	Inclusion criteria	Exclusion criteria	Main findings
Culturally adapted interventions				
Bo et al. 2023[288] Systematic review and meta-analysis 30 studies Total *N* not available	Through September 2022	**Language**: Any **Publication Type:** Any **Population:** U.S. Black, Hispanic, American Indian, Asian American, Native Hawaiian or Pacific Islander, and multiracial adolescents ages 11–18 **Study design**: RCTs (individual or cluster) **Intervention**: Culturally sensitive universal, selective, or indicated individual-, family-, school-, or community-based substance use prevention programs or positive youth development programs conducted in the United States; culturally sensitive components included culturally appropriate language or contents that address cultural values, racial and/or ethnic identity, pride, acculturation, discrimination, or racial and/or ethnic socialization **Outcomes:** Behavioral substance use outcomes and substance use-related consequences or problems; all kinds of substances were eligible for inclusion.	**Population:** Adolescents with a substance or alcohol abuse and dependence diagnosis; adolescents who had graduated from high school; studies with mainly White participants **Intervention:** Studies that were only surface structure adapted (e.g., only used culturally relevant language) or that had only passive intervention messages	**Tobacco use**: Culturally sensitive interventions had a significant effect on tobacco use outcomes, *g* = -0.21 (95% CI, -0.32 to -0.10). **Alcohol use**: Culturally sensitive interventions had a significant effect on alcohol use outcomes, *g* = -0.14 (95% CI, -0.20 to -0.09).

Note. BAI = brief alcohol interventions; CI = confidence interval; ES = effect size; JIT = just-in-time; JITAI = just-in-time adaptive intervention; MUR = mean usage reduction; OR = odds ratio; RCT = randomized controlled trial; SMD = standardized mean difference.

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Chapter 9
Economic, Environmental, and Policy Factors Related to Tobacco and Alcohol Use

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Chapter 9
Economic, Environmental, and Policy Factors Related to Tobacco and Alcohol Use

Introduction

Tobacco and alcohol initiation, use, reduction, and cessation are strongly influenced by the policies, systems, and environments that shape accessibility, affordability, and marketing of these substances.[1-11] Restrictions on locations or times of sales,[6-8,10,11] increased taxation,[1,9] and regulation of product marketing[1-3,12,13] are examples of policies that can reduce tobacco and alcohol use. Policies that influence the purchase or use of tobacco and alcohol have the benefit of directly affecting large portions of the population. However, the aims and evaluations of these approaches often focus only on the targeted substance, such as examining how indoor smokefree policies affect tobacco use and secondhand smoke exposure, or how increases in alcohol purchase age affect alcohol consumption. Lack of evaluation of cross-substance policy effects is a significant limitation in efforts to address co-use of tobacco and alcohol and to fully understand effects of policy choices. In addition, there are some shared policy approaches to reducing tobacco and alcohol use as well as their health and societal consequences (e.g., taxation, limiting retailer density, mass media campaigns for prevention and control). This overlap in context and policy approaches for both substances suggests that it is important to consider whether and how tobacco and alcohol economic, environmental, and policy factors intersect, such as how alcohol policies could affect tobacco consumption.

Focusing on tobacco and alcohol at the macro level, this chapter reviews and synthesizes research about the influence of economic, environmental, and policy factors related to alcohol on tobacco use, including initiation, co-use, reduction, and cessation. The chapter begins with a description of the tobacco and alcohol industries in the United States and economic factors including the retail environment for tobacco and alcohol and trends in the tobacco and alcohol markets. This is followed by a section addressing whether and how the retail environment for alcohol (i.e., quantity, type, and location of alcohol retailers) and the marketing environment of alcohol may influence tobacco use. This section also summarizes research on where the retail environments for tobacco and alcohol intersect, how marketing strategies for tobacco and alcohol intersect, the extent of exposure to marketing for both products, and discusses how these exposures may promote tobacco and alcohol co-use. The policy factors section addresses cross-substance effects of policies, focusing on how alcohol regulations influence tobacco use outcomes, including effects of (1) increasing alcohol tax and price on tobacco use and (2) restricting alcohol availability on tobacco use. Because some settings in which people use alcohol are a focus of tobacco marketing and are also important targets for tobacco control policy, the policy section also considers the impact of smokefree policies on alcohol use, including in substance use treatment settings.

In addition, the chapter notes areas where economic, environmental, and policy factors affect differences in exposures and behavioral outcomes among demographic groups, such as place-based differences in the concentration of tobacco and alcohol outlets and associations between availability and affordability of these products and their use. The primary focus of the chapter is on literature from the United States; however, international evidence is also incorporated, particularly for topics where no U.S. studies were found. The search strategy used to identify literature for this chapter is described in Appendix 9A.

Systems of Influence on Tobacco and Alcohol Use

Understanding the economic, environmental, and policy factors related to tobacco and alcohol depends, in part, on understanding that tobacco and alcohol use occurs in a system of influences and at various levels, from the intrapersonal to the macro-environmental. A systems focus recognizes that efforts to change individuals' behaviors benefit from addressing the social, relational, and institutional forces that help shape individual behavior,[14] and that targeting such forces may help overcome the difficulty of delivering individual-level interventions to the large number of people in need of them.[15] The commercial entities that are part of a system of influences on tobacco and alcohol use include the tobacco and alcohol manufacturers; distributors; retail, bar, and restaurant outlets; and the associated groups that promote sales and consumption of the products.[16-20] Multinational tobacco and alcohol companies seek to shape systems to support their business interests.[17] For example, these activities include organizing corporate social responsibility activities, accessing and influencing policymaking, undertaking legal challenges to policies, and funding industry-friendly research.[21] These corporate entities often use a message frame of cooperation to normalize their role in policymaking and to position themselves as partners in addressing the adverse effects of their products. This positioning can lead to further normalization of their businesses and acceptance of voluntary codes of conduct (e.g., industry marketing codes)[22] which have consistently been found to be ineffective at reducing youth exposure to cigarette advertising.[22,23,24] For alcohol sales, government entities may overlap with commercial ones (e.g., in jurisdictions with state-run liquor outlets).[25]

Historically, the tobacco and alcohol industries have been closely aligned and shared strategies to further their commercial activity that have shaped tobacco and alcohol use in the United States and globally.[26-28] There has been cross-ownership of tobacco and alcohol companies, as well as tobacco companies that have also manufactured alcohol products, such as Philip Morris USA's former ownership of Miller Brewing[29] and the former ownership of Ste. Michelle Wine Estates business by Altria, the parent company of Philip Morris USA,[30] as well as Altria's minority ownership stake in Anheuser-Busch.[30] Given the diversification of transnational corporations and the overlap between tobacco and alcohol manufacturers, it is important to continue to understand cross-industry influences and their impact on tobacco and alcohol use and their effect on public health.[31,32] It is also important to understand that the tobacco and alcohol industries have engaged in similar strategies that may appear in line with public health but primarily serve to further their business interests, such as developing voluntary self-regulation codes, deploying corporate social responsibility programs, and reframing policy debates from industry responsibility toward individual responsibility.[23,31,33]

Economic Factors

This section provides a brief overview of the tobacco and alcohol industries in the United States and then discusses the retail environment for tobacco and alcohol and trends in the tobacco and alcohol markets. Economic policies such as pricing and taxation are discussed in the "Policy Factors" section of this chapter.

Industry

Manufacturers

The cigarette and beer industries in the United States have been long dominated by several large manufacturers. As of 2022, two cigarette manufacturers hold around 76% of the total U.S. market share—Philip Morris USA, a subsidiary of Altria, holds a market share of 45.5%, followed by R.J. Reynolds, which has a market share of 30.3%.[34] Large cigarette manufacturers also have significant control of market share at a global level. In 2017, R.J. Reynolds was acquired by British American Tobacco (BAT),[35] bolstering the position of BAT as one of the five leading global tobacco manufacturers, along with Philip Morris International; Japan Tobacco, Inc.; Imperial Brands; and China National Tobacco Corporation.[36]

Like the tobacco industry, the alcohol industry is also controlled by leading multinational producers, especially in beer and distilled spirits markets. In 2022, approximately 65% of the U.S. beer market was dominated by 2 companies, Anheuser-Busch InBev SA/NV and MillerCoors, that held market shares of 41.1% and 23.4%, respectively.[34] In 2017, Anheuser-Busch InBev SA/NV manufactured about 27% of the commercial beer in the world,[37] and Diageo—a company based in the United Kingdom—had 20% of the global distilled spirits market.[37] The least concentrated segment of the alcohol industry is wine.[37] As of 2016, the leading 10 wine manufacturers together produced about 13% of wine in the global market.[37] The level of concentration of the U.S. alcohol markets reflects the global dominance of large alcohol companies.

While both the beer[38,39] and cigarette market over the past decade showed a high degree of concentration, market shares within the e-cigarette and other electronic nicotine delivery systems (ENDS) industry shows greater fluctuation.[40] Starting in 2012, major cigarette companies entered the U.S. e-cigarette market. In a merging of the ENDS and cigarette industries, cigarette companies have acquired or developed e-cigarette products—Lorillard acquired Blu, Japan Tobacco acquired Logic, R.J. Reynolds developed Vuse, and Philip Morris introduced MarkTen.[40] By 2022, the tobacco manufacturer Reynolds American Inc. held 35.9% of the U.S. ENDS market, and Juul Labs, a formerly independent company in which Altria acquired a 35% stake in 2018, held a U.S. market share of 32.3%.[34]

Evolving Tobacco and Alcohol Product Characteristics

When considering economic policies and the retail environment, it is important to consider the variety of products in the tobacco and alcohol categories and changes in the marketplace for both products. This includes new tobacco and alcohol product availability and promotion, particularly as both markets witness the emergence of products with new flavors and increasing potency, such as e-cigarettes and ready-to-drink (RTD) beverages.[41,42]

Since the 1920s, the U.S. tobacco market has been dominated by cigarettes.[43] However, with the introduction and emergence of a wide array of nicotine and tobacco products such as e-cigarettes, heated tobacco products (HTPs), and nicotine pouches, and with declines in cigarette smoking, the landscape of the nicotine and tobacco industry has grown increasingly complex.[44,45] A total of 7.0% of U.S. adults reported currently using e-cigarettes in 2024,[46] and 7.8% of U.S. high school students reported currently using e-cigarettes in 2024,[47] making e-cigarettes the second most used tobacco product among adults and the most used tobacco product among youth.[48,49] Meanwhile, tobacco companies continue to increase investment in noncigarette tobacco products, including via purchasing and/or launching their own e-cigarettes, smokeless tobacco, nicotine pouch, and HTP brands.[44] As a result, when evaluating how alcohol policies affect tobacco use, it is important to examine not only cigarette smoking but also the use of e-cigarettes and other tobacco products.

The alcohol market has historically been highly diverse in terms of product types, flavor options, and alcohol concentration and remains so.[50] This diversity is reflected in a broad spectrum of alcoholic beverage options, including beer, cider, wine, liquor (liqueurs and distilled spirits), RTD beverages, and other subtypes of alcohol-containing beverages.[37,42] In the last several decades there has been a rapid increase in sales of RTD beverages. The RTD category contains products that are distilled spirit cocktails and are often sweet, flavored, and similar in type and appearance to common nonalcoholic beverages like soda, seltzer, or juice.[42] According to Euromonitor data, the U.S. retail sales volume of RTD beverages increased from 588 million liters in 2008 to around 2.8 billion liters in 2022, showing a quick growth in sales.[34]

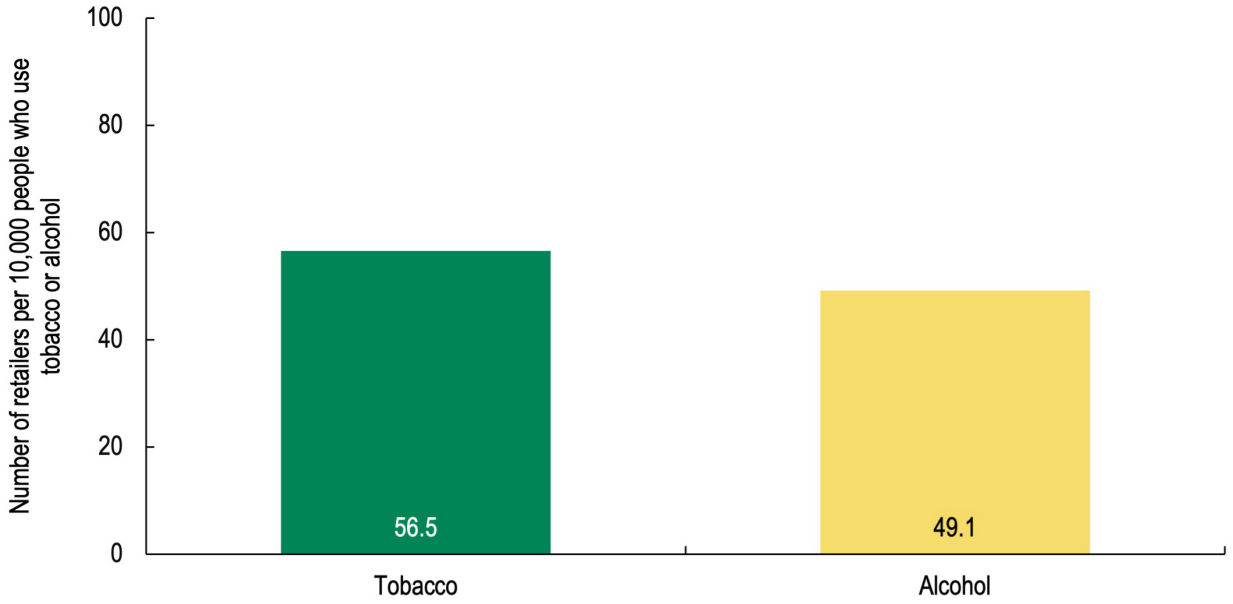
Retailers

Retail environments for tobacco and alcohol encompass on-premise and off-premise use facilities. “Off-premise use” refers to retail outlets where tobacco or alcohol is sold but the outlet is not designed for consumption of the product on site (e.g., a liquor store, a convenience store). In contrast, “on-premise use” refers to retailers where tobacco or alcohol is sold and the outlet is designed for consumption of the product on site (e.g., a hookah lounge, a bar, a casino, a restaurant).[51,52] In some states, some alcohol retailers are not permitted to sell tobacco (e.g., state-run liquor stores), and some tobacco retailers are not permitted to sell alcohol (e.g., grocery or convenience stores not licensed to sell alcohol).[53,54] However, a 2014 estimate suggested that 13% of tobacco retailers are off-premise liquor stores,[55] and many retail locations (e.g., grocery stores, convenience stores) sell both tobacco and alcohol products,[55,56] indicating overlap of the retail environments for these substances.

Retail environments for tobacco and alcohol are both at least partially characterized by brick-and-mortar retailers that provide off-premise or on-premise use. However, tobacco retailers are dominated by off-premise use, whereas alcohol retailers are composed of a mix of on-premise and off-premise use. Absent a national license requirement for tobacco and alcohol retailers in the United States, common data sources for identifying retailers are state/local licensing and commercial business lists.[57,58] Using commercial business lists, 1 study estimated that, in 2018, there were 325,884 tobacco retailers in the United States that sold tobacco products for off-premise consumption.[59] In comparison, there were 652,159 off-premise and on-premise alcohol retailers in the United States in 2019,[60] or about twice as many physical locations for alcohol retailers as for tobacco retailers. Retail alcohol sales are also characterized by regulations

determining requirements for sales of beer, wine, and spirits; 17 states along with some counties and cities in other states have monopolies on wholesale or retail sales of some or all alcohol-containing beverages.[25] Differences in prevalence of alcohol use and tobacco use lead to different relative densities of retail outlets per person who uses tobacco or alcohol. In 2018, there were approximately 57 tobacco retailers per 10,000 adults who reported past-month tobacco use, which is 1.2 times greater than the number of alcohol retailers per 10,000 adults who reported past-month alcohol use (approximately 49 retailers per 10,000; see Figure 9.1). In contrast, there were 115,562 food stores in the United States in 2019, or about 3 grocery/convenience stores per 10,000 people.[61,62]

Figure 9.1 Retail Outlets for Tobacco and Alcohol, United States, 2018: Number of Retailers per 10,000 People Who Use Tobacco or Alcohol



Note. Alcohol retailers include on- and off-premise retailers. *Alcohol use* is defined as use in the past month by individuals ages 21 and older; *tobacco use* is defined as use in the past month by individuals ages 18 and older.
Source: Beverage Information Group 2019,[63] Kong et al. 2022,[59] SAMHSA 2020.[64,65]

For both tobacco and alcohol, the legal age of sale or purchase is 21 in the United States; tobacco sale laws prohibit retailers from selling tobacco products to individuals under 21 years of age, and alcohol laws prohibit possession or purchase of alcohol products by individuals under 21 years of age (see chapter 8, "Minimum Legal Drinking Age and Tobacco 21 Laws"). States and localities vary in terms of exemptions and penalties for youth purchase, possession, and use of tobacco and alcohol.[66-71] Tobacco and alcohol are also sold online, which can facilitate underage access to both products.[72-74] In 2017, 24.8% of people who used e-cigarettes and were younger than the minimum legal age of sale for tobacco products in the United States (then 18 years of age) reported buying their products online.[75] In 2018, 13.0% of middle and high school students ages 9 to 17 who used tobacco reported buying products on the internet.[76] In 2022, 9.5% of U.S. youth ages 16 to 19 who reported past-year e-cigarette use purchased e-cigarettes online.[77] In a 2022 survey conducted in Rhode Island, 7.4% of young adults (ages 18–25) who reported

lifetime drinking purchased alcohol using an application (app) or the internet to obtain their products through home delivery services.[78] Around 10% of these purchases were made by people who were younger than the minimum legal age for drinking and purchase (21 years old).[78] The increasing use of home alcohol delivery services in 2020 and 2021, during and post the COVID-19 pandemic, further boosted internet alcohol sales.[79]

Online sales of tobacco and alcohol pose significant regulatory challenges. In 2009, the U.S. Congress passed the Prevent All Cigarette Trafficking (PACT) Act to prohibit cigarettes and smokeless tobacco sales to consumers younger than the legal sales age and to prevent sales that do not collect applicable taxes.[80] Despite the extension of the PACT Act to cover online e-cigarette sales in 2021, studies find that online e-cigarette vendors often fail to comply with required tax collections and sales bans to minors.[81-84] Consequently, consumers cite low prices and convenience as the main motivations for purchasing e-cigarettes from the internet.[85] Similarly, although alcohol online purchase and home delivery should be accompanied by ID verifications to ensure compliance with the National Minimum Drinking Age Act of 1984, which prohibits the purchase or public possession of alcohol by individuals under 21 years old, some states do not have explicit policies for age verification in online purchases and home deliveries of alcohol, suggesting noncompliance,[86] and successful orders by underage buyers are sometimes delivered without appropriate age verifications.[78]

Environmental Factors: Retail Availability and Marketing Environments

This section begins with a brief description of the cue reactivity literature that may explain why greater availability and marketing of alcohol could contribute to tobacco initiation and use and deter cessation. Cue reactivity refers to a complex neurobiological state where a person has cognitive, psychological, and behavioral reactions in response to substance-related stimuli.[87] Next, this section reviews research about retail availability for tobacco and alcohol, characterizing where the environments intersect and associations of greater availability with tobacco and alcohol use and other health harms. Finally, this section reviews research on the marketing environments for tobacco and alcohol, compares marketing expenditures, and reviews literature about the comarketing of these substances and effects of exposure to alcohol marketing on tobacco use by youth and adults. Recurrent themes in this section suggest gaps in understanding where the retail environments for tobacco and alcohol intersect, whether alcohol availability influences tobacco use or co-use, and whether exposure to alcohol marketing is associated with tobacco use or co-use.

The substance use literature on cue reactivity provides a theoretical rationale for why and how consumers react to tobacco and alcohol marketing and comarketing, and how cue reactivity plays a role in tobacco and alcohol use. With regard to tobacco and alcohol marketing, studies have demonstrated that certain aspects of the marketing can stimulate and reinforce cognition and neurobiological states (e.g., craving) regarding tobacco or alcohol that can influence use.[88-93] Further, this literature indicates that comarketing of tobacco and alcohol products will likely reinforce the use of both tobacco and alcohol. Tobacco and alcohol marketing frame the product and the behavior in positive ways (e.g., romance, achievement, adventure) without showing the harmful consequences of use.[23,94,95] This further supports the hypothesis that exposure to alcohol marketing could encourage tobacco use. According to a 2022 systematic review of 37 studies, alcohol cue exposure was found to increase tobacco cravings in 60% of studies and increased ad

libitum smoking in 18%.[91] In an eye tracking study involving people who drink heavily (>7/14 drinks per week for women/men) and smoke cigarettes (>5 cigarettes per day), participants attended more to cigarette and alcohol cues than to water cues when all 3 cues were presented simultaneously.[96] In the first study to disentangle the separate and combined effects of smoking and alcohol cues, results suggest that both smoking and alcohol cues enhance cue-specific craving and reward value (as measured by spending) for these substances relative to single-substance and neutral cues.[97] Such findings have important implications for people who use tobacco and alcohol and who are trying to quit or reduce use of either or both substances. Moreover, other psychological theories can also inform understanding of the impact of exposure to marketing and media portrayals of tobacco or alcohol on use by youth and young adults, including social learning and social cognitive theories and the message interpretation processing model.[98-100] For example, the message interpretation processing model predicts that positive affect evoked by alcohol portrayals is associated with the desire to emulate characters portrayed in alcohol ads, which predicts positive expectancies and liking of/desire for beer-related toys and brands and subsequent alcohol use. To the extent that tobacco and alcohol use co-occur in media portrayals and marketing, the message interpretation processing model would predict an effect of alcohol portrayals and marketing on tobacco-related attitudes and behaviors. Further investigation of cross-cue reactivity and cross-substance effects of marketing exposure is needed. Moreover, given that cross-substance marketing is common (e.g., tobacco marketing includes alcohol-related cues, see the "Marketing Environments for Tobacco and Alcohol" section later in this chapter), whether exposure to alcohol marketing could promote susceptibility to use tobacco, encourage initiation, stimulate craving, increase use frequency, and/or deter cessation warrants investigation.

Retail Environment for Tobacco and Alcohol

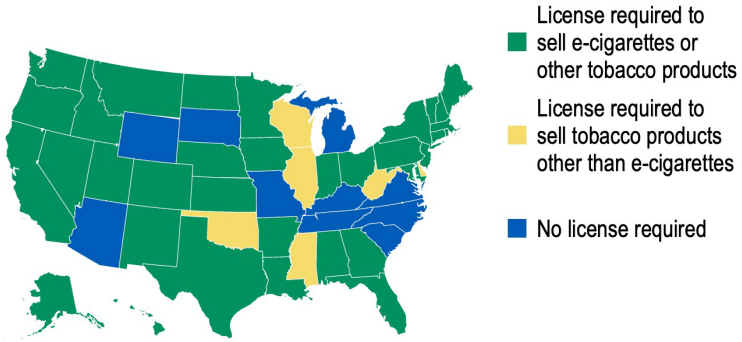
Availability of Tobacco and Alcohol

Both tobacco and alcohol products are obtained primarily from brick-and-mortar retailers,[53,86,101-104] yet licensing of these establishments is not required by every state, and there are no standard requirements across state licensing programs.[67,105-107] No national licensing requirement exists for either tobacco or alcohol retailers. Although the National Academy of Medicine recommends that all U.S. states license retailers that sell tobacco to reduce tobacco sales to minors,[108] not all states require licenses. As shown in Figure 9.2, as of September 30, 2024, the District of Columbia, 40 states, and 6 U.S. territories (American Samoa, Guam, Northern Mariana Islands, Palau, Puerto Rico, Virgin Islands) required tobacco retailers to have a state license; this tobacco licensing requirement encompasses e-cigarette sales in most but not all of these locations, with 6 states and 3 U.S. territories only requiring a license to sell tobacco products other than e-cigarettes.[67] Of the 40 states that require a state tobacco license, 36 of them have an annual fee associated with acquiring the state license, ranging from $6 in New Hampshire to $800 in Connecticut.[67] Four states require a state license but do not require a state licensing fee for selling tobacco.[67] Ten states and one U.S. territory did not require a state license for tobacco retailers. In terms of licensing for alcohol sales, as of January 1, 2024, a majority of states license private vendors to sell spirits, wine, and beer (Figure 9.2). Some states use a state-run system or a mix of state-run and licensing systems for the sales of spirits (13 states), beer (6 states), and wine (12 states).[105-107] When states do not use state-run stores or a mix of

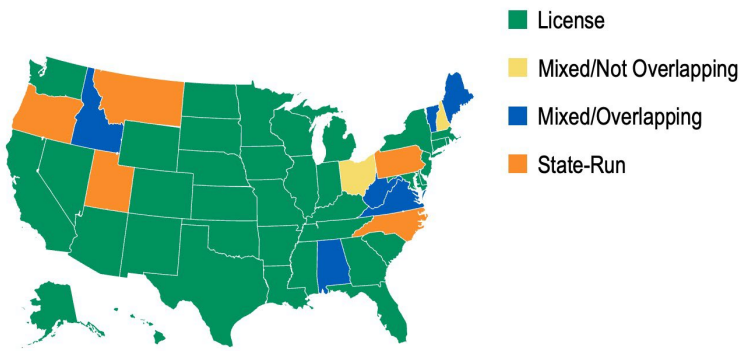
state-run stores and licensing systems for sale of all alcohol (or subsets of alcoholic beverages), they require a license for private retail distribution of alcohol.[105-107] Fees for state-based licenses range widely depending on factors such as whether the sale is for on-premise or off-premise consumption, the type of location for on-premise consumption (e.g., restaurant, theater, hotel, club), the size of the venue, and the type of alcohol sold.[109-111]

Figure 9.2 Licensing for Tobacco and Alcohol Sales by State

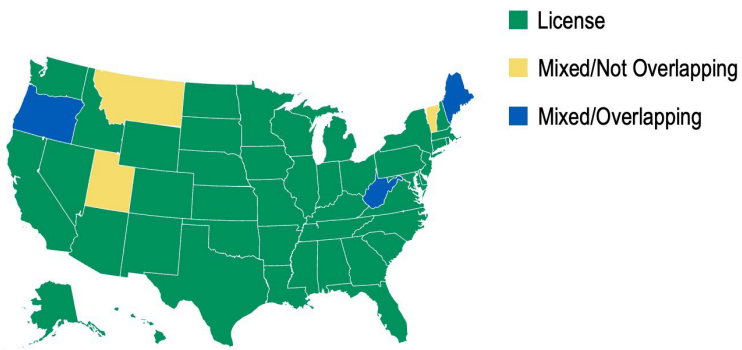
Panel A: Licensing for Tobacco Retailers (as of September 30, 2024)



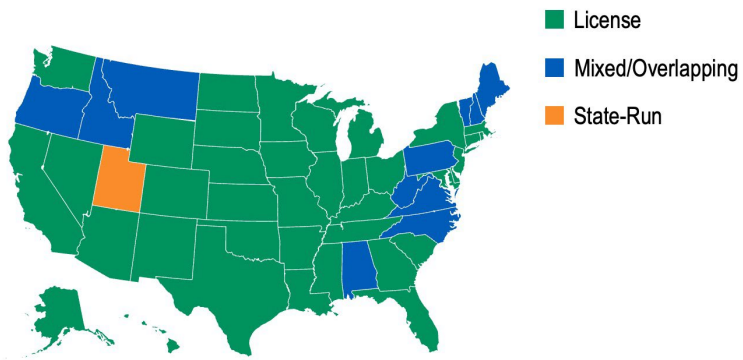
Panel B: Retail Distribution System for Spirits (as of January 1, 2024)



Panel C: Retail Distribution System for Beer (as of January 1, 2024)



Panel D: Retail Distribution System for Wine (as of January 1, 2024)



Note. Definitions of alcohol distribution systems are as follows: A *license* system is a system in which a state licenses private vendors to conduct retail sales. A state-run system is a system in which a state has the exclusive authority to set the price of and gain revenue from retail sales. A *mixed/not overlapping* system is a system in which some beverage subtypes are sold at retail through the state-run system, and some subtypes are sold through the license system, but none are sold through both. A *mixed/overlapping* system is a system in which one or more beverage subtypes are sold at retail through both state-run and license systems. Some categories have been combined for presentation, so maps may differ from those presented by NIAAA.[105-107]
Source: CDC 2024,[67] NIAAA 2025.[105-107]

Some tobacco and alcohol retailers allow for on-premise use of either or both substances,[112,113] but the intersection of these retail environments is not well understood. In 2019, 55.8% of all U.S. alcohol retailers allowed on-premise use.[60] Tobacco retailers that allow for on-premise use are likely to represent a subset of tobacco specialty shops, including hookah lounges, cigar bars, e-cigarette (vape) shops, and smoke shops[52,53]; these represented an estimated 4.1% of all U.S. tobacco retailers in 2017.[53] The ban on tobacco vending machines (excluding adult-only facilities) included in the 2009 Family Smoking Prevention and Tobacco Control Act[114] has the potential to reduce the number and proportion of on-premise alcohol retailers that allow sales of tobacco; however, this effect has not been documented. Understanding the impacts of tobacco vending machines and other sales restrictions is important given that on-premise retail sales facilitate product use. Comprehensive smokefree indoor air laws for restaurants and bars may be another potentially useful strategy to reduce on-premise use of tobacco and alcohol, particularly in the 19 U.S. states that, as of 2024, do not have comprehensive laws requiring smokefree restaurants and bars.[115] (For more information about state tobacco laws, see the American Lung Association's State Legislated Actions on Tobacco Issues resource.)

Data about the prevalence and location of U.S. tobacco retailers who also sell alcohol are incomplete. In 2017, 10.7% of U.S. tobacco retailers were classified as alcohol stores, but the total proportion of tobacco retailers that sell alcohol could not be determined.[53] In North Carolina, where off-premise sales of liquor are limited to state-controlled stores, 39% of 11,920 tobacco retailers sold alcohol in 2017.[116] However, in California, which does not limit sales of alcohol to state-controlled liquor stores, observational data from a statewide random sample of licensed tobacco retailers in 2019 found that 71.6% sold alcohol (L. Henriksen, Ph.D., personal communication, July 2025).[117] Apart from liquor stores, alcohol-selling tobacco retailers in California were supermarkets, pharmacies, convenience stores, small markets, discount stores, tobacco stores, gas stations, and unspecified "other" store types.

A study of community pharmacies in Los Angeles found that 87.5% of chain and grocery store pharmacies sell alcohol.[118] Studies also indicate that pharmacies sell cigarettes at significantly lower prices and are more likely to advertise cigarette discounts than other tobacco retailers.[119,120] The sale of tobacco and alcohol in pharmacies is particularly concerning for both tobacco and alcohol availability. Moreover, tobacco and alcohol sales in pharmacies are incompatible with the goals of cancer prevention and health promotion. Research that compared metropolitan areas with high and low density of CVS stores found that the chain's voluntary removal of tobacco sales in 2014 was associated with a significant decline in cigarettes smoked per day among U.S. adults who smoked nondaily, but not among those who smoked daily.[121] In addition, compared to U.S. adults living in counties without CVS stores, those living in counties with CVS stores reported higher odds of quit attempts following the chain's removal of tobacco sales.[122]

Availability of tobacco and alcohol from the same retail source may facilitate use of both substances.[6,123,124] Intersecting retail environments also facilitate contemporaneous exposure to tobacco and alcohol marketing, contributing to social norms that may reinforce the acceptability of co-use.[125-127] In addition, retail availability of tobacco and alcohol creates opportunities to purchase both products. Among northern California adults who purchased alcohol from a grocery store in the past year, 4.8% also purchased cigarettes from the same store.[128] However, purchasing patterns might differ for store types other than grocery stores, and this study did not

analyze the proportion of cigarette purchasers who bought alcohol. Data that characterize which products consumers purchase as well as the price paid are needed to better understand simultaneous purchases of tobacco and alcohol in the United States. For example, a study conducted in Finland found that cigarettes were bought more frequently with purchases of beer/hard cider than purchases without alcohol, but the relationship was nonlinear, with the greatest cigarette expenditures in shopping baskets with midprice purchases of beer/hard cider.[129]

Place-Based Disparities in Availability

The geographic distribution of off-premise tobacco and alcohol retailers are both influenced by economic forces that encourage stores to locate near one another or in specific neighborhoods and locations and policy factors, such as zoning laws for commercial land use.[130-136] The number of brick-and-mortar tobacco and alcohol retailers demonstrates that both products are highly available and accessible, although retail availability varies by state, locality, and product type.[6,137]

Like tobacco retailers,[138,139] off-premise alcohol retailers are concentrated in neighborhoods characterized by greater socioeconomic disadvantage and higher proportions of populations that have experienced discrimination based on race and/or ethnicity.[140,141] This is particularly true for non-Hispanic Black people[140,141] despite their lower rates of alcohol use compared with non-Hispanic White people.[142-144] For example, in Louisiana and Alabama, a higher level of isolation and segregation between White and Black residents was associated with higher alcohol outlet density in counties with some restrictions on alcohol sales.[145]

For both tobacco and alcohol outlets, decisions made decades ago regarding neighborhoods are still consequential today, because they are associated with the density of tobacco and alcohol retailers and contribute to differences in health outcomes. Research suggests that redlining (a historical practice of grading neighborhoods based on investment risk for home loans where race and/or ethnicity of the residents was a factor in the assessment) is associated with contemporary differences in the availability of alcohol[146] and tobacco.[134] Compared to census block groups in a 6-city region of northern California that were historically graded in 1937[133] through Federal Home Owners' Loan Corporation designations as A (Green - Best), block groups that were graded B (Blue - Still desirable), C (Yellow - Definitely declining), and D (Red - Hazardous)[134] had 3.4-, 8.7-, and 9.6-times higher density of off-premise alcohol outlets in 2015, respectively.[133] In New York City, block groups that were historically redlined (i.e., Grade D between 1935 and 1940) had significantly greater density of on-premise and off-premise alcohol outlets in 2014–2018 than block groups that were not redlined (i.e., Grades A, B, and C).[147] Similarly, land parcels in Baltimore, Maryland, and Genessee County, Michigan, that were redlined (Grade D designations in the 1930s) had a significantly greater density of alcohol outlets in 2020 (both off-premise and on-premise) than parcels that were not redlined (R. Sadler, Ph.D., personal communication, September 19, 2023).[146] As of 2026, only 1 study investigated whether redlining was related to the distribution of tobacco outlets; in 13 Ohio cities that were redlined, as neighborhood grades decreased from A (Best) to D (Hazardous), tobacco outlet density increased monotonically.[134]

Other studies about the availability of tobacco and alcohol have further identified associations between neighborhood characteristics and retail outlet density. For example, a study in North Carolina found there was a positive association between a tract-level index of neighborhood disadvantage (based on socioeconomic and demographic characteristics) and the density of tobacco retailers and between neighborhood disadvantage and alcohol retailer density, but there was an even stronger positive association between neighborhood disadvantage and retailers that sold both tobacco and alcohol.[116] Another North Carolina study found that census tracts with the greatest segregation between non-Hispanic White and non-Hispanic Black residents had 65% more alcohol retailers and 38% more tobacco retailers than tracts with the least segregation.[148] In Worcester, Massachusetts, the number of tobacco outlets and the number of alcohol outlets per 1,000 residents increased as the percent racial minority population increased and as the median household income decreased.[149] In California, census tracts with higher proportions of foreign-born Latino residents had greater tobacco retailer density but lower alcohol retailer density than those with lower proportion of foreign-born Latino residents.[150] Results from studies in Scotland,[151,152] New Zealand,[153] and Germany[154] found similar evidence supporting positive associations between the density of tobacco and alcohol retailers and magnitude of neighborhood socioeconomic deprivation.

Evidence suggests that availability of tobacco near schools normalizes use and facilitates youth access.[3,23] Similarly, availability of alcohol retailers near schools is thought to affect youth access and use.[155] In a 2011–2012 representative survey of U.S. households with youth (ages 13–16), 44.4% attended school within 1,000 feet of a tobacco retailer.[156] No nationally representative comparative data for proximity to alcohol retailers were identified. However, a study of approximately 242 adolescents in a 7-county area of Minneapolis-St. Paul, Minnesota, observed an average of 3.9 off-premise alcohol outlets (range 0–57) within a 3,000 meter (1.86 miles) roadway network distance of home and an average of 5.6 off-premise alcohol outlets (range 0–30) within the same distance from schools.[157] Research published as of 2026 suggests that tobacco retailers cluster near K-12 schools in the United States,[158] and a study in California suggests this pattern of tobacco retail clustering extends to college campuses as well.[159] Structural reasons for such patterns, such as zoning or other economic factors, could be investigated in future research. Parallel analyses for alcohol retailers are also warranted, as well as studies to understand potential associations between retailer density and tobacco and alcohol initiation and use.

Widespread availability of tobacco and alcohol implies that both are highly accessible, but fewer studies measure accessibility (e.g., travel time, travel costs) than availability. From a geographic perspective, availability refers to the supply of tobacco and alcohol within a given area, whereas accessibility refers to the relationship between these available supplies and the location of people who use tobacco and alcohol.[160] For example, 2 tracts could have identical availability of tobacco supplies in terms of total number of outlets and number of outlets per 1,000 people or per square mile, but a tract with a more highly connected space, such as through more direct roads or more extensive and affordable transit options, leading to lower travel times and lower travel costs, would have higher accessibility. According to a 2021 systematic review, only 1 of 40 studies about the tobacco retail environment measured travel time.[160] No comparable systematic review for off-site or on-site alcohol retailers was identified. However, a study conducted in the United States found that in 2013, the average driving time to 1 of the 7 nearest on-premise alcohol outlet was 5.89 minutes and average driving distance was 2.63 miles (about 4.3 kilometers).[161] At the

state level, a population-weighted measure of drive time (or distance) to 1 of the 7 nearest on-premise alcohol outlets ranged from 1.67 minutes (or .69 miles) in the District of Columbia to 15.29 minutes (or 7.91 miles) in Arkansas, with a median of 6.67 minutes (or 2.95 miles). Comparable analyses to characterize tobacco accessibility are warranted, and socioeconomic and racial and/or ethnic disparities in accessibility could be examined in future research.

The reviewed evidence suggests that tobacco and alcohol retailers are similarly concentrated in areas characterized by economic disadvantage and a higher proportion of racial and/or ethnic minority residents. However, few studies focused on where tobacco and alcohol sales co-occur; retail outlets that sell both tobacco and alcohol are an understudied source of cues that promote co-use. In addition, no systematic reviews or meta-analysis of place-based disparities in the density or proximity of alcohol retailers were identified.

Role of Alcohol Availability in Tobacco Initiation, Use, Cessation, and the Co-Use of Tobacco and Alcohol

Much evidence suggests that greater retail availability of tobacco is associated with greater use, with the caveat that directionality is difficult to disentangle.[2,160,162] A systematic review of 40 studies about the tobacco retail environment and smoking-related outcomes observed positive associations between retail density and smoking prevalence in 16 out of 21 studies (76.2%), greater tobacco use and smoking initiation in 9 out of 14 studies (64.3%), and lower cessation outcomes in 11 out of 13 studies (84.6%).[160] In addition, findings from a 2022 systematic review and meta-analysis of 37 studies from 8 countries suggest evidence of a positive association between tobacco availability and adult tobacco use.[162] Furthermore, the 2020 U.S. Surgeon General's report, *Smoking Cessation: A Report of the Surgeon General,* concluded that the "evidence is suggestive but not sufficient to infer that decreasing the retail availability of tobacco products and exposure to point-of-sale tobacco marketing and advertising increases smoking cessation".[2,p.12] This report also highlights that "evidence from alcohol control research indicates that limiting alcohol retail density can reduce excessive alcohol consumption; this relationship may translate to tobacco."[2,p.12]

Using comparable measures of alcohol availability, a systematic review of 13 studies observed significant positive associations between alcohol availability (retail outlet density) and drinking or heavy drinking in 17 of 34 reported effect estimates.[163] A scoping review of 34 studies (most published since 2012) concluded that the greater physical availability, as well as temporal availability (i.e., sales and delivery hours), of alcohol can contribute to permissive drinking environments.[164] However, most of these studies were conducted in the United Kingdom, Australia, or South Africa. One study undertook separate systematic reviews of natural experiments in the United States, Canada, Sweden, and Russia about reduction in sales days/hours and alcohol outlet density on alcohol consumption.[165] It concluded that all seven studies restricting temporal availability and three of four studies restricting physical availability reduced per capita alcohol consumption. Although there were too few studies about physical availability for a meta-analysis, the meta-analysis of temporal availability studies found that adding 1 day of sale was associated with a 3.4% increase in per capita consumption of total alcohol.[165]

Conclusions from systematic reviews of retail availability of tobacco and youth use indicate inconsistent evidence between availability and use, with more positive associations of youth cigarette smoking with availability near home than near school.[166-169] However, few studies examined availability of both tobacco and alcohol in relation to youth use of these substances. A telephone survey of 1,538 young adults (ages 18–30) from randomly selected households in 24 California cities assessed co-use of alcohol with tobacco or cannabis in places that respondents could recall drinking alcohol in the past 3 months: own home, someone else's home, bar/restaurant, and outdoor/public places.[170] Overall, 12.5% of events involved simultaneous co-use of tobacco and alcohol, but co-use was unrelated to place; 6.8% of events involved simultaneous use of all 3 substances, and simultaneous polysubstance use was significantly less likely to occur in bars/restaurants than in own/other's home or outdoor spaces. In a cross-sectional survey of 205 Latino high school students living near the California-Mexico border, proximity (i.e., roadway distance from residence to nearest outlet, a measure of accessibility) to tobacco and alcohol outlets was positively associated with a combined measure of lifetime use of tobacco and alcohol use after controlling for individual characteristics.[171]

Because tobacco and alcohol use share common risk factors, some studies investigate whether features of the alcohol environment are independently associated with tobacco and alcohol use. For example, in a nationally representative survey of college students, participants attending colleges where a higher percentage of students perceived that alcohol was very easy to obtain had substantially higher odds of both cigarette smoking and binge drinking compared to students at campuses with lower perceived availability, even after controlling for other individual-level factors (e.g., sex, age, race, on- or off-campus residence, individual's own perception of alcohol availability), campus-level factors (e.g., school-level binge drinking rates), and geographical region.[172] Additional research investigating whether greater alcohol availability promotes urges to smoke and deters smoking cessation, and whether this varies by setting (e.g., home, school, other setting) is needed.

Availability and Affordability

The higher concentration of tobacco retailers and off-premise alcohol retailers in socioeconomically disadvantaged neighborhoods may lead to greater competition between stores, suggesting that neighborhood residents are exposed to greater availability and lower prices for both products, two factors that encourage product use.[139,173-178] However, more evidence of place-based differences in price has been found for tobacco than for alcohol. For example, a 2020 systematic review of 20 studies found that cigarettes cost significantly less in neighborhoods characterized by lower socioeconomic status (versus higher socioeconomic status) and higher proportions of youth and Black residents (versus neighborhoods with lower proportions of these groups).[173] Similarly, a study in two Canadian provinces (Ontario and Quebec) found that cigarette prices near secondary schools were significantly lower in neighborhoods with a median household income one standard deviation below the provincial median than one standard deviation above the median.[179] Such findings align with evidence from tobacco industry documents indicating that manufacturers have used race, class, and geography

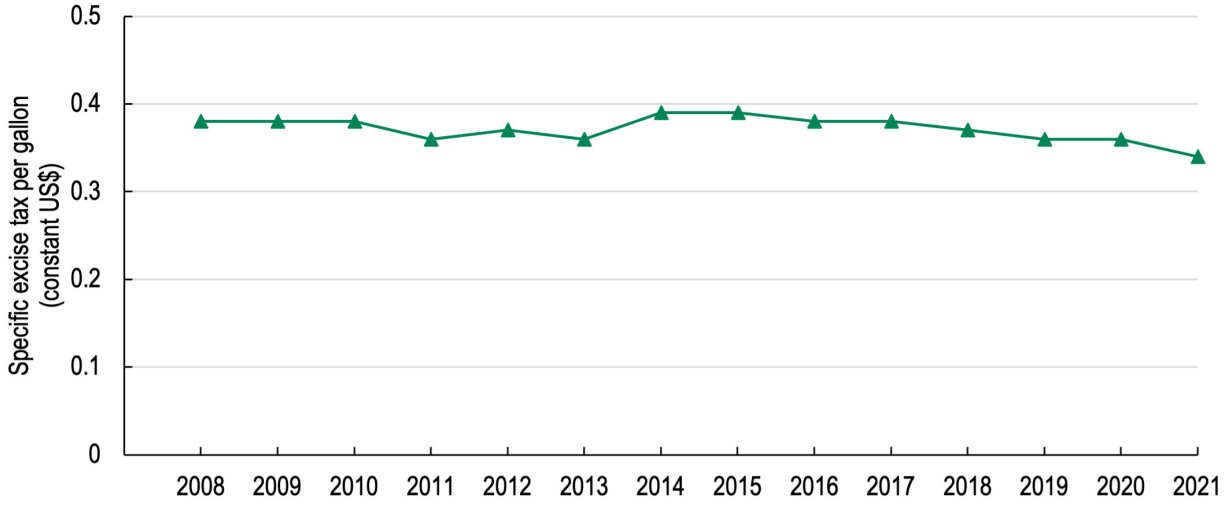
for target marketing of price promotions.[139,180] Although studies of availability pricing do not control for localized competition (number of tobacco retailers nearby), models typically adjust for store type and neighborhood demographics, such as population density, income and race and/or ethnicity,[181] factors that are typically correlated with the number and density of tobacco retailers.[138,182]

Comparatively fewer studies of place-based differences in alcohol prices were identified. In Australia, a 1 decile increase in an index of socioeconomic advantage was associated with a 1.3% increase in the minimum price for a 750 ml bottle of wine, with lower prices observed in areas with more students. In the United States, a study of alcohol outlets in the year 2000 observed that prices of two leading beer brands were related to neighborhood income and racial composition, but not the proportion of youth in the store neighborhood.[174] However, focusing on two leading brands could miss place-based differences in presence and price of beer sub-brands targeted to audiences defined by economic disadvantage and race and/or ethnicity. No studies were found that compared prices of tobacco and alcohol in the same stores. This is a noteworthy gap in the literature, given the strong association between price and use.

As will be discussed in the section "How Alcohol Tax and Pricing Policies and the Affordability of Alcoholic Beverages Affect Tobacco Use," price is an important determinant of product use. The price of an alcoholic drink relative to disposable income decreased between 1950 and 2011, making alcohol more affordable.[183] Beginning in the 1960's the inflation-adjusted value of state excise taxes on beer, spirits, and wine declined sharply until the early 1980s, and since then have declined slowly or for beer have stayed fairly constant (see Figure 9.3).[184] Declines in state-level beer taxes, combined with the federal tax reduction for beer in 2017, has reduced the overall tax burden on beer, leaving room for the alcohol industry to keep prices stable (see Figure 9.4). In contrast to the largely unchanged average inflation-adjusted price per gallon of beer, the average inflation-adjusted price per pack of cigarettes has increased steadily (see Figure 9.4).

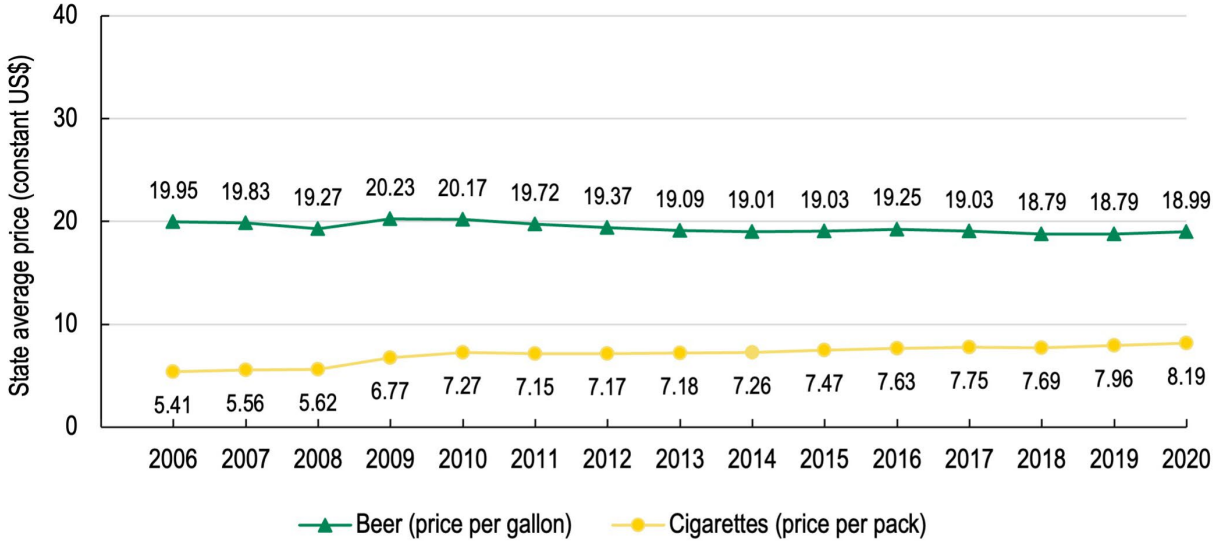
Whether increasing alcohol prices will reduce tobacco use hinges on the economic relationship between the two products. If tobacco and alcohol are economic substitutes, higher alcohol prices will increase tobacco use. In contrast, if tobacco and alcohol are economic complements, increasing alcohol prices will reduce tobacco use. However, the lack of changes in beer prices since 2006 poses challenges in identifying how these prices affect tobacco use. As a result, studies on the economic relationship between tobacco and alcohol using recent data are limited by this constrained variability.

Figure 9.3 Inflation-Adjusted Average State-Level Specific Excise Taxes per Gallon of Beer (5% Alcohol) in the United States, 2008–2021



Note. Specific tax amounts adjusted for inflation using 2022 as the base year. Tax amounts for beer are per gallon (5% alcohol).
Source: NIAAA.[185,186]

Figure 9.4 Inflation-Adjusted Average State Price per Gallon of Beer and per Pack of 20 Cigarettes in the United States, 2006–2020



Note. Beer prices are based on Heineken beer, which was selected by the Council for Community and Economic Research to consistently measure beer price over time and across locations. Beer price data are derived from the Council for Community and Economic Research. Cigarette price data are derived from the Centers for Disease Control and Prevention State Tobacco Activities Tracking and Evaluation (STATE) system and the Orzechowski and Walker Tax Burden on Tobacco data set.
Source: CDC 2019,[187] Council for Community and Economic Research 2022,[188] Orzechowski and Walker 2021.[189]

Summary: Retail Environment for Tobacco and Alcohol

Both tobacco and alcohol products are obtained primarily from brick-and-mortar retailers, but variations in state licensing programs, fees, and on-premise use policies for both products make it difficult to estimate how frequently tobacco and alcohol products are available from the same retail outlet and the impact on co-use. Intersecting retail environments can facilitate exposure to marketing for both products that may normalize co-use and purchase of both products. There is consistent evidence that both tobacco and alcohol retailers are concentrated in neighborhoods with greater socioeconomic disadvantage. Tobacco and alcohol retail availability are both associated with higher rates of tobacco and alcohol use, and some evidence suggests that limiting retail availability can increase smoking cessation or decrease alcohol consumption. Place-based differences in availability may also result in lower prices for tobacco and alcohol in neighborhoods with lower socioeconomic status. Most studies addressing this question have focused on tobacco prices.

Marketing Environments for Tobacco and Alcohol

Although the intersection of tobacco and alcohol in product marketing and other media is widely studied, too little is known about the effects of exposure to alcohol marketing on tobacco use or co-use. This section first compares marketing expenditures for tobacco and alcohol. It then summarizes literature on the comarketing of both substances, tobacco and alcohol brand placement in media, portrayals of tobacco and alcohol use in media, and associations of exposure to alcohol marketing with tobacco use. It is important to note that regulation of tobacco and alcohol marketing differ significantly. Although U.S. federal law bans the advertising of cigarettes, cigars, and smokeless tobacco on television and radio, it allows the advertising of other tobacco products and alcohol products using these channels. A detailed comparison of marketing restrictions for tobacco and alcohol is presented in Appendix 9B.

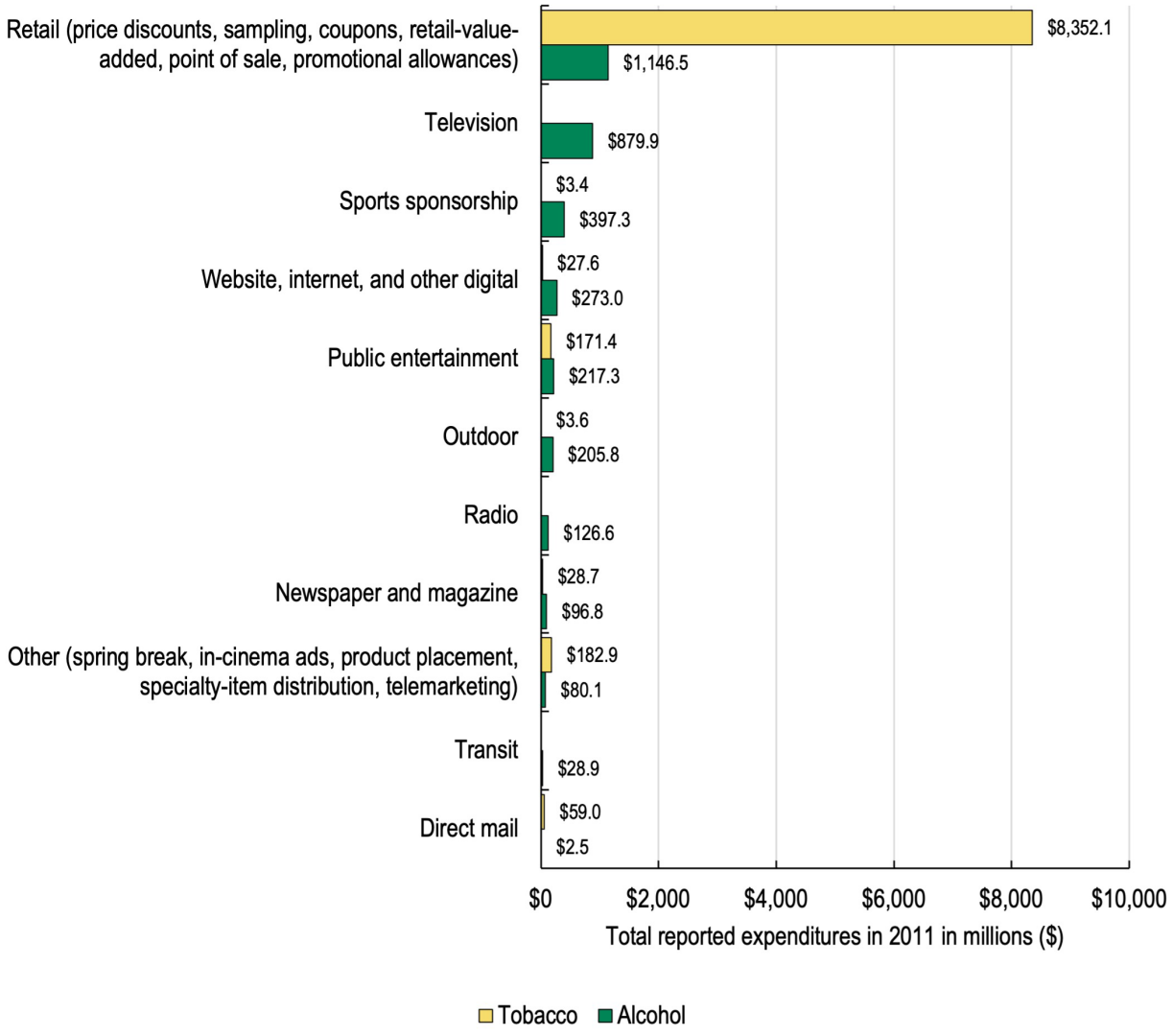
Marketing Expenditures for Tobacco and Alcohol Products

The U.S. Federal Trade Commission (FTC) is a federal agency that protects consumers from unfair or deceptive practices and advertising, including both tobacco and alcohol marketing.[190-192] The agency has reported to Congress on cigarette sales and marketing expenditures for cigarettes since 1967, smokeless tobacco since 1986, and e-cigarettes since 2015.[193-195] Additionally, the FTC has addressed concerns about the effects of alcohol marketing on youth and has published periodic reports on the status of the alcohol industry's compliance with its own self-regulatory standards.[196-199]

In its 2014 alcohol report (the most recent alcohol report available as of 2026), the FTC issued Special Orders to 14 major alcohol companies, which represented 79% of the volume of alcohol sold in the United States. The Special Orders compelled the companies to disclose annual marketing expenditures for 2011. In that year, reported alcohol marketing expenditures totaled about $3.45 billion.[196] Figure 9.5 compares expenditures from FTC reports on tobacco (cigarettes and smokeless combined), totaling about $8.83 billion in 2011 (the only year in which both tobacco and alcohol marketing expenditure data are available from FTC). Although reported marketing expenditures were 2.6 times higher for tobacco than alcohol, alcohol companies did not report expenditures related to price discounts,[196] a noteworthy data gap considering that price discounts represented 81.2% of tobacco marketing expenditures in 2011.[193,194] Price discounts

are paid to retailers or wholesalers in order to reduce the price of cigarettes to consumers; price discounts may include off-invoice discounts, buy-downs, voluntary price reductions, and trade programs.[193] These primary vehicles for tobacco marketing contribute to tobacco-related health disparities related to socioeconomic status, race and/or ethnicity, and sexual orientation.[139,200-202]

Figure 9.5 Tobacco and Alcohol Marketing Expenditures in 2011 Reported to the Federal Trade Commission



Note. Marketing expenditures for tobacco include cigarettes and smokeless tobacco. *Price discounts* (under the Retail category) are included for tobacco marketing totals but are not included for alcohol product marketing totals. *Retail-value-added* refers to promotions involving free or discounted products. *Point of sale* expenditures include materials displayed or distributed at retail locations. *Promotional allowances* are paid to retailers, wholesalers, or others to facilitate the sale or placement of products. *Specialty-item distribution* refers to distribution of non-tobacco or alcohol product items, such as coasters, shirts, hats, sunglasses, keychains, and lighters.
Source: Federal Trade Commission.[193,194,196]

Marketing of Tobacco and Alcohol

Some tobacco marketing strategies employed in the United States may reinforce co-use of tobacco and alcohol. This potential for synergy is enhanced by co-ownership, such as Altria's approximately 8% ownership of Anheuser-Busch,[203] Philip Morris's former ownership of Miller Brewing, and Altria's former ownership of wineries.[28,30] Moreover, the potential for synergy is also increased by instances of cooperation between tobacco and alcohol companies. For example, tobacco industry documents indicate that tobacco companies also studied co-use of cigarettes and alcohol and the drinking behaviors of customers who smoked cigarettes.[28] To link cigarettes with alcohol, tobacco companies engaged in (1) joint sponsorship with alcohol companies of sporting, art, and other cultural events; (2) joint promotion of cigarette and alcohol products; and (3) development of cigarette promotions featuring alcohol or alcohol paraphernalia and offering cigarette-branded gifts with alcohol purchases.[28] A Philip Morris document details a corporate leverage project that analyzed the implications of cross-promoting the brands they owned in 1987 (specifically referring to the operating units of Philip Morris USA, Miller Brewing Company, and General Foods). They concluded that sponsorships "represent an important area for operation units to work together" and identify a "mutual need to capitalize on other operating units' involvement".[204,p.63] However, they also note that, because of a negative view of cigarettes, an implicit cross-promotion of the brands (e.g., retailer sponsorship event, placing a group of brand-specific advertising for each brand together in one promotional insert) involved less risk of reducing a favorable view of the brands than explicit association methods (e.g., "cross-ruff" coupons or placing coupons for cigarettes on beer products).[204] This document also noted that promotion directors "believe capitalizing on joint arrangements or programs can assist in achieving specific operating unit consumer promotion goals or objectives" and specifically identified consumer promotion, event marketing, and materials purchasing as potential opportunities for joint activities.[204,p.22-24]

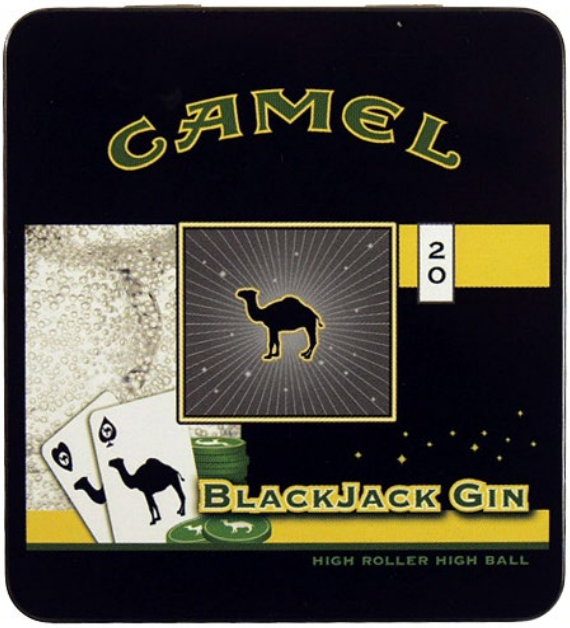
The Role of Alcohol in Marketing Tobacco Products. Tobacco industry documents describe the development of alcohol-flavored tobacco products, emphasizing the overlap of tobacco and alcohol in product use. These efforts to develop and market alcohol-flavored tobacco products include Lorillard's proposal to develop crème de menthe flavored cigarettes (which was not implemented) and R.J. Reynolds' effort to test brandy-flavored cigarette prototypes, called Project Prestige Family.[205] Prior to the enactment of the 2009 U.S. Family Smoking Prevention and Tobacco Control Act, which banned non-menthol characterizing flavors, including alcohol-flavors, in cigarettes,[206] R.J. Reynolds marketed alcohol-flavored Exotic Camel cigarette varieties under names like Margarita Mixer and Kauai Kolada.[205]

Industry development of alcohol-flavored tobacco products was not unique to cigarettes (see Figure 9.6). Although Brown & Williamson abandoned an effort to develop bourbon-flavored snuff,[207] examples of alcohol-flavored chewing tobacco that have been sold in the United States include Stoker's Moonshine (National Tobacco Company)[208] and bourbon-flavored Copenhagen Black (U.S. Smokeless Tobacco Company).[209] Between 2016 and 2019, sales of alcohol-flavored cigarillos decreased from 8.0% to 5.9% of total unit sales, but sales of alcohol-flavored large cigars increased from 2.6% to 4.6% of total unit sales over the same period.[210] Across cigar categories, alcohol-flavored products ranked fourth in sales, less frequently purchased than tobacco, fruit, and candy/sweet flavors, but more frequently purchased than menthol, coffee, and other flavors.[210] Between 2009 and 2020, the market share for wine-flavored cigars in

U.S. convenience stores ranged from a high of 19.5% in 2010 to 9.0% in 2020.[211] As of 2026, the sale of alcohol-flavored cigars continues[212]; examples include Banana Daiquiri, Blackberry Mojito, Boozy Watermelon, Coastal Cocktail, Hard Lemonade, and Irish Cream.[213] A 2018–2019 study of tobacco retailers in Washington, DC, found that stores in census tracts with the highest (versus lowest) percentage of Black residents had more products with alcohol flavor descriptors; a majority of the flavored tobacco products identified in this study were cigars.[214]

Figure 9.6 Examples of Tobacco Products with Alcohol Flavorings

Panel A: Camel Blackjack Gin Cigarettes



A) Pack of flavored cigarettes called "Blackjack Gin", introduced in 2005 by R.J. Reynolds Tobacco Company; the product is now discontinued.[215]

Panel B: MarkTen Bourbon Blend

B) Image from a 2017 email advertising the sale of a bourbon-flavored cartridge for the MarkTen e-cigarette from Altria, the parent company of Philip Morris USA; the product is now discontinued.[216]

Source: Reproduced with permission from Trinkets & Trash.[215,216]

Tobacco companies also continue to market alcohol-flavored tobacco in other forms. In a convenience sample of more than 8,000 noncigarette tobacco products available from online retailers, Morris and Fiala (2015)[217] reported that liquor-flavored tobacco products were available for all 13 tobacco product categories examined, except for dissolvable and roll-your-own tobacco.[217] In a 2018 study of online searches for the top 20 brands in the cigar/cigarillo and e-cigarette categories, 48 types of alcohol-flavored tobacco products marketed by 409 brands of cigars/cigarillos and e-cigarettes were identified.[218] Alcohol-flavored tobacco products were categorized predominately as mixed drinks (52%), followed by spirits (23%), liqueurs (15%), and wine or beer (10%). To characterize e-cigarettes flavors, Krüsemann and colleagues[219] reviewed 28 articles and proposed an e-liquid flavor wheel of 13 main flavor categories, one of which was alcohol. A content analysis of 59 e-cigarette websites in 2011 showed that alcohol-flavored e-cigarettes were featured on 10% of 59 e-cigarette websites.[220] Of 14,477 e-liquids available for sale online in 2021, 2,433 (16.8% of total) were alcohol-flavored, almost double the number of tobacco-flavored e-liquids.[221] In addition, the U.S. Food and Drug Administration (FDA) has issued warning letters to online retailers for selling unauthorized e-cigarettes, including those with packaging that imitates bottles of alcohol.[222] The marketing and availability of alcohol-flavored tobacco products, including e-cigarettes, are concerning because alcohol flavors in tobacco products are shown to be associated with both tobacco product preference and alcohol use among U.S. youth.[100,218,221] However, among adults, a volumetric choice experiment survey (where respondents are presented with an array of units of a good or service and choose how much to select)[223] conducted among 181 people who currently used cigarettes, e-cigarettes, and alcohol suggested that availability of alcohol flavors in e-cigarettes would not significantly affect alcohol, cigarette, or e-cigarette purchases.[224]

Several studies document the use of alcohol cues to promote tobacco products (see examples in Figure 9.7). A content analysis of social media accounts for 47 premium cigar brands identified references to alcohol (predominately bourbon, whiskey, and gin) in 12.8% of social media posts.[225] In premium cigar magazines, alcohol use was mentioned in 40% of articles and depicted in 27.6% of advertisements.[226] As of 2026, comarketing of tobacco and alcohol is not unique to cigar products. Several studies document the use of alcohol cues to promote tobacco products. For example, marketing for ZYN and Velo nicotine pouches portrays product use with alcohol in bars or clubs.[227] Lifestyle magazines (*Unlimited, All Woman, CML, Pleasure Scene [P.S.], Real Edge,* and *Flair*; 1996–2007, as available) produced by tobacco companies featured alcohol on the cover of 19% of 73 issues, and alcohol appeared in 27% of tobacco advertisements but only 8% of nontobacco advertisements.[228] In addition, a content analysis of cigarette and alcohol advertisements in young-adult magazines (2003–2004) observed that 28% of 114 cigarette advertisements contained a written or visual reference to alcohol, and such advertisements were more likely than other cigarette advertisements to portray smoking.[229] Notably, none of the 317 alcohol advertisements referenced cigarettes, suggesting that the comarketing of tobacco and alcohol is predominately initiated by tobacco companies as a tobacco marketing appeal. Globally, tobacco and alcohol companies also similarly market their products to population subgroups defined by age, education, and race and/or ethnicity,[230] which may increase the use and co-use of tobacco and alcohol among targeted groups.

Figure 9.7 Examples of Tobacco Product Advertisements that Include Alcohol

Panel A: Cheyenne Cigars Instagram Post

A) A 2019 Instagram post from a cigar brand's account featuring a pack of cigars and beers on a table, captioned "Cheers to it being National Beer Day!"

Panel B: Skoal Magazine Advertisement

B) A 2020–2022 magazine advertisement for Skoal smokeless tobacco (from Altria, parent company of U.S. Smokeless Tobacco Company), featuring a smokeless tobacco tin and alcohol on a table, captioned with the phrase "A pinch better together."
Source: Reproduced with permission from Trinkets & Trash.[231,232]

The Role of Tobacco in Marketing Alcohol Products. Tobacco-flavored alcoholic beverages are also marketed in the United States. For example, Perique liqueur is derived from and named for a rare variety of tobacco grown primarily in Louisiana.[233] The distilled spirit was first released in 2006 and is predominately sold in specialty shops.[234,235] Consumers may associate the liqueur with Natural American Spirit's Perique blend cigarettes,[236] which is concerning because studies show that U.S. adolescents and adults perceive Natural American Spirit cigarettes to be less harmful than other brands.[237,238] Tobacco is also used as an ingredient to develop tinctures, bitters, and infusions for cocktails.[239] For example, "nicotini" products, which are derived from tobacco-infused vodka without distillation, may contain high concentrations of nicotine.[240] However, little is known about the composition and toxicity of such products, bartenders' and customers' use of alcohol with tobacco ingredients, and awareness of potential health harms.

Tobacco and Alcohol Brand Placement in Media

As discussed previously, the literature on cross-cue reactivity indicates the potential for cross-substance effects of exposure to alcohol marketing on tobacco use and vice versa. One marketing strategy to consider is brand placement in various media channels. Although the 1998 Master Settlement Agreement eliminated paid cigarette and smokeless tobacco brand placement in U.S. films and television,[241] the practice of unpaid tobacco brand appearances persists (especially evident in social media)[242,243] alongside continued alcohol brand placement (paid and unpaid).[244] For example, a content analysis of the 100 highest grossing movies from 1996 through 2009 observed 2,433 alcohol brand appearances and 500 tobacco brand appearances.[244] These results are concerning because tobacco and alcohol brand recognition and exposure to brand placement can influence tobacco use, alcohol use initiation, and drinking behavior among adolescents.[245-248] Tobacco and alcohol brand placement and exposure to tobacco and alcohol imagery exist in myriad channels beyond movies, including popular music lyrics,[249] music videos,[250,251] and television programs,[247,252] many of which are now available on apps and streaming platforms.[253]

Like tobacco advertising,[23] alcohol advertising contains framing that may appeal to youth, such as depictions of positive emotional experiences, camaraderie, achievement/success, social positioning, adventure/spontaneity, romantic and sexual connotations, humor, and animations,[254] magnifying public health concerns about the effects of exposure to alcohol marketing and potential effects on co-use of tobacco and alcohol. A review of 47 studies of alcohol marketing online identified content that featured strategies that could appeal to young audiences (e.g., interviews with celebrities; free downloads of content such as music, pictures, and messaging accessories; free applications for entertainment, hangover advice, and alcohol intake monitoring) and that exposure was positively associated with alcohol use.[255] Also concerning is evidence of direct advertising of alcohol to youth,[256] in violation of the alcohol industry's self-regulation codes related to digital marketing.[257-259] A 2016 study created profiles for fictitious underage and of-age youth on Twitter (now X) and Instagram and found that all user profiles, regardless of age, could view and interact with (e.g., comment, like, share) alcohol content, and that on Instagram, all users could follow official alcohol brand pages and received alcohol advertisements.[256]

In 2010, 23.7% of alcohol television advertisements in the 25 largest U.S. television markets were placed during programs for which >30% of the audience was between ages 2–20, exceeding the threshold for youth audience established in the alcohol industry's 2003 self-regulatory codes.[260] From 2001 to 2011, youths were overexposed to alcohol magazine advertising relative to adults around 73% of the time.[261] It is not known whether exposure to such targeted marketing for alcohol stimulates tobacco initiation and whether it exacerbates disparities in tobacco use. However, these studies provide additional evidence that voluntary advertising regulation strategies are ineffective at protecting youth from exposure to alcohol advertising.

A comprehensive assessment of tobacco and alcohol marketing exposure could include billboards, which youth report as a source of exposure to marketing for alcohol[262] and e-cigarettes.[263,264] An analysis of marketing expenditures suggests that billboards are a likely source of exposure to marketing for nicotine pouches, as well.[265] There is also evidence of a disproportionate presence of alcohol billboards in neighborhoods with a higher proportion of African American and Hispanic residents.[266,267] Future research could consider whether exposure to alcohol billboards is associated with tobacco-related outcomes, such as cravings and urges to smoke.

In 2021, exposure to marketing or advertising for any tobacco product was reported by 75.7% of U.S. middle and high school students who reported any potential route of exposure (e.g., going to a convenience store, supermarket, or gas station; using the internet; watching television or streaming services or going to the movies; reading newspapers or magazines).[268] A figure comparing U.S. youth and young adult exposure to tobacco and alcohol marketing would be informative, but comparative data (measured the same way in the same instrument or across surveys) were not identified.

Tobacco and Alcohol Use Depictions in Media

Content analyses that report on both tobacco and alcohol portrayals (or mentions) have examined various media: video-on-demand platforms, social media, television shows, movies, and music videos. As is true of tobacco depictions,[23] alcohol depictions in popular media rarely portray harmful consequences of its use.[269] A 2019 analysis of Twitter (now X) content on tobacco use in the context of intoxication observed that 75% of tweets were favorable toward co-use of tobacco and alcohol, 10% reported increased susceptibility to use tobacco when intoxicated, and only 2% mentioned alcohol undermining tobacco cessation attempts.[270] A 2018 study of popular/trending content on video-on-demand platforms (e.g., Netflix, Videoland, YouTube) in the Netherlands found that nearly one-half (45.4%) of 529 programs portrayed alcohol use and 21.9% portrayed tobacco use.[269] Notably, 32.9% of programs containing alcohol use also portrayed tobacco use, more than any other risk behavior examined (drug use, barrierless sex, reckless behavior such as driving under the influence, intentional self-harm), suggesting that these programs reinforce co-use of alcohol with tobacco over other substances (e.g., cannabis, opioids).

Among 163 teenage characters in 81 top-grossing films for teen audiences (2007–2009), 20.9% drank alcohol and 5% smoked cigarettes.[271] Of the 19 teenage characters who were offered alcohol, only 2 refused. In Oscar-nominated films (2008–2011), a total of 515 substance use incidents were depicted, with nearly as many for tobacco (44.8%) as for alcohol (50.1%).[272] In a sample of male adolescents' favorite television shows (2015–2016), the number of visual or verbal displays for alcohol dwarfed those for tobacco (756 versus 49), and the incident rate was approximately 10 alcohol portrayals every hour compared to 1 tobacco portrayal every 2 hours.[273]

References to using tobacco and drinking alcohol are also present in books adolescents read, in music they hear, and music videos they watch. In a sample of 40 novels popular with teens in June to July 2008, 357 instances of substance use occurred at a rate of once every 39 pages, with alcohol (67.2%) and tobacco (22.1%) most frequently mentioned.[274] Among the most popular songs from *Billboard Magazine* year-end charts (2009–2011) in 4 genres—country, pop, urban, and rap—23% mentioned alcohol and 6.4% contained brand-specific references.[249] In addition, mentions of alcohol in popular music have increased over time. Between 1968 and 2008, the percentage of top-100 songs with any reference to alcohol (settings, use, or brands) increased nearly 4-fold, from 5% to 19%.[275] In a content analysis of music videos on YouTube for Billboard top-100 songs (2014–2020), alcohol appeared in 81.6%, cigarettes/cigars in 19.3%, e-cigarettes in 4.8%, and hookah in 3.3%.[276] In addition, alcohol was the most frequently branded substance in music videos (37 brands), followed by cigarettes/cigars (2 brands). However, this study did not report the co-occurrence of tobacco and alcohol use within the instances of brand appearance. Longitudinal studies that investigate the prevalence of tobacco and alcohol co-use depictions, trends in these portrayals, and associations with co-use behavior would be informative.

Some content analyses indicate that depictions of tobacco and alcohol use occur more frequently among cast members or characters of certain racial and/or ethnic groups. An analysis of MTV reality television shows (2004–2011) described the behaviors of 622 characters, all of whom were young adults or high-school aged youth.[277] A majority of cast members were shown drinking alcohol (63.7%), including 69.2% of young adults and 12.2% of high-school age youth. The prevalence of cast members shown drinking alcohol was 55.7% for non-Hispanic White cast members, 62.2% for Black cast members, 76.9% for Hispanic/Latino cast members, and 75.0% for mixed race cast members. Only 10.3% of cast members were shown smoking, but this was lowest for non-Hispanic White cast members (9.0%), compared with 13.3% for Black, 23.1% for Hispanic/Latino, and 30.0% for mixed race cast members; no high-school age youth were depicted smoking.

Associations of Exposure to Alcohol Marketing and Tobacco Use

The causal association between exposure to tobacco marketing and initiation and use of tobacco by youth and young adults has now been well documented.[1,23] Robust evidence also supports a causal effect of alcohol marketing on youth alcohol use.[12,127] Indeed, a synthesis of 11 narrative and systematic reviews of exposure to alcohol marketing and youth drinking found evidence for all nine Bradford Hill causality criteria (biological plausibility, experimental evidence, coherence

with other knowledge of the health risk, analogous causal relationships with similar exposures and outcomes, and evidence for the strength, consistency, specificity, temporality, and dose-response nature of the association).[12] In addition, 24 of 25 longitudinal studies investigating the relationship between exposure to alcohol advertising and drinking initiation found supporting evidence that greater exposure was associated with increased odds of initiation.[127]

One cross-sectional survey in 1995 investigated whether recognition and liking of advertisements for cigarettes and alcohol are associated with susceptibility to use these substances (separately).[278] The study was conducted among 386 eighth grade students, more than one-half of whom were classified as "nonsusceptible nonusers" or "susceptible nonusers." The study defined nonsusceptible nonusers as those who responded "no, definitely not" to questions regarding smoking and drinking intentions in the next 2 months; susceptible nonusers were those who gave any other response to smoking and drinking intentions questions.[278] After exposure to masked advertisements (brand names removed) for cigarettes and alcohol, a survey assessed whether students could correctly identify the brand name (brand recognition), liking, and intention to try the product. The relationship between liking and use status differed for tobacco and alcohol. Susceptible students who never smoked liked cigarette advertisements as much as those who currently smoked and more than nonsusceptible students who never smoked. For alcohol advertisements, liking scores increased monotonically, with lowest scores for nonsusceptible students who never drank, followed by susceptible students who never drank, and highest scores for those who ever drank. For both tobacco and alcohol, the most frequently recognized advertisements were not always the most liked,[278] perhaps indicating evidence of wear out (decreased liking with repeated exposure) even among a young audience. Because the study predates the 1998 Master Settlement Agreement, which placed restrictions on cigarette advertising, further research that examines whether recognition or liking of alcohol advertisements predicts susceptibility to tobacco and alcohol use is warranted.

A 2018 study compared attitudes toward full-page magazine advertisements for alcohol, tobacco (cigarettes, e-cigarettes, and smokeless tobacco), and nonalcoholic beverages among 1,220 adolescent boys (ages 11–16) from urban and rural Appalachian counties.[279] Ten-point Likert scale ratings of each advertisement (enjoyable, likeable, and appealing) were dichotomized to compare any favorable (nonzero) response with zero, finding a significantly greater percent of nonzero ratings for alcoholic and nonalcoholic beverages than for tobacco. Controlling for ever use of tobacco, adolescent boys whose peers used tobacco had significantly greater odds of favorable responses to all three categories of ads, including those for alcohol. However, this study did not assess alcohol use.[279] Indeed, one challenge to studying the cross-substance effects of exposure to tobacco and alcohol marketing on tobacco and alcohol use is to disentangle the separate and combined effects of exposure on susceptibility, initiation, use, and cessation. In addition, comparative studies of responses to tobacco and alcohol marketing are needed to keep pace with the rapidly changing landscape for tobacco products and social norms about their use,[280,281] as well as the changing landscape for marketing with the advent of algorithmically targeted programmatic advertising.[282,283]

Summary: Marketing Environments for Tobacco and Alcohol

The marketing environment for tobacco and alcohol products exerts a substantial influence on product use. Tobacco and alcohol products are subject to different marketing restrictions (see Appendix 9B) as well as different requirements for disclosure of marketing expenditures. Tobacco companies are required to regularly disclose annual sales and marketing expenditures for cigarettes, smokeless tobacco, and e-cigarettes to the FTC. No similar annual marketing expenditure disclosure requirement exists for alcohol companies.

The literature on cross-cue reactivity indicates that comarketing of tobacco and alcohol products may contribute to reinforcing tobacco and alcohol use. The enactment of the Family Smoking Prevention and Tobacco Control Act banned the sale of cigarettes in flavors other than menthol, including alcohol-flavored cigarettes, but this restriction did not extend to other tobacco products. As a result, tobacco companies continue to manufacture alcohol-flavored tobacco products, including cigars and e-cigarettes. Similarly, tobacco-flavored alcohol products are also available. In addition, tobacco advertising sometimes includes alcohol cues in the form of written or visual references to alcohol products. These explicit comarketing strategies may influence tobacco and alcohol use and co-use patterns.

The 1998 Master Settlement Agreement ended the use of paid tobacco brand placement in U.S. films. Nonetheless, unpaid tobacco brand placement continues, as does paid and unpaid alcohol brand placement in films, as well as in channels other than films, including music videos and television programs. Although federal law prohibits the advertising of cigarettes, little cigars, and smokeless tobacco on television and radio, alcohol advertising in these media channels is subject only to voluntary self-regulation. As a result, youth and adults are exposed to alcohol advertising in these and other media channels. In addition, depictions of tobacco use, alcohol use, and their co-use are common in various media channels, including television, movies, social media, and music videos, as well as in books including those read by adolescents. Depictions of tobacco and alcohol in films and other media are often framed in positive ways (e.g., romance, achievement, adventure) and only rarely show the harmful consequences of tobacco or alcohol use.

Tobacco marketing is causally associated with initiation and use of tobacco products by youth and young adults. Similarly, robust evidence supports a causal association between alcohol marketing and youth alcohol use.

Policy Factors

Policy approaches, such as taxation, warning labels, sales restrictions, and restrictions related to on- and off-premise use are applicable to both tobacco and alcohol but are often designed or implemented differently for the two types of products (see Table 9.1). Identifying cross-substance policy effects, such as how alcohol regulations may influence tobacco use, is critical for the development of cohesive and optimal regulatory systems that benefit public health.[284-287] For example, increasing taxes on alcohol may influence the consumption of tobacco and vice versa. In the United States, the National Institute on Alcohol Abuse and Alcoholism Alcohol Policy Information System is a key resource for obtaining information about state and federal laws related to alcohol and recreational use of cannabis.[288]

Table 9.1 Selected Federal Laws Pertaining to Tobacco and Alcohol

Relevant U.S. federal law (year passed)	Summary
Tobacco	
Federal excise taxes on tobacco[289-292] • Cigars, chewing tobacco, snuff: 1862 • Cigarettes: 1864 • Pipe tobacco: 1989 • Roll-your-own tobacco: 2000	Tax levied on tobacco products. Listed below are 2025 tobacco-specific federal excise taxes on the following tobacco products: • Small cigarettes and small cigars: $1.01 per pack of 20 • Large cigarettes: $2.11 per pack of 20 • Large cigars: 52.75% of sale price (cap: $0.4026 each) • Pipe tobacco: $0.1769 per 1 oz tin or pouch • Chewing tobacco: $0.0315 per 1 oz tin or pouch • Snuff: $0.0944 per 1 oz tin or pouch • Roll-your-own tobacco: $1.5488 per 1 oz tin or pouch
Synar Amendment (1992)[293]	Requires all U.S. states and territories to implement laws setting the minimum sale age for tobacco products to at least 18 in order to receive full Substance Use Prevention, Treatment, and Recovery Services Block Grant awards. Superseded in 2019 by Tobacco 21 legislation.
Family Smoking Prevention and Tobacco Control Act (FSPTCA) (2009)[206,294]	Provides FDA with authority to regulate the manufacture, marketing, sale, and distribution of tobacco products. including conducting public education campaigns, monitoring compliance with federal tobacco laws and regulations, establishing tobacco product standards, and conducting research to support regulation.
Preventing All Cigarette Trafficking (PACT) Act (2010)[80,295,296]	Requires distributors of cigarettes, ENDS, and other smokeless tobacco products who sell or advertise in interstate commerce to register with and report to the Bureau of Alcohol, Tobacco, Firearms, and Explosives; broadly bans mailing of these tobacco products through the U.S. Postal Service; requires age verification at the points of purchase and delivery
Tobacco 21 (2019)[297]	Raises the federal legal age for sale of tobacco products, including ENDS, from 18 to 21 years of age. Starting September 30, 2024, tobacco retailers are required to use photo identification to verify the age of purchasers <30 years old, and tobacco products may not be sold through vending machines in facilities where individuals <21 years old are present or permitted to enter.
Alcohol	
Federal excise taxes on alcoholic beverages (1791)[298]	Tax levied on alcohol products. Federal excise taxes on alcoholic beverages vary based on quantity of alcohol produced by a brewer, distilled spirits producer, or wine producers and the specific alcohol products. Listed below are approximations of 2025 federal excise taxes for alcoholic beverages for large producers: • Beer: $0.05 per 12 oz can or bottle ($0.09 per oz of pure alcohol content) • Wine: $0.04 per 5 oz glass ($0.06 per oz of pure alcohol content) • Spirits: $0.13 per 1.5 oz shot ($0.21 per oz of pure alcohol content)
18th Amendment to the U.S. Constitution (1919)[299]	Prohibited the production, transport, or sale of alcohol in the United States
21st Amendment to the U.S. Constitution (1933)[300,301]	Repealed the 18th Amendment, permitting the production, transport, and sale of alcohol in the United States and granting states power to regulate alcoholic beverages
National Minimum Drinking Age Act (1984)[302]	Requires states to prohibit the purchase or public possession of alcohol by anyone under 21 years of age as a condition of receiving state highway funds

Note. Discussion of some of these laws and policies is covered in chapter 8, including the Synar Amendment, the National Minimum Drinking Age Act, and Tobacco 21. oz = ounce; ENDS = electronic nicotine delivery system; FDA = U.S. Food and Drug Administration.

This section summarizes studies of the cross-substance impact of regulating alcohol products (e.g., tax, minimum price floors, alcohol content), their temporal availability (e.g., sales hours), physical availability (e.g., restrictions on retailer locations), affordability and acceptability (e.g., packaging and warning labels or marketing restrictions such as advertising and display bans) to understand whether and how alcohol policies affect tobacco use. Because bars and restaurants are both common on-premise settings for alcohol use and a target for smokefree policies, this section also considers how smokefree policies affect alcohol use in on-premise use venues. Finally, the impact of smokefree policies in alcohol and other substance use treatment settings on patients' use behavior is also discussed.

The World Health Organization's (WHO) "best buys" for reducing noncommunicable diseases specify different long-term objectives to reduce tobacco use and to reduce harmful alcohol use (i.e., alcohol use that causes detrimental health and social consequences, and alcohol use patterns associated with increased risk of adverse health outcomes).[303,304] WHO provides recommendations to reduce both tobacco and alcohol by increasing taxes (thereby reducing affordability) and restricting marketing (see Table 9.2). Notably, "best buys" to reduce retail availability are limited to alcohol and focus on reducing sales hours, based on cost-effectiveness analyses indicating a cost-effectiveness ratio of $100 or more per healthy life year gained. To reduce the harmful use of alcohol, WHO also recommends interventions to reduce retailer density, enact and enforce a minimum age of purchase or consumption, and restrict or ban promotions targeting young people. However, these interventions are not considered "best buys" due to the lack of cost-effectiveness evidence.[304] Overall, the WHO best buys do not directly address co-use of tobacco and alcohol, although the evidence summarized in this chapter suggests some of these policies could have cross-substance effects.

Table 9.2 World Health Organization's "Best Buys" for Tackling Noncommunicable Diseases

Reduce tobacco use	Reduce harmful alcohol use
• Increase excise taxes and prices on tobacco products. • Implement large graphic health warnings on all tobacco packages, accompanied by plain/standardized packaging.[a] • Enact and enforce comprehensive bans on tobacco advertising, promotion, and sponsorship.[a] • Eliminate exposure to secondhand tobacco smoke in all indoor workplaces, public places, and public transport.[a] • Implement effective mass media campaigns that educate the public about the harms of smoking/tobacco use and secondhand smoke and encourage behavior change.[a] • Provide cost-covered, effective, population-wide support (including brief advice, national toll-free quitline services, and text-message cessation programs) for tobacco cessation for all tobacco users.[b]	• Increase excise taxes on alcoholic beverages.[c] • Enact and enforce bans or comprehensive restrictions on exposure to alcohol advertising (across multiple types of media).[a] • Enact and enforce restrictions on the physical availability of retailed alcohol (via reduced hours of sale).

Note. "Best Buys" are defined as the most cost-effective and feasible interventions for implementation by countries in all settings, with an average cost-effectiveness ratio greater than or equal to $100 per healthy life years gained in low-income and lower middle-income countries. [a]Requires capacity for implementing and enforcing regulation and legislation. [b]Requires trained providers in sufficient numbers and an effective health system. [c]Should be combined with other price measures, such as bans on discounts or promotions.
Source: Adapted from World Health Organization 2024.[304]

Table 9.3 summarizes a subset of the Healthy People 2030 objectives for tobacco and a similar subset of the Healthy People 2030 targets for alcohol.[305-307] The Healthy People project, an initiative of the U.S. Department of Health and Human Services Office of Disease Prevention and Health Promotion, identifies public health priorities that will improve health and well-being in the United States. It includes priority areas specific to tobacco and alcohol use. The associated objectives and targets are updated every 10 years, with its first iteration released in 1980, for Healthy People 1990.[308] The Healthy People 2030 objectives target 11.3% prevalence of past-month tobacco use for adolescents and an even lower prevalence (6.3%) of past-month alcohol use for adolescents.[309,310] Healthy People 2030 also includes policy-related objectives that address tobacco use, such as increasing taxes on cigarettes, increasing Medicaid coverage of evidence-based tobacco cessation treatments, and increasing the number of states and territories that have smokefree policies that apply to all indoor areas of worksites, restaurants, and bars.

Table 9.3 Selected Healthy People 2030 Objectives for Tobacco and Alcohol

Healthy People 2030 objective	Most recent statistic[a]	Healthy People 2030 target
Tobacco (subset)		
Reduce the lung cancer death rate	29.5 lung cancer deaths per 100,000 population[b]	25.1 lung cancer deaths per 100,000 population[b]
Reduce current tobacco use in adults	20.2%[b]	17.4%[b]
Reduce current cigarette smoking in adults	10.1%[b]	6.1%[b]
Reduce the proportion of people who don't smoke but are exposed to secondhand smoke	24.1%[b]	17.4%[b]
Reduce current tobacco use in adolescents (grades 6–12)	23.0%	11.3%
Reduce current e-cigarette use in adolescents (grades 6–12)	13.1%	10.5%
Reduce current use of flavored tobacco products in adolescents (grades 6–12) who use tobacco	77.5%	73.2%
Increase abstinence from cigarette smoking among pregnant women	97.0%	95.7%
Increase Medicaid coverage of evidence-based treatment to help people quit using tobacco	22 states (not including Washington, DC)	51 states (including Washington, DC)
Increase the national average tax on cigarettes	$2.16	$2.60
Increase the proportion of smokefree homes	90.0%	92.9%
Increase the number of states, territories, and DC that prohibit smoking in worksites, restaurants, and bars	33 states (including Washinton, DC, and some territories	58 states (including Washington, DC, and territories)
Alcohol (subset)		
Reduce cirrhosis deaths	13.0 deaths per 100,000 population[b]	10.9 deaths per 100,000 population[b]
Reduce the proportion of people (aged 12 and older) who had alcohol use disorder in the past year	10.5%	9.9%

Table 9.3 (continued)

Healthy People 2030 objective	Most recent statistic[a]	Healthy People 2030 target
Reduce the proportion of adolescents (ages 12–17) who drank alcohol in the past month	6.8%	4.8%
Reduce the proportion of people aged 21 years and over who engaged in binge drinking in the past month	23.8%	22.7%
Reduce the proportion of people under 21 years (ages 12–20) who engaged in binge drinking in the past month	8.2%	6.0%
Increase abstinence from alcohol among pregnant women (ages 15–44)	90.3%	92.2%

[a]Most recent available statistics were accessed in April 2026. [b]Age adjusted to the year 2000 standard population.
Source: Adapted from Office of Disease Prevention and Health Promotion.[311]

How Alcohol Tax and Pricing Policies and the Affordability of Alcoholic Beverages Affect Tobacco Use

As evidence-based policy interventions, pricing policies that reduce product affordability are among the most effective strategies to reduce tobacco use and alcohol consumption.[312,313] Excise taxes are the most important component of pricing policies, which are specifically designed to target commercial products that harm human health (e.g., alcohol, tobacco, sugar-sweetened beverages, cannabis).[314,315] Excise taxes also generate revenues that can be spent on public goods and other government initiatives, providing incentives for policymakers to view excise taxes as a win-win strategy for both public health and finance. Therefore, excise taxes are generally considered to be policies that are both effective and cost-effective in advancing public health goals.[313,316,317]

The literatures investigating the impact of pricing policies on tobacco and alcohol use have largely developed in parallel, with most studies focusing on the estimation of the own-price elasticities of each product. However, several studies examine cross-substance effects of pricing policies, including how alcohol prices affect or are associated with cigarette smoking in the United States.[224,318-325] In this context, increasing alcohol prices could be associated with decreased cigarette smoking (i.e., cigarettes and alcohol are economic complements), increased cigarette smoking (i.e., cigarettes and alcohol are economic substitutes), or have no significant association with cigarette smoking (i.e., cigarettes and alcohol are independent).

A targeted literature search of Web of Science and PubMed identified 9 U.S.-based studies published since 2000 that examined cross-substance or cross-price effects between tobacco and alcohol and explicitly assessed the impacts of alcohol prices or taxes on cigarette smoking (Appendix 9C). Three studies analyzing state-level data or adult and household surveys concluded that increasing alcohol prices will reduce cigarette smoking (i.e., cigarettes and alcohol are economic complements). Using state-level data, Goel and colleagues (2016)[322] found that a 10% increase in beer taxes decreased the consumption of cigarettes by 0.3%–0.4%. Decker and Schwartz (2000)[320] found larger cross-price elasticities using individual data from the 1985 and 1993 Behavioral Risk Factor Surveillance System surveys. Their results suggest that a 10% increase in beer prices was associated with a 1.4% decrease in adult cigarette

consumption,[320] with the possibility that the long-run effect of alcohol price on cigarette consumption is greater than the short-run effect.[322] Koksal and Wohlgenant (2016)[323] analyzed 2002–2008 household consumer expenditure surveys and estimated that the long-run cross-price impact for alcohol on smoking could be as large as –0.723, suggesting that a 10% increase in alcohol prices will lead to a 7.2% decrease in cigarette smoking. In contrast to the studies conducted by Goel and colleagues (2016)[322] and Decker and Schwartz (2000)[320], which analyzed the effects of beer prices, this study[323] used a composite price index of several alcoholic beverages.

Three studies using macro or adult data have found that alcohol prices have no significant impact on cigarette smoking. Frye (2020)[321] conducted a hypothetical purchase task to assess the economic relationship between cigarettes and alcohol and found these two products to be independent. Using state-level sales data from 1980 to 2007, Bates and colleagues (2015)[319] found that beer taxes were not significantly associated with cigarette sales per capita. Petry (2001)[325] conducted a hypothetical purchase task among 53 adults who primarily use alcohol but also concurrently use multiple substances and found their cigarette purchasing was not responsive to alcohol prices. However, a limitation of this analysis, in so far as it provides cross-price elasticity estimates for cigarettes and alcohol, is that participants were not required to be people who smoke cigarettes; rather, participants were required to use at least three other substances (cannabis, opioids, cocaine, and/or sedatives) in addition to alcohol.

One volumetric choice experiment conducted in 2020–2021 examined the effect of alcohol prices (via tax increases) on cigarette and e-cigarette consumption among adults who reported past 30 day alcohol use and reported current cigarette smoking or having smoked within the previous 60 days, and starting to use e-cigarettes or reinitiating use in the past 30 days.[224] In this study, hypothetical increases in beer and liquor/wine prices did not have a significant effect on the hypothetical cigarette consumption. However, increasing alcohol prices had a significant effect on hypothetical e-cigarette consumption, with a 10% increase in beer prices raising hypothetical e-cigarette consumption by 1.25% and a 10% in liquor/wine prices increasing hypothetical e-cigarette consumption by 0.5%, suggesting that e-cigarettes and alcohol are economic substitutes among people who co-use cigarettes, e-cigarettes, and alcohol. However, this study included only people who started or re-initiated e-cigarette use within the past 30 days; further studies are needed to determine the relationship between alcohol prices and tobacco consumption at the population level.

Studies analyzing youth data are also inconclusive regarding how alcohol prices or taxes affect youth cigarette smoking. Markowitz and Tauras (2009)[324] examined the economic relationship between substances using panel data from 1997–2001 from the National Longitudinal Survey of Youth 1997 cohort. In that study, the cross-price effect for beer on cigarette smoking was nonsignificant or negative, depending on different model specifications.[324] Therefore, the authors could not draw conclusions on how beer pricing affects smoking. A separate study constructed a structural model of youth substance use using the National Longitudinal Survey of Youth 1979 cohort and found mixed results.[318] When a specified functional form was used for policy simulation, cigarettes and alcohol appeared to be substitutes and higher beer prices increased smoking participation. However, when conducting a survival analysis, beer prices had no significant impact on quitting duration.

Taken together, the U.S.-based studies find mixed evidence on the nature of the relationship between alcohol pricing policies and cigarette outcomes, likely due to different study designs and populations. The limited evidence from international studies of the effect of alcohol tax/price changes on tobacco use suggests a similar conclusion, although much of this evidence is based on cross-sectional data.[326-330] Nonetheless, evidence from Spain,[328] Australia,[331] Italy,[327] and Sweden[330] point to tobacco and alcohol being complements rather than substitutes, showing the potential of using alcohol taxes and prices to curb tobacco use. Importantly, given that the majority of U.S. studies indicate that cigarettes and alcohol are either independent or are economic complements (rather than substitutes), increasing alcohol taxes and prices is unlikely to increase cigarette smoking and may actually decrease cigarette smoking.

Finally, little research has addressed other tobacco products. Although it is encouraging that alcohol policies, such as beer taxes, have been controlled for in some studies that investigate e-cigarette use outcomes, these studies in general did not explicitly report the effects of alcohol policies on tobacco use and none were powered to examine such effects.[332-335]

Effects of Policies to Reduce Alcohol Availability on Tobacco Use

Studies have evaluated the effect of policies that reduce the availability of either tobacco or alcohol.[336,337] Given the large proportion of retailers that sell both tobacco and alcohol, cross-substance effects of policies to reduce availability of alcohol on tobacco use and co-use are plausible. However, no studies were identified that evaluated an ancillary impact of reducing alcohol availability on tobacco use (or vice versa).

Sales bans. Studies have considered the effects of sales bans, both real and hypothetical, on tobacco and alcohol consumption.[338] Several studies investigated the impact of hypothetical policies to restrict sales of tobacco to off-premise alcohol outlets (i.e., stores that sell alcohol for use off-site). In Scotland, such a policy was estimated to yield a 94.1% reduction in the number of tobacco retailers and a mean reduction of 85.9% in tobacco retail density, but would also be expected to exacerbate existing socioeconomic inequalities of exposure to tobacco retailers.[339] In New Zealand, such a policy was estimated to reduce the number of tobacco retailers by 83.4%, reduce density from 1 tobacco retailer per 115 adults (age 15 and older) who smoke cigarettes to 1 per 692, and dramatically reduce the presence of tobacco retailers near schools.[340] Although the existence of an integrated licensing scheme for tobacco and alcohol could be advantageous for enforcement of sales and marketing regulations, the study authors acknowledged that such a policy would undermine a public health goal of decoupling tobacco and alcohol sales in the retail environment.[340]

In New Zealand, modeling to inform tobacco endgame legislation (policies with a goal of phasing out tobacco sales and use) considered a strategy of permitting tobacco to be sold only in 50% of liquor stores and in no other locations.[341] The study estimated cigarette cost (pack price plus travel costs) and use prevalence relative to other tobacco retail reduction strategies. Compared to the other strategies considered, this hypothetical policy was associated with the highest pack costs and the lowest estimated smoking prevalence (9.1%), at 0.8 percentage points less than business-as-usual (9.9%) within 10 years. Similar modeling studies could be used to examine how limiting tobacco sales to off-premise alcohol retailers would affect tobacco use and co-use in the United States.

One study considered the impact of a hypothetical policy to achieve a dramatic reduction in tobacco retail availability by limiting tobacco sales to state-controlled liquor stores in the subset of U.S. states where these exist.[124] A potential positive consequence of a dramatic reduction in the large number of tobacco retailers was offset by concerns about potential negative consequences, including risk for relapse to drinking among people who smoke daily who also have a history of alcohol use disorder, especially for women and people with lower levels of education.[124] However, these concerns were based on secondary analyses of the subset of respondents to the 2014–2015 and 2019–2020 U.S. National Alcohol Surveys who lived in the 12 states with state-controlled alcohol stores. A second study exploring the idea of transitioning tobacco sales to state-controlled alcohol stores as a way to reduce tobacco product availability was published in 2024.[126] This study utilized interviews with 103 tobacco control advocates, public health officials, alcohol policy experts, and alcohol control system representatives from 10 states with alcoholic beverage control systems. The study identified potential benefits related to reduced access and exposure to advertising particularly among racial and/or ethnic minorities, but it also raised concerns about focusing on revenue generation, increasing power of the tobacco and alcohol industries, and creating difficulty for those in recovery. A third study on this topic, published in 2025, examined travel time to the nearest tobacco retailer in 10 states with state-controlled alcohol stores.[342] The authors concluded that transitioning tobacco sales to state-controlled stores would increase mean driving time from residents' homes to the nearest tobacco retailer by 119%, or 5.4 minutes. In seven states, estimated increases in travel time were larger for Black and/or Hispanic or Latino people (compared with White people) and people with incomes below the poverty line (compared with those at or above the poverty line).

During the COVID-19 lockdown period in 2020, South Africa enacted temporary (20 week) sales bans on nonessential items, including tobacco and alcohol, to limit the spread of COVID-19 and reduce the burden on the health care system. The tobacco and alcohol sales bans during this period were associated with reduced consumption of these products, despite the presence of illicit sales.[338,343-346] However, the evidence for reduced alcohol use in the context of South Africa's sales bans is limited to 2 studies, a cohort of 61 patients who were in treatment for tuberculosis[338] and a convenience sample of 798 participants in a Facebook survey,[345] and therefore not generalizable to the population level. In addition, the context of simultaneous bans made it difficult to isolate any effects of alcohol sales restrictions on tobacco use.

Restricting temporal availability. In contrast to tobacco control strategies, state and local alcohol policies often involve regulating temporal availability, either by limiting hours of sale or days of sale. Typically, these policies have been justified as needed to address public health and safety concerns associated with heavy alcohol consumption (e.g., risk of violent crime, alcohol-impaired driving, and unintentional injuries).[347,348] Additionally, some U.S. policies have their origins in religious reasons (e.g., to promote church attendance). In a systematic review, 19 of 24 studies evaluated the effects of increasing temporal availability (e.g., removing Sunday sales bans) on alcohol-related harms, with 10 of 19 studies finding that increases in days or hours of alcohol sales were associated with increases in alcohol-related harms, including motor vehicle accidents, casualties and fatalities, violent incidents, assaults, and other crime.[349] However, none of the studies assessed the impact of such laws on tobacco sales or use.[349]

Effects of Smokefree Bar/Restaurant Laws on Alcohol Use

This chapter primarily considers whether and how alcohol policy influences tobacco use. However, smokefree laws are often implemented in venues where alcohol is consumed, such as bars and restaurants, providing the opportunity to explore cross-substance policy effects. As briefly described here, research has evaluated the effect of indoor smokefree restaurant and bar laws and found cross-substance policy effects. Three prospective studies were identified. Linking state-level policy data to longitudinal data from the 2001–2002 and 2004–2005 National Epidemiologic Survey on Alcohol-Related Conditions (NESARC), one study observed that smokefree restaurant and bar laws were associated with a 6% higher probability of quitting smoking among men with an alcohol use disorder compared to those in states without such laws.[350] In a separate study using 2001–2002 and 2004–2005 NESARC data, protective effects of smokefree restaurant and bar laws (i.e., an association with lower risk of alcohol use disorder onset) were observed in the overall sample of people who reported drinking in public at least once per month in the past year and among all population subgroups except participants ages 40 or older.[351] In addition, smokefree restaurant and bar laws were associated with (1) greater likelihood of remission from alcohol use disorder overall among individuals who reported any alcohol use at baseline, as well as among men, young adults (ages 18–29), and adults ages 50 and older; and (2) greater likelihood of remission from an alcohol use disorder among the subset of participants who drank alcohol in public at least once per month.[351]

In a longitudinal analysis of the 2005, 2007, and 2008 four-country International Tobacco Control Survey (Australia, Canada, United Kingdom, and United States), neither overall changes in frequency and amount of alcohol consumption nor overall changes in frequency of binge drinking were associated with the implementation of smokefree bar laws.[352] However, among adults who reported hazardous drinking (>7/14 drinks per week among women/men and/or drinking >4/5 drinks within a 2-hour period among women/men) or heavy smoking (heaviness of smoking index score of 3 or greater), those who frequented bars that became or remained smokefree reported a significantly larger reduction in the amount of alcohol typically consumed compared to those who frequented bars that continued to allow smoking.

Cross-sectional studies have also examined the relationship between smokefree restaurant and bar laws and tobacco use. A cross-sectional analysis of data from the 2009 U.S. National Health Interview Survey linked with state-level policy data found that stronger smokefree bar laws were associated with lower odds of current smoking among all adults and those who reported alcohol use, regardless of subgroup (e.g., any past-month use, any binge drinking).[353] A 2020–2021 volumetric choice experiment survey of 181 adults who reported use of cigarettes, e-cigarettes, and alcohol found that hypothetical bans on smoking or vaping in bars and restaurants would have no significant effect on alcohol consumption.[224] However, more longitudinal cohort studies are needed to evaluate the cross-substance effects of smokefree bar/restaurant policies on alcohol use as well as co-use of tobacco and alcohol.

Effects of Smokefree Policies in Substance Use Treatment Settings on Tobacco and Alcohol Use

Substance use treatment settings are another important setting in which to consider cross-substance policy effects on tobacco and alcohol use, as many people who seek substance use treatment for alcohol and/or other drugs also use tobacco products.[354] There remains a misconception that people with substance use disorders (SUDs) (and other behavioral health

conditions) do not want to quit tobacco use, are not able to quit tobacco use, and that smokefree policies in SUD treatment facilities, as well as providing treatment for tobacco use while in treatment, might undermine successful treatment for SUD.[2] As discussed in chapter 7, incorporating smoking cessation treatment into alcohol use treatment does not adversely affect the success of the alcohol use treatment. Furthermore, smokefree policies are effective in increasing smoking cessation, including among individuals in substance use treatment.[2,355-357] Notably, "the tobacco industry has opposed smokefree policies in psychiatric hospitals, donated cigarettes to mental health facilities, and funded research suggesting that patients with psychiatric illnesses need tobacco for self-medication".[2,p.602] Furthermore, "in the past, many behavioral health clinicians believed that treating nicotine dependence and tobacco cessation jeopardize sobriety or mental health recovery… a misconception that has been actively fostered by the tobacco industry".[2,p.602] However, efforts to integrate behavioral health care with primary health care, along with the recognition that people dealing with behavioral health conditions are disproportionately likely to prematurely die of a disease caused by smoking, have led to policy changes at substance use treatment settings and at the state level.[2] As of February 2026, 18 states require tobacco-free grounds for most mental health facilities, and 19 states require tobacco-free grounds for most substance use treatment facilities.[358] In the 2024 National Substance Use and Mental Health Services Survey, 36.7% of 15,919 U.S. substance use treatment facilities reported 100% smokefree grounds policies (smoking policies in which smoking is not permitted anywhere outside or within any building).[359] More than one-half of substance use treatment facilities (57.6%) had policies that banned smoking indoors but permitted smoking in designated outdoor areas.[359] In addition, as of September 2024, nine states require that substance use treatment facilities include treatment for tobacco use.[360] In 2024, a majority of substance use treatment facilities reported screening for tobacco use (83.9%) and providing smoking/tobacco cessation counseling (72.6%), while a smaller number reported providing nicotine replacement therapy (43.2%) or non-nicotine cessation medications (39.1%).[361]

A 2022 systematic review concluded that, although smokefree policies alone in mental health and substance use treatment settings do not affect smoking cessation outcomes, the use of smokefree policies in combination with multicomponent interventions and continued support after discharge does support cessation.[362] However, studies primarily represented mental health treatment facilities and included only a small number of studies conducted in substance use treatment facilities. Further, this review focused only on post-discharge smoking cessation outcomes and changes in smoking behaviors post-discharge.

Implementation of tobacco-free policies in substance use treatment facilities has been associated with increased positive beliefs among staff about treating tobacco use,[363] increased tobacco use assessment,[364-369] and increased provision of tobacco treatment services.[355,356,363,365,367,370-374] Most studies have observed decreased cigarettes smoked per day[355,373,375] or decreased smoking prevalence[355-357,363,372,373,375,376] among clients and/or staff after implementation of tobacco-free policies. In New York, for example, there was a small but significant decrease in client smoking prevalence from 69.4% to 62.8% 1 year after the implementation of a statewide tobacco-free services regulation in state-certified addiction treatment programs,[377] although this change was not sustained 5 years after implementation.[378] In California, a state-sponsored initiative to promote tobacco-free grounds and increase smoking cessation services in residential SUD treatment programs was associated with a 27.6 percentage point reduction in smoking prevalence in the first cohort of participating programs (preintervention: 54.2%, 15 months

post-intervention: 26.6%, adjusted odds ratio [aOR] = 0.25, 95% confidence interval [CI] = 0.13–0.45)[372] and a 19.8 percentage point reduction in smoking prevalence in a replication cohort (preintervention: 60.3%, 18 months post-intervention: 40.5%, aOR = 0.46, 95% CI = 0.27–0.78).[356] However, implementation was inconsistent, with only 8 out of 11 participating programs in the first cohort adopting policies.

No studies were identified that reported the effects of smokefree policies in substance use treatment facilities specifically on alcohol use outcomes. However, most studies have found that smokefree policies are associated with no change in patient census, admissions, irregular discharges, or treatment completion,[357,364,369,379-381] indicating that smokefree policies do not appear to deter people from receiving or completing treatment for substance use.

Effects of Pictorial Warning Labels on Knowledge and Risk Perceptions of Tobacco and Alcohol

A strong evidence base exists for the effectiveness of large pictorial warning messages on tobacco products. The National Cancer Institute's 2016 Tobacco Control Monograph 21, *The Economics of Tobacco and Tobacco Control,* concluded that "large pictorial health warning labels on tobacco packages are effective in increasing smokers' knowledge, stimulating their interest in quitting, and reducing smoking prevalence."[313,p.303] The 2024 Surgeon General's report, *Eliminating Tobacco-Related Disease and Death: Addressing Disparities*, states that "requiring pictorial health warnings on cigarette packages and advertisements increases both understanding of the harms of smoking and intention to quit among different population groups,"[139,p.589] and the 2020 Surgeon General's report concluded that "the evidence is sufficient to infer that large, pictorial health warnings increase smokers' knowledge about the health harms of smoking, interest in quitting, and quit attempts and decrease smoking prevalence."[2,p.12] The 2009 Family Smoking Prevention and Tobacco Control Act directed the FDA to "issue regulations requiring color graphics depicting the negative health consequences of smoking" to accompany new textual warning statements and require cigarette packages and advertisements to include new warnings.[206,382] As of 2026, the pictorial warning labels proposed by FDA remain the subject of litigation.[382]

Warning labels for tobacco products, including pictorial warning labels, are also recommended by the guidelines for implementation of Article 11 of WHO's Framework Convention on Tobacco Control (an international treaty that aims to reduce demand for and supply of tobacco products).[383] According to WHO, health warnings on alcoholic beverages also increase awareness of the risks associated with drinking, but the evidence is less robust for whether health warnings reduce alcohol consumption or change behavior.[384] The discrepant findings may be due in part to the more limited implementation of pictorial warnings for alcohol than for tobacco. Indeed, at least three times as many countries mandate pictorial warning labels for tobacco than for alcohol. By 2021, 134 countries/jurisdictions mandated pictorial cigarette warnings covering more than half the pack[385] (see example warnings in Figure 9.8, Panel A). A total of 55 countries reported using alcohol warning labels in 2019,[386] but pictorial warnings are not widely adopted.[387,388] As is true for pictorial warning labels for cigarettes, exposure to pictorial warning labels for alcohol is associated with greater awareness of the health harms of alcohol use, including cancer.[389,390]

In the United States, inconspicuous text-only alcohol warning labels caution about the risk of alcohol use during pregnancy and the dangers of drinking and driving. The 2024 Surgeon General's Advisory, *Alcohol and Cancer Risk*, recommends "an update to the Surgeon General's warning label for alcohol-containing beverages to include a cancer risk warning."[391,p.17] This advisory also recognizes that "prominent, pictorial, and rotating messages, compared to less conspicuous, text-only, and static messages, are more effective for influencing awareness and behavior."[391,p.17] A study using 2024 data from the Health Information Trends National Survey found substantial support for warning labels in the United States, with 61.6% of respondents expressing support for cancer-related warning labels and 29.6% neither supporting nor opposing labels.[392]

In 2025, the Alaska state legislature passed a bill that included a provision to require warning signs where alcohol is sold indicating that "alcohol use can cause cancer, including breast and colon cancers."[393,394] The requirement went into effect for bars, restaurants, and liquor stores on August 1, 2025. Legislation passed in Ireland requires all alcohol products sold to include a warning reading "There is a direct link between alcohol and fatal cancers" starting in May 2026.[395,396]

In Yukon, Canada, researchers examined the effects of alcohol warning labels featuring a cancer warning, national drinking guidelines, or standard drink information (see Figure 9.8, Panel B).[397] In a quasi-experimental study that compared participants in an alcohol warning label-exposed intervention site to an unexposed control site over multiple timepoints, intervention-site participants were significantly more likely than control-site participants to recall and support the standard drink warning label. Intervention participants were also significantly less likely than control-site participants to report intentions to use the standard drink warning label information to purchase higher strength alcohol for a lower cost (thereby avoiding a potential unintended consequence of this intervention). An interrupted time-series analysis of per-capita alcohol sales in Yukon and in neighboring regions where alcohol warning labels did not change found decreases in alcohol sales in the intervention site of Yukon relative to the comparison sites.[398]

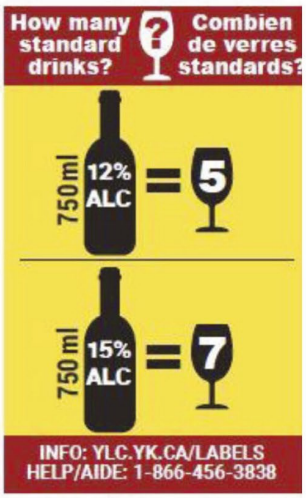
Figure 9.8 Examples of Cigarette and Alcohol Warning Labels in Canada

Panel A: Example Cigarette Warning Labels in Canada, 2023–Present

Panel B: Alcohol Warning Labels in Yukon, Canada, 2017–2018

Label 1 - Cancer Warning

Label 2 - Canada's National Drinking Guidelines



Label 3 - Standard Drink Information (example for wine)

Note. For additional examples of pictorial health warnings, visit the World Health Organization's pictorial health warnings on tobacco products database.

Source: Health Canada 2025,[399,400] Schoueri-Mychasiw et al. 2021.[397] Cigarette warning labels reproduced with the permission of the Minister of Health (Canada), 2025. Alcohol warning labels reproduced with permission of the National Cancer Institute, from "Use as directed: do standard drink labels on alcohol containers help consumers drink (ir)responsibly? Real-world evidence from a quasi-experimental study in Yukon, Canada," Schoueri-Mychasiw et al., *Drug and Alcohol Review*, volume 40, issue 2, Copyright © 2020 Her Majesty the Queen in Right of Canada. Drug and Alcohol Review © 2020 Australasian Professional Society on Alcohol and other Drugs; permission conveyed through Copyright Clearance Center, Inc.

One online experiment made a cross-substance comparison, exposing adult participants to a warning label on a hypothetical brand of alcohol, cigarettes, and sugar-sweetened beverages in random order.[401] Across all substances, participants were randomly assigned to view (1) text-only warnings without efficacy information to increase consumers' confidence in their ability to stop using the product, (2) text-only warnings with efficacy information, (3) pictorial warnings without efficacy information, or (4) pictorial warnings with efficacy information. Compared to text-only warnings, pictorial warnings led to greater fear, more thinking about the harms of consumption, and lower product appeal for the product in the warning. This was true across all three product categories, suggesting that the evidence base supporting cigarette pictorial warnings is relevant to alcohol warning labels.[401]

In addition to pictorial warning labels, the use of "plain packaging" has been explored for tobacco and alcohol.[2,402] Plain packaging, or standardized packaging, does not have additional logos, fonts, colors, images, or text that one might associate with a specific brand. Instead, product type is identified in a fixed font, size, and location on packaging that is otherwise identical across different tobacco or alcohol products. Plain packaging for tobacco reduces the appeal of tobacco products, makes health warnings on packages more salient and effective, limits misunderstandings of tobacco harms, and may decrease tobacco use.[2,313] As of 2023, 25 countries/territories had adopted plain packaging for cigarettes, and 14 countries/territories were considering its implementation.[403] As of 2026, plain packaging for alcohol has not been adopted in any country, and research on its potential effects is therefore limited. One online experiment comparing alcohol plain packaging with branded alcohol packaging found that plain packaging decreased ratings of product-based characteristics (e.g., attractiveness, popularity) and consumer-based characteristics (e.g., a consumer of this product is trendy, successful), but it did not significantly affect ratings of how boring the bottle was.[402]

Effects of Tobacco Display Bans on Alcohol Use

Restrictions on retail displays of tobacco products are another strategy to limit exposure to tobacco marketing.[3] A database of tobacco control laws from 212 countries compiled by Campaign for Tobacco-Free Kids found that 37 countries, as of 2026, have adopted total bans on retail displays of tobacco products and 49 countries either have adopted some restrictions on retail displays or have adopted bans that allow exemptions for tobacco displays, such as in duty-free stores or tobacco specialty shops.[404] In the United States, where retail tobacco displays are not restricted, a tobacco "powerwall", a prominent display of many cigarette and other tobacco products, typically dominates the retail space behind the cash register, and warning statements are often not visible to retail customers.[405,406] A study of point-of-sale marketing in California in 2002 found that retailers displayed an average of 124 cigarette pack facings on the front row of stores shelves and displays.[407] The literature search conducted for this chapter identified 26 studies of tobacco display bans.[408-433] After a tobacco display ban was implemented in England in 2015, children ages 11–15 had reduced odds of exposure to cigarettes in stores, regardless of alcohol use status (ever alcohol use: aOR 0.59, 95% CI = 0.47–0.75; never alcohol use: aOR 0.57, 95% CI = 0.48–0.67).[424] However, none of the identified studies of tobacco display bans evaluated effects on alcohol perceptions or use.

Summary: Policy Factors

Both tobacco and alcohol products are subject to federal, state, and local regulations on their use, sale, purchase, promotion, taxation, marketing, and temporal and geographic retail availability. Findings on cross-substance effects of alcohol taxation suggest that for adults, increasing alcohol prices either decreases cigarette smoking or has no discernable effect on cigarette smoking. This suggests that these products are economic complements and not economic substitutes, and that increasing alcohol taxes and prices is unlikely to increase cigarette smoking. However, the evidence for cross-substance effects of alcohol taxation were less clear among youth, a particularly price-sensitive population. Researchers have examined restrictions on the temporal or geographic sale or use of alcohol on cigarette smoking largely through hypothetical modeling or relatively unique sample populations. However, research has shown that implementation of smokefree policies in bars/restaurants is associated with decreased alcohol use. Further, implementation of smokefree policies in SUD treatment facilities does not interfere with SUD treatment outcomes and may support smoking cessation, especially when paired with smoking cessation treatment. Pictorial warnings, plain packaging, and retail display restrictions are effective tobacco control policies, and some evidence shows the effectiveness of these policies to address alcohol use; however, little to no research exists on their cross-substance effects. Understanding the effects of substance-specific policies on both tobacco and alcohol use is an important area for future research.

Chapter Summary

This chapter underscores important commonalities in the contextual factors influencing tobacco and alcohol use. These include product diversity, use of appealing flavors, likeness to other consumer products, and extensive marketing in various venues, such as brick and mortar stores, as well as several media channels, such as social media. The availability, accessibility and marketing of tobacco products have significant effects on tobacco use, and the availability, accessibility and marketing of alcohol products have significant effects on alcohol use, clearly indicating the need to consider cross-substance effects.

Both tobacco and alcohol products are obtained primarily from brick-and-mortar retailers. Marketing of both products in shared retail environments can facilitate exposure to marketing for both products that may normalize co-use and purchase of both products. Tobacco and alcohol retail availability are both associated with higher rates of tobacco and alcohol use and some evidence suggests that limiting retail availability can increase smoking cessation or decrease alcohol consumption.

Similar to the retail environment, the marketing environment for tobacco and alcohol products exerts a substantial influence on product use. The literature on cross-cue reactivity indicates that comarketing of tobacco and alcohol products may reinforce tobacco and alcohol use. Tobacco advertising sometimes includes alcohol cues in the form of alcohol imagery and written references to alcohol products. Further, tobacco companies continue to market alcohol-flavored tobacco products, including cigars, e-cigarettes, and nicotine pouches, and conversely, alcohol companies market tobacco-flavored alcohol products. Youth and adults are exposed to tobacco

and alcohol advertising and depictions of tobacco and alcohol use in various media channels including television, movies, social media, and music videos, which are often framed in positive ways (e.g., romance, achievement, adventure). Tobacco marketing is causally associated with initiation and use of tobacco products by youth and young adults. Similarly, robust evidence supports a causal association between alcohol marketing and youth alcohol use.

Both tobacco and alcohol products are subject to federal, state, and local policies and regulations. These policies address their use, sale, purchase, promotion, taxation, marketing, and temporal and geographic retail availability. However, few types of policies have been studied in the cross-substance context. With regard to taxation, research suggests that increasing alcohol prices either decreases cigarette smoking or has no effect on cigarette smoking, indicating that cigarettes and alcohol are economic complements, rather than economic substitute, and that increasing alcohol taxes and prices is unlikely to increase cigarette smoking. With regard to smokefree policies, implementation of smokefree policies in bars/restaurants is associated with decreased alcohol use. Further, implementation of smokefree policies in substance use treatment facilities does not interfere with substance use treatment outcomes and may support smoking cessation, especially when paired with smoking cessation treatment. Studies of the cross-substance effects of other policies, such as pictorial warnings, plain packaging, and retail display restrictions, are needed.

The existing literature on policies related to tobacco and alcohol predominantly focuses on two products: beer and cigarettes. This focus likely reflects prevalence of use for each of these substances. Despite consistent declines in prevalence of cigarette smoking, cigarettes remain the most used tobacco product among adults in the United States, with 9.9% of U.S. adults reporting current cigarette smoking in 2024.[434] The declines in cigarette smoking are somewhat offset by the increasing prevalence of e-cigarette use, up to 7.0% of U.S. adults in 2024.[46] Patterns in alcoholic beverage consumption have also changed. Since 1977, beer had been the alcoholic beverage with the highest per capita ethanol consumption in the United States.[435] However, in 2022, spirits surpassed beer to become the alcoholic beverage with the highest per capita ethanol consumption.[435] Thus, the last several decades reflect significant changes in the types of tobacco and alcohol products available in the market as well as shifts in the relative prevalence of competing products. New products (e.g., ready-to-drink hard seltzers, e-cigarettes, and nicotine pouches) continue to be developed, manufactured and marketed with flavors that are attractive to youth (including alcohol-flavored tobacco products).

This chapter's review of how alcohol availability, marketing, and policies affect tobacco consumption in the United States underscores many important research gaps. An overarching theme is evidence of many parallels in tobacco and alcohol research, but too few studies have examined the effects of alcohol environmental and policy factors on tobacco use. It is theoretically possible that reducing alcohol affordability, reducing alcohol availability, and restricting alcohol marketing have the potential to reduce tobacco use as well as co-use of tobacco and alcohol. However, few studies are designed to test cross-substance impacts of alcohol availability, marketing, or policies on tobacco use and co-use of tobacco and alcohol. Instead, many studies test how alcohol availability, marketing, and regulations affect alcohol consumption (e.g., how alcohol availability and price affect drinking), without considering potential effects on tobacco use. The parallel findings for alcohol policy and tobacco control research suggest that cross-substance policy impact research is feasible.

Research Needs

Systems approaches are needed to conduct additional research on the cross-substance effects of alcohol-related economic, environmental, and policy factors on tobacco use. Systems approaches explicitly incorporate efforts to understand how factors from multiple levels and domains interact to influence outcomes of interest. Systems approaches require collaboration across different disciplines and are particularly important where individual, social, and economic factors interact. For example, tobacco researchers could collaborate with alcohol policy researchers in geospatial studies of outlet density, and alcohol researchers may harness tobacco control experience in social media studies of marketing effects and in industry document research. In addition, a comparative review of place-based disparities in the retail environment for tobacco and alcohol could inform strategies to reduce their availability and help to inform environmental-related health disparities. Leveraging these collaborations could advance both tobacco and alcohol research fields and produce a unified tobacco-alcohol research agenda.

Further economic studies of patterns of aggregate consumption (e.g., total spending on tobacco and alcohol products) in combination with individual-level cross-sectional studies of use, and longitudinal studies of use over time, are needed to understand how tobacco and alcohol use may be affected by shared influences. To the extent that alcohol consumption and cigarette smoking share many of the same risk and protective factors, an integrative model addressing both tobacco and alcohol use could illustrate connections between individual and macro-level causes of their use as well as racial and/or ethnic and social and/or economic differences in use. A systems perspective may also prove useful to motivate and guide the design of such analyses and modeling. For instance, causal loop modeling could help identify whether and how the effects of alcohol availability, marketing, price, and policies influence tobacco susceptibility, initiation, use, and cessation.[436]

More theoretical and theoretically-informed research on how alcohol marketing may influence tobacco use and vice versa is needed. Several psychological theories can inform understanding of the impact of exposure to marketing and media portrayals of substance use on tobacco or alcohol use by youth and young adults, including social learning and social cognitive theories and the message interpretation processing model.[98-100] In addition, a social ecological framework underlies much of the research on the effects of retail availability on tobacco and alcohol use. Further research is needed to understand how aggregate social forces influence contextual factors (such as retailers' locations) and how these and other environmental characteristics influence how and where individuals use tobacco and alcohol. One challenge to studying the cross-substance effects of exposure to tobacco and alcohol marketing on tobacco and alcohol use is to disentangle the separate and combined effects of exposure on susceptibility, initiation, use, and cessation. In addition, comparative studies of responses to tobacco and alcohol marketing are needed to keep pace with the rapidly changing landscape for tobacco products and social norms about their use,[280,281] as well as the changing landscape for marketing with the advent of algorithmically targeted programmatic advertising.[282,283]

More research to ascertain whether higher alcohol taxes or prices reduce or increase tobacco consumption is needed, including attention to study design and quality. The majority of studies find that cigarette consumption and alcohol are independent or are economic complements, rather than economic substitutes. This suggests that increasing alcohol taxes and prices is unlikely to influence cigarette smoking. Additional research could lead to a high return on investment by informing policy design related to pricing and taxation applicable to multiple substances, including within product classes. Analyses of the variety and development of tobacco and alcohol products is needed. Overall, these studies could help address the important question of how alcohol-related economic factors affect tobacco use.

Additional research is needed to better understand the effects of retail availability on co-use of tobacco and alcohol. Multiple questions are not fully answered in the literature, such as (1) Is greater retail availability of alcohol products associated with increased tobacco use and co-use? (2) Do online sales/delivery of alcohol influence tobacco use (e.g., increase frequency of tobacco use and/or inhibit cessation)? (3) What are the determinants and characteristics of the locations of retail outlets selling both tobacco and alcohol? An important gap in the data needed to address these cross-substance retail research questions is the actual number and location of tobacco retail establishments that sell alcohol. Further research on tobacco-free pharmacy policies, such as CVS' national policy of eliminating tobacco sales, could inform an understanding of cross-substance effects of tobacco and alcohol retail availability. In addition, future research that utilizes hypothetical purchase tasks or other experimental methods may be useful to assess behavioral responses to sales restrictions that limit tobacco sales to liquor stores.

Further analysis and systematic review of the spatial distribution of tobacco and alcohol retailers in relation to neighborhood demographics (e.g., socioeconomic status and race and/or ethnicity) and individual behavior (e.g., initiation and cessation for alcohol and tobacco) are needed. Although there is rich literature on the retail availability of tobacco and alcohol, their impacts on behaviors and health disparities are understudied, especially in the cross-substance context. For example, studies of the association between retail availability and use that investigate the moderating effects of sociodemographic characteristics are needed to understand subgroup differences, including potential tobacco and alcohol disparities by neighborhood characteristics. Further research is needed to tease apart the detailed causal pathways involved in the association between tobacco and alcohol retail outlet density and neighborhood characteristics. This work could benefit from greater application of multilevel models addressing state, county, and individual characteristics as well as more research on factors influencing choices by companies and individuals concerning store locations.

The assessment of environmental factors further calls for research on the place-based pricing of alcohol along with comparisons to existing information on tobacco pricing both within and between retail outlets.[173,179] Such evidence could better inform an understanding of environmental disparities in the price of tobacco and alcohol and inform recommendations for policies to regulate the retail environment, including restrictions on licensing, retail availability, temporal availability, discounts, and minimum price.

Studies of cross-substance effects of tobacco and alcohol policies (in addition to taxation and pricing) on tobacco and alcohol use or co-use are lacking. With regard to packaging, further research is needed to investigate the effects of plain packaging of alcohol products on perceptions of tobacco and alcohol, tobacco and alcohol purchasing, and consumption behaviors. With regard to tobacco display bans, no studies examined the effects on alcohol use. However, studies about tobacco display bans may have collected data on alcohol use, which would present opportunities to investigate cross-substance effects of such policies on alcohol use with secondary analyses of existing data.

Both secondary data analyses and original research that investigate the cross-substance impacts of alcohol availability, affordability, marketing and counter-marketing, and alcohol policies (e.g., pricing) on tobacco use and co-use of tobacco and alcohol are needed to understand how alcohol policies could complement existing efforts to reduce tobacco use. Data sources that could be leveraged include proprietary sales data about tobacco and alcohol, particularly market basket data that characterize which products are purchased by whom. Future research could continue to leverage ongoing natural experiments that alter the retail landscape for tobacco and alcohol. For example, when Maryland amended its smokefree air law in 2024 to prohibit vaping in certain indoor areas, it required a study about issuing alcohol licenses to tobacconists and prohibited the issuance of alcoholic beverage licenses to tobacconists for the following two years, until July 1, 2026.[437] Also of interest is whether and how the expansion of off-premise and on-premise alcohol outlets that follows privatization may affect tobacco use in addition to alcohol use.[438] Tobacco and alcohol companies operate in overlapping marketplaces and often market their products in the same retail establishments, in the same media channels and market their products in similar ways, such as targeting specific populations, and attempting to limit tax increases and other policies that reduce the consumption of their products. Recognizing these commonalities could help generate research that captures the potential cross-substance impacts of policies. Leveraging these strengths could help further unify a tobacco-alcohol research agenda.

More research is needed to understand the effect of smokefree policies on smoking and alcohol co-use. More longitudinal cohort studies are needed to evaluate the cross-substance effects of smokefree bar/restaurant policies on alcohol use as well as co-use of tobacco and alcohol. In substance use treatment settings, smokefree policies have been associated with smoking cessation and existing evidence indicates that these policies do not harm alcohol (or other substance use) treatment outcomes, though more research is needed to examine these effects. Further research exploring the specific effects of smokefree policies within treatment settings on tobacco and alcohol use outcomes, and identification of necessary support (e.g., behavioral therapies, continued nicotine replacement therapy provision) would provide valuable insights. Additional research examining the effects of smokefree policies on smoking cessation during and after alcohol use treatment specifically is also warranted.

In sum, there are many parallels in tobacco and alcohol research, but more studies are needed to understand the cross-substance impact of alcohol economic, environmental, and policy factors on tobacco use.

Conclusions

1. **Co-exposure to tobacco and alcohol marketing and product availability is common, suggesting the potential for cross-product effects.** There is substantial spending by the tobacco and alcohol industries on marketing and advertising, and research has documented tobacco and alcohol co-marketing and media placement, with high levels of exposure reported by young people and by both youth and adults who co-use tobacco and alcohol.
2. **Both tobacco and alcohol retail outlets tend to be concentrated in neighborhoods characterized by greater socioeconomic disadvantage.** These findings exacerbate the adverse effects of geographic segmentation by race and/or ethnicity, socioeconomic disadvantage, and poor environmental conditions on health.
3. **Greater retail availability of tobacco is associated with greater tobacco use among adults, and some evidence shows that greater availability of alcohol is associated with greater alcohol use among adults.** Evidence that greater retail availability of tobacco and alcohol is associated with increased use of those products among youth is less consistent. No studies as of 2026 have considered whether greater availability of alcohol is associated with increased tobacco use among youth or adults.
4. **Smokefree bar laws are associated with cross-substance effects.** Evidence from a small body of literature finds that smokefree laws are associated with reductions in risk for alcohol use disorder and less frequent alcohol use among people who smoke heavily and report hazardous alcohol use.
5. **Smokefree policies in mental health and substance use treatment settings do not undermine effectiveness of substance use treatment.** Further, these policies can aid in smoking cessation, especially when paired with evidence-based smoking cessation treatment.

Appendix 9A Literature Search Strategy

To gather evidence pertaining to the economic, environmental, and policy factors that could provide insights into the potential effects of alcohol policies on tobacco, customized search strings were developed for PubMed and Web of Science. Product search terms included [“tobacco” OR “smoke*” OR “cigar*” OR “e-cigarette*” OR “electronic cigarette*” OR “electronic nicotine delivery*” OR “electronic nicotine device*” OR “vape” OR “vaping” OR “e-liquid” OR “hookah” OR “water pipe” OR “nicotine pouch*” OR “smokeless tobacco” OR “snus” OR “snuff” OR “loose leaf” OR “dissolvable tobacco”] AND [“drink*” OR “beer*” OR “wine*” OR “spirit*” OR “alcohol*” OR “liquor*” OR “ethanol*”]. The literature search was conducted between August and December 2023, focusing on English-language literature published at any time, and prioritizing studies published since the Master Settlement Agreement in 1998, with a few exceptions. In this chapter, summaries of evidence also prioritized systematic reviews and meta-analyses and more recent evidence not included in those publications. Additional studies published after 2023 were incorporated into the chapter based on their relevance.

Appendix 9B Marketing Restrictions for Tobacco and Alcohol Products

Restriction	Description of tobacco marketing restrictions[a]	Relevant federal regulation(s) for tobacco[b]	Description of alcohol marketing restrictions[c]	Relevant federal regulation or self-regulatory codes[b] for alcohol
Advertising (direct)				
Television and radio	*Federal-level*: Television and radio advertising is prohibited for cigarettes, little cigars, and smokeless tobacco. *State-level*: States may enact restrictions regarding place and manner of advertising.	PHCSA 1969; LCA 1973; CSTHEA 1986	*Federal-level*: Television and radio advertising allowed. *Voluntary self-regulation*: Self-regulatory codes require that more than 70% of the audience for advertisements (71.6% for spirits; 73.8% for wine and beer advertisements) be expected to consist of people ages 21 or older. *State-level*: States may enact restrictions regarding place and manner of advertising.	BI, DISCUS, WI codes
Magazines and newspapers	*Federal-level*: Cigarette and smokeless tobacco companies are prohibited from targeting youth in advertising, which includes advertising in magazines with large youth readerships. *State-level*: States may enact restrictions regarding place and manner of advertising.	MSA; STMSA	*Federal-level*: Magazine and newspaper advertising allowed. *Voluntary self-regulation*: Self-regulatory codes require that more than 70% of the audience for advertisements (71.6% for spirits; 73.8% for wine and beer advertisements) be expected to consist of people ages 21 or older. *State-level*: States may enact restrictions regarding place and manner of advertising.	BI, DISCUS, WI codes
Outdoor and transit	*Federal-level*: Billboards and transit advertisements for cigarettes and smokeless tobacco are prohibited. Outdoor advertising in certain locations (retail outlets, events sponsored by the tobacco industry) is allowed. *State-level*: States may enact restrictions regarding place and manner of advertising.	MSA; STMSA	*Federal-level*: Outdoor and transit advertising allowed. *Voluntary self-regulation:* Self-regulatory codes require that outdoor stationary advertisements be placed no closer than 500 feet from any elementary or secondary school or place of worship, except on a licensed premise. The Beer Institute's code further restricts advertising within 500 feet of public playgrounds, fields, or sports facilities intended primarily for youth athletics. *State-level*: States may enact restrictions regarding place and manner of advertising.	BI, DISCUS, WI codes
Direct mail	*Federal-level*: Direct mail advertising allowed. *State-level*: States may enact restrictions regarding place and manner of advertising.	FSPTCA	Direct mail advertising allowed. *State-level*: States may enact restrictions regarding place and manner of advertising.	
Point-of-sale advertising and displays	*Federal-level*: Point-of-sale advertising and displays allowed. *State-level*: States may enact restrictions regarding point-of-sale advertising and displays.	FSPTCA	*Federal-level*: Point-of-sale advertising and displays allowed. *State-level*: States may enact restrictions regarding point-of-sale advertising and displays.	

Appendix 9B (continued)

Restriction	Description of tobacco marketing restrictions[a]	Relevant federal regulation(s) for tobacco[b]	Description of alcohol marketing restrictions[c]	Relevant federal regulation or self-regulatory codes[b] for alcohol
Internet and social media	*Federal-level*: Social media posts must include required health warnings. Paid posts by bloggers or social media influencers must be disclosed. *State-level*: States may enact restrictions regarding place and manner of advertising. *Social media platform policies*: Platforms may enact policies that prohibit paid advertising for tobacco products or prohibit sponsored content promoting tobacco.	FSPTCA; FDA rule (21 CFR 1100, 21 CFR 1140, 21 CFR 1143); FTC Endorsement Guides (16 CFR 255)	*Federal-level*: Posts made by paid or otherwise compensated users are considered advertisements and must contain mandatory statements applied to other forms of alcohol advertising. *Voluntary self-regulation*: Self-regulatory codes require that more than 70% of the audience for advertisements (71.6% for spirits; 73.8% for wine and beer advertisements) be expected to consist of people ages 21 or older. Self-regulatory codes also require associated webpages to have individuals to verify that they are 21 years old or older. *State-level*: States may enact additional restrictions regarding place and manner of advertising.	CFR Title 27, Chapter I, Subchapter A; BI, DISCUS, WI codes
Advertising (indirect)				
Paid product placement in movies and television	*Federal-level*: Paid product placement is prohibited for cigarettes and smokeless tobacco brands.	MSA; STMSA	*Federal-level*: Paid product placement allowed. *Voluntary self-regulation*: Self-regulatory codes recommend that product placements be guided by code principles (e.g., refrain from depicting underage drinking or drinking and driving) and be considered on a case-by-case basis.	BI, DISCUS, WI codes
Branded merchandise	*Federal-level*: Branded merchandise for cigarettes and smokeless tobacco products is prohibited.	MSA; STMSA; FSPTCA; FDA rule (21 CFR 1140)	*Federal-level*: Consumer specialty items (e.g., t-shirts, hats, magnets) must have the company name of the responsible advertiser or the brand name of the product. *Voluntary self-regulation*: Self-regulatory codes do not allow logos, trademarks, or brand names to be used on clothing, toys, games, equipment, or other materials to be used primarily by individuals below the legal purchasing age.	BI, DISCUS, WI codes
Free distribution	*Federal-level*: Free distribution is prohibited for all tobacco products except for smokeless tobacco products distributed in a "qualified adult-only facility." *State-level:* States may prohibit or otherwise restrict distribution of free samples of smokeless tobacco.	MSA; STMSA; FSPTCA; FDA rule (21 CFR 1140)	*Federal-level*: All states prohibit providing alcohol to individuals under 21. *State-level*: States may enact restrictions regarding free goods and services.	-

Appendix 9B (continued)

Restriction	Description of tobacco marketing restrictions[a]	Relevant federal regulation(s) for tobacco[b]	Description of alcohol marketing restrictions[c]	Relevant federal regulation or self-regulatory codes[b] for alcohol
Other marketing activities				
Promotional discounts and coupons	*Federal-level*: Promotional discounts and coupons allowed. *State-level*: States may enact restrictions regarding tobacco product pricing and coupons.	FSPTCA	*Federal-level*: Promotional discounts and coupons allowed. *State-level*: States may enact restrictions regarding alcohol pricing and coupons, including restrictions on drink specials (e.g., happy hours) at on-premise retailers and distribution of giveaways as a reward for purchasing a producer's or distributor's products.	-
Sponsored events	*Federal-level*: Sponsorship in the brand name of cigarettes or smokeless tobacco products is prohibited for athletic, musical, artistic, or other social and cultural events, and teams or entries in events. Sponsorship in a corporate name is allowed.	MSA; STMSA; FSPTCA; FDA rule (21 CFR 1140)	*Federal-level*: Sponsored events allowed. *Voluntary self-regulation*: Sponsorships are subject to self-regulatory code provisions related to advertising placement and content. Codes do not allow for marketing at events where most attendees are below the legal purchasing age. *State-level*: States may enact restrictions regarding alcohol industry sponsorship of events, including college and school events.	BI, DISCUS, WI codes
Advertising content				
Cartoon characters in advertisements	*Federal-level*: Cartoons in advertisements for cigarettes and smokeless tobacco are prohibited.	MSA; STMSA	*Federal-level*: Cartoon characters in advertisements allowed. *Voluntary self-regulation*: Self-regulatory codes do not allow advertisements to depict cartoon characters, Santa Claus, or other images or figures that primarily appeal to people below the legal purchasing age.	BI, DISCUS, WI codes
Health warnings in advertisements	*Federal-level*: Advertisements for tobacco products are required to include a statement regarding the addictiveness of nicotine: "WARNING: This product contains nicotine. Nicotine is an addictive chemical."	FDA rule (21 CFR 1100, 21 CFR 1140, 21 CFR 1143)	*Federal-level*: Advertisements may not make false or misleading health-related statements. Advertisements for distilled spirits must include the alcohol content as a percentage by volume. There is no regulation requiring health warnings in alcohol advertisements.	FAA Act and 27 CFR 4.64, 27 CFR 5.235, 27 CFR 7.235

Note. BI = Beer Institute; CFR = Code of Federal Regulations; CSTHEA = Comprehensive Smokeless Tobacco Health Education Act; DISCUS = Distilled Spirits Council of the United States; FAA = Federal Alcohol Administration; FDA = U.S. Food and Drug Administration; FSPTCA = Family Smoking Prevention and Tobacco Control Act; FTC = Federal Trade Commission; LCA = Little Cigar Act; MSA = Master Settlement Agreement; PHCSA = Public Health Cigarette Smoking Act; STMSA = Smokeless Tobacco Master Settlement Agreement; WI = Wine Institute.
[a]Advertisements for cigarettes, smokeless tobacco, and other covered tobacco products must include required warning statements. The Tobacco Control Act (2009) affirms the authority of state, local, territorial, and Tribal governments to further regulate access to and advertising of tobacco products to individuals of any age. [b]Additional sources of information on federal- and state-level restrictions are listed in the table sources below. [c]Alcohol advertisements must include contact information on the responsible advertiser and the class, type, or distinctive designation to which the product belongs. Spirits must also include alcohol content as percent alcohol by volume.
Additional sources. Alcohol and Tobacco Tax and Trade Bureau 2023, 2024,[439-441] Beer Institute 2023,[257] Campaign for Tobacco-Free Kids 2017,[442] Center on Alcohol Marketing and Youth 2012,[443] CDC 2024,[444] Counter Tools 2024,[445] Distilled Spirits Council of the United States 2022,[258] FTC 2023,[446] NIAAA 2025,[447] FDA 2010, 2020,[115,448] Kong et al. 2024,[449] Henriksen 2012,[450] MSA,[241] NIAAA,[451] Public Health Law Center 2022,[452] STMSA,[453] Tobacco Control Legal Consortium 2011,[454] Wine Institute 2024.[259]

Appendix 9C Studies That Assess the Relationship Between Alcohol Taxes and Prices on Tobacco Outcomes in the United States

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Asarkaya 2010[318]	A 10% permanent increase in the price of alcohol is simulated. Projections are based on a structural model that draws on panel data from the National Longitudinal Survey of Youth (1979), a survey of youth ages 14–22 in 1979. These data are merged with city-level alcohol price data, which are aggregated up to the state and region levels (south, west, northeast, north-central).	1979–1994	Respondents are ages 14–22 in 1979 and ages 29–37 in 1994. *N* = 1,030	Real city-level price of a can of beer averaged at the region level	Cigarettes	Participation in cigarette consumption: Dichotomous indicator that equals 1 for respondents who indicated that they smoked in the last 30 days before the interview and 0 otherwise. Quitting smoking: Exiting a cigarette consumption state.	Inconclusive Substitutes: No elasticities are reported. A table of the simulated percentage change in cigarette consumption following a 10% permanent increase in the price of alcohol is shown. This increase in alcohol price increases cigarette consumption by 1 percentage point in the first period and later periods (time between surveys). Independent: No effect. Beer price has no significant relationship with quitting smoking.	The authors also estimate that a 10% increase in the price of cigarettes increases past 30-day alcohol use by 0.5 percentage point in the first period and by similar amounts in later periods. These findings imply that alcohol is a substitute for cigarettes.

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Bates et al. 2015[319]	Estimation of cross-price elasticities using state-level panel data	1980–2007	Data from individuals in 48 U.S. states (excluding Alaska and Hawaii) constructed from multiple sources *N* = 1,344	Real sum of federal and state beer excise taxes	Cigarettes	Per capita cigarette sales (10-pack). Authors do not specify whether measure is of total or adult per capita sales.	Independent: Elasticity = −0.122; not statistically significant	The authors also find a negative and statistically significant cross-price elasticity estimate on the (log of) cigarette excise tax in the beer demand equation, suggesting that the 2 goods are complements. The implication is that people who drink beer consider the price of cigarettes when deciding how much beer to drink, whereas people who smoke cigarettes are indifferent to the price of beer.

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Decker & Schwartz 2000[320]	Estimation of cross-price elasticities using cross-sectional, individual-level data from BRFSS. These data are representative at state-level and are merged with the city-level price data.	1985–1993	Adults ages 18 and older from 45 states *N* = 474,096 (men = 203,338, women = 270,758)	Beer price: City-level price of a 6-pack of beer, aggregated at the state-level	Cigarettes	Smoking participation (smoking prevalence), cigarette consumption (cigarettes per day), and smoking intensity (cigarettes per day among people who smoke)	Complements (smoking participation, cigarette consumption) and substitutes (smoking intensity) Pooled sample (women and men): Smoking participation: complements (elasticity = –0.19). Cigarette consumption: complements (elasticity = –0.14). Smoking intensity: substitutes (elasticity = 0.04). Women only: Smoking participation: complements (elasticity = –0.22). Cigarette consumption: complements (elasticity = –0.16). Smoking intensity: substitutes (elasticity = 0.04). Men only: Smoking participation: complements (elasticity = –0.12). Cigarette consumption: complements (elasticity = –0.09) Smoking intensity: substitutes (elasticity = 0.04).	The authors conclude that "most of the price response derives from changes in smoking participation, rather than changes in consumption among those who continue to smoke and drink." In pooled regression models that have drinking participation and drinking intensity as the dependent variables, and cigarette prices as controls, the authors find that cigarettes and alcohol substitutes in consumption.

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Frye 2020[321]	Experimental, income-compensated, cross-price purchase task. Data collected via Amazon Mechanical Turk.	Not reported	Inclusion criteria: ages 21 or older, smoked tobacco products for longer than 3 months, smoked cigarettes each day in the past week, smoked more than 10 cigarettes in a day, drank more than 4 drinks in the past 30 days (female participants), drank 5 or more drinks in the past 30 days (male participants) *N* = 75	Hypothetical alcohol price: a ticket worth 1 alcoholic beverage at baseline. Alcoholic beverages were increased to 2 tickets per drink and then were increased again to 4 tickets per drink in the next condition (mini packs of cigarettes were 1 ticket per pack in both conditions).	Cigarettes	Cigarette purchases (mini packs of 2 cigarettes per pack)	Independent	"For the vast majority of participants, in each condition, the goods were independent of one another (i.e., a cross-price elasticity score of zero was predominant)… logistic regression models indicated that a cross-price elasticity score of zero was significantly more likely than a non-zero score."
Goel et al. 2016[322]	Estimation of price elasticities using panel, state-level data from multiple sources	2005–2014	48 states (excludes Alaska and Hawaii) *N* = 480	Real state beer tax (dollars/gallon) and real beer taxes in border states	Cigarettes	State-level cigarette sales per capita (20-packs). Authors do not specify whether these are total or adult per capita sales.	Complement (elasticity ranging from −0.035 to −0.031 depending on model specifications, $p < .05$)	Findings also indicate that higher beer prices in bordering states increase a state's cigarette sales (elasticity = 0.247, $p < .01$).

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Koksal & Wohlgenant 2016[323]	Estimation of cross-price elasticities using repeated cross-sections of household consumer expenditure survey diary data from the Consumer Expenditure Survey, analyzed with pseudo-panel estimation technique.	2002–2008	12 cohorts over 28 quarters. Cohorts were defined by the interaction of 3 generations (born before 1950, born between 1950 and 1964, born in 1965 or later), and 4 geographic regions (Northeast, Midwest, South, West). *N* = 336	Real prices of beer, wine, and spirits—Lewbel price indices calculated from restaurant and at home expenditures of each consumer unit for the 3 different subcategories of alcoholic beverages.	Cigarettes	Average weekly cigarette consumption (weekly cigarette expenditures divided cigarette price)	Complement (long-run elasticity = −0.723, $p < .1$)	The paper also found that the long-run cross-price elasticity of alcohol with respect to cigarette price is positive, but insignificant. Taken together, the findings suggest that "increasing alcohol prices would decrease cigarette consumption, while increasing cigarette prices does not have the same effect on alcohol consumption."

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Ma et al.2025[224]	Estimation of own- and cross-price elasticities for beer, liquor/wine, and cigarettes using data from an online volumetric choice experiment conducted among people who smoke and use e-cigarettes and alcohol.	2020–2021	Inclusion criteria: ages 21 or older; started using e-cigarettes or re-initiated e-cigarette use within the past 30 days; have smoked at least 100 cigarettes in lifetime; currently smoke cigarettes every day or some days, or smoked within the past 60 days; drank alcohol within the past 30 days *N* = 169	Price of beer (per 12 ounce can or bottle) or table wine or distilled liquor (per 5-ounce glass), with 0%, 25%, 50%, or 75% tax burden based on retail prices estimated using the 2019 Economist Intelligence Unit city-level price data	Cigarettes and closed system e-cigarettes	Consumption of cigarettes and e-cigarettes: Hypothetical cigarette and e-cigarette purchases (for consumption in 1 week)	Independent (cigarettes) and substitutes (e-cigarettes) Cigarette consumption in response to beer price: independent (elasticity = −0.025; not statistically significant) Cigarette consumption in response to liquor/wine price: independent (elasticity = −0.013; not statistically significant) E-cigarette consumption in response to beer price: substitutes (elasticity = 0.125, $p < .05$) E-cigarette consumption in response to liquor/wine price: substitutes (elasticity = 0.050, $p < .05$)	The sample included only people who currently smoked and started or re-initiated e-cigarette use within the past 30 days. They also needed to report alcohol drinking in the past 30 days.

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Markowitz & Tauras 2009[324]	Estimation of cross-price elasticities using longitudinal, individual-level data from the National Longitudinal Survey of Youth 1997. These data are merged with city-level beer prices.	1997–2001	Youths ages 12–16 as of December 31, 1996 *N* = 25,954 (male participants = 13,443; female participants = 12,511)	Real city-level price of a six-pack of beer aggregated at a county-level and core based statistical area level.	Cigarettes	Smoking participation: Participation in current smoking is represented by a dichotomous indicator that equals 1 for respondents who indicated that they smoked cigarettes in the 30 days prior to the survey and is equal to 0 otherwise.	Depends on the regression specification and sub-sample analyzed Pooled regression (both male and female participants): Complements (elasticity = -0.24, $p < .1$) in the region fixed-effects model and substitutes (elasticity = 0.11, insignificant) in the individual-level fixed effect model Male participants only: Complements (raw coefficient = -0.025, insignificant) in the region fixed-effects model and complements (raw coefficient = -0.009, insignificant) in the individual-level fixed effect model Female participants only: Complements (raw coefficients = -0.012, insignificant) in the region fixed-effects model and substitutes (raw coefficients = 0.030, insignificant) in the individual-level fixed effect model	In a pooled sample regression that has alcohol participation as the dependent variable, the authors find alcohol to be a substitute for cigarettes in both the regional- and individual-effects models (regional effects: elasticity = 0.008, insignificant; individual-effects: elasticity = 0.13, $p < .1$).

Appendix 9C (continued)

Study	Study design	Time period	Study sample *N*	Alcohol measure	Tobacco product analyzed	Tobacco use measure	Economic relationship	Notes
Petry 2001[325]	Experimental cross-price purchase task. Data collected via in-person interview.	Not specified	Eligibility criteria included alcohol abuse or dependence and a history of using at least 3 other types of substances: cannabis, opioids, cocaine, and sedatives. *N* = 53	Hypothetical alcohol price. Participants were given $35 of imitation money. At baseline, alcohol was $1 per drink, and cigarettes were $2 per 20-pack. The price of alcohol was then varied from $0.50, $1, $2.50, and $5 per drink, while all other substance prices remained constant. Participants were required to report the types and quantities of other substances they would purchase under each alcohol price change scenario.	Cigarettes	Purchases of cigarettes (in packs of 20)	Independent (elasticity = –0.03, not statistically significant)	This study was not exclusively focused on the cross-price elasticity between cigarettes and alcohol. A limitation of this purchase task study as it pertains to assessing the responsiveness of cigarette consumption to alcohol prices is that the recruited participants were not required to currently smoke cigarettes, they only needed to have sampled ≥3 other substances (the study investigated substitutability in people who reported polysubstance use).

Note. BRFSS = Behavioral Risk Factor Surveillance System.

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NCI Monograph 24
Tobacco and Alcohol: Understanding Co-Use to Reduce Cancer Incidence and Mortality

Chapter 10
Future Research Directions

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Chapter 10
Future Research Directions

Introduction

This monograph examines the co-use of tobacco and alcohol across aspects of epidemiology, biology, genetics, clinical treatment, and prevention and policy interventions. The overarching goal of the monograph is to explore how cancer control efforts can be strengthened by considering the role of alcohol use in tobacco use behavior, tobacco use prevention and cessation, and in conferring additional cancer risk and synergistic cancer risks for certain cancers. Tobacco and alcohol are both well-established causes of cancer[1-4] and are the first- and third-ranked modifiable risk factors for new cancer cases in the United States.[5] Continued efforts to prevent initiation of tobacco use, increase tobacco cessation, and reduce the harmful effects of alcohol use are critical steps towards further progress in cancer prevention and control.

The chapters in this monograph review and discuss the most up-to-date research on tobacco and alcohol co-use. Key findings are also compared to papers addressing tobacco and alcohol separately. The literature on the effects of the co-use of tobacco and alcohol, or intervention strategies that address co-use, is limited. This is somewhat surprising in light of the long history of public health surveillance documenting the prevalence of tobacco and alcohol use and co-use and convincing evidence for the synergistic effects of these substances on cancer at certain sites.[6,7] Available evidence indicates that smoking cessation interventions are effective for people who smoke and have an alcohol use disorder. Overall, the evidence indicates that coordinated treatment of tobacco and alcohol co-use is warranted.

Although there is sufficient evidence to support efforts to address co-use of tobacco and alcohol, research gaps concerning how to optimize prevention of initiation and to improve cessation of tobacco use and cessation or reduction of alcohol use remain. The co-use of tobacco and alcohol is driven by complex biological and genetic factors, including the neurobiology of addiction, the physiological effects of tobacco and alcohol exposures, behavioral genetics, carcinogenesis, and psychological processes. Tobacco and alcohol co-use is also influenced by an intricate and interconnected landscape of social, economic, and policy factors. Gaining a deeper understanding of the interplay between tobacco and alcohol use across these numerous, diverse factors holds promise for advancing health promotion and cancer prevention by enhancing support for preventing tobacco initiation, increasing tobacco cessation, and reducing harmful alcohol consumption.

Key Monograph Findings

The preceding chapters have presented the state of the science, as of 2026, regarding what is known about tobacco and alcohol co-use, its effects on cancer risk, and impacts on tobacco control. Overall, the monograph provides considerable evidence supporting greater efforts to address tobacco and alcohol co-use through individual- and population-level interventions informed by research on the many factors that influence tobacco and alcohol use and co-use. The major conclusions of the monograph are detailed in chapter 1 and here:

1. **Tobacco and alcohol are addictive and carcinogenic substances that are commonly used together.** Approximately 12% of adults in the 2015–2019 National Survey on Drug Use and Health reported co-use of cigarettes and alcohol in the past month, representing nearly 30 million people. Adults who use tobacco (e.g., cigarettes, smokeless tobacco, cigars) are more likely to engage in alcohol use, binge drinking, and risky weekly alcohol use than those who do not use tobacco.
2. **Co-use of tobacco and alcohol occurs from the earliest ages of substance use initiation, and its prevalence is substantially higher among certain population subgroups.** Tobacco and alcohol use often begins in adolescence, and long-term use of these two substances makes it more difficult to quit or reduce their use. Adults who report nondaily use of tobacco report the highest prevalence of alcohol use, binge drinking, and risky weekly alcohol use compared with other tobacco use groups. Tobacco use is positively associated with alcohol use across all racial and/or ethnic groups. Compared with the U.S. adult population overall, tobacco and alcohol co-use is elevated in a number of subgroups, including veterans, people living in rural areas, people who are part of a sexual minority group, and people with some types of mental health conditions.
3. **Tobacco use and alcohol consumption individually increase the risk for numerous types of cancer, even at low levels of use, and there is a synergistic increase in risk of squamous cell cancers of the head and neck and esophagus caused by tobacco and alcohol co-use.** In 2019, an estimated 19.3% of incident cancer cases and 28.5% of cancer deaths in the United States were attributable to cigarette smoking. Smoking one or fewer cigarettes per day increases the risk of numerous cancers. Further, an estimated 5.4% of incident cases of cancer and 4.1% of cancer deaths in the United States in 2019 were attributable to alcohol consumption. The risk for head and neck cancers increases with one drink per day and possibly at lower levels of consumption, and the risk of breast cancer increases at one or fewer drinks per day. Strong evidence from multiple countries demonstrates a synergistic relationship between tobacco and alcohol use and risk for squamous cell cancers of the head and neck and esophagus.
4. **Tobacco and alcohol co-use results in substantially increased acetaldehyde exposure.** Acetaldehyde is a potent carcinogen which damages DNA in multiple ways; it is both a component of tobacco smoke and an important metabolite of ethanol. Acetaldehyde plays a crucial role in the synergistic increases in risk observed for squamous cell head and neck and esophageal cancers in response to tobacco and alcohol co-use.

5. **People with genetic variants that reduce the rate of metabolism of acetaldehyde are at significantly increased risk of cancer if they consume alcohol.** An estimated 540 million people worldwide have genetic variants that reduce the rate of metabolism of acetaldehyde by aldehyde dehydrogenase 2 (ALDH2); these genetic variants are most common in East Asian populations. People with this variant who drink alcohol are at increased risk of squamous cell head and neck and esophageal cancers. Addressing alcohol use and tobacco and alcohol co-use represents important education and intervention opportunities to reduce the burden of cancer in this population.
6. **Tobacco and alcohol use affect multiple, complex neurophysiological processes in the brain.** Networks of brain regions influence how people respond to nicotine and ethanol. Shared biologic pathways in the brain, including those involved in risk-taking and impulsivity, interact and drive use of substances including tobacco and alcohol and substance use disorders.
7. **Studies have identified a substantial number of genetic variants that are associated with both alcohol use and tobacco use across populations, providing evidence for shared underlying mechanisms.** A better understanding of the shared biological mechanisms from additional genetic studies will inform efforts to prevent use disorders and develop pharmacotherapies for tobacco and alcohol co-use and polyuse with other substances.
8. **Smoking cessation interventions are effective for people who smoke and have an alcohol use disorder.** Engaging in smoking cessation treatment during treatment for alcohol use disorder does not jeopardize sobriety outcomes. However, many people in treatment for alcohol use disorder who smoke do not receive smoking cessation treatment. This is a missed opportunity to reduce the burden of cancer in this population.
9. **Addressing alcohol use as a part of smoking cessation treatment is highly likely to advance tobacco cessation goals.** Alcohol use, especially heavy alcohol use, is a barrier to quitting smoking. For this reason, people trying to quit smoking should be advised and assisted to reduce or stop their alcohol use. Integrated tobacco use treatments that address both tobacco and alcohol use may increase reach and effectiveness across multiple settings, including inpatient and outpatient clinical settings and community settings.
10. **Ensuring broad access to, and use of, evidence-based therapies for tobacco and alcohol treatment will help reduce cancer incidence and mortality.** Pharmacological and behavioral therapies for both tobacco and alcohol treatment are underutilized, especially the combined use of both types of therapies, which is the most effective treatment approach for both substances. Moreover, increased understanding of individual genetic variation may inform precision medicine approaches to addressing tobacco and alcohol use.

11. **Prevention programs and interventions that address both tobacco and alcohol may be effective to prevent initiation and ongoing use of both substances.** Effective interventions to prevent tobacco and alcohol use, especially among adolescents and young adults, exist at multiple levels, including the individual, group, community, mass media, and policy levels. Evidence suggests that prevention programs that address both tobacco and alcohol use are feasible. Integrated interventions are needed to prevent both tobacco and alcohol use early in life and across different population subgroups. In addition, intervention studies that focus on either tobacco or alcohol but measure effects on both substances will be highly informative.
12. **Factors pertaining to the marketing, sale, and distribution of tobacco and alcohol products (and the policies to address them) influence patterns and prevalence of tobacco and alcohol use individually and may also influence co-use.** Co-exposure to tobacco and alcohol product marketing and retail availability in the same outlets is common, suggesting the potential for cross-product effects. Greater retail availability of tobacco is associated with greater tobacco use among adults, and some evidence shows that greater availability of alcohol is positively associated with alcohol use among adults. In addition, both tobacco and alcohol retail outlets tend to be concentrated in neighborhoods characterized by greater socioeconomic disadvantage. Assessing how tobacco and alcohol industries' strategies for marketing and distribution of their products influence the marketplace and contribute to tobacco and alcohol co-use is important. Improved understanding of such influences can inform policy approaches to reduce the harms caused by tobacco, alcohol, and their co-use.
13. **Addressing the co-use of tobacco and alcohol has the potential to strengthen tobacco control and reduce cancer incidence and mortality.** Further research on tobacco and alcohol co-use will accelerate progress in preventing tobacco use initiation, improving rates of cessation, and avoiding smoking cessation lapses.
14. **Increased public awareness of both tobacco and alcohol as modifiable risk factors for cancer is an important step towards reducing the cancer burden.** Cancer prevention efforts have primarily focused on smoking and heavy alcohol use. The complex marketplace of available products, the high prevalence of co-use of tobacco and alcohol, and the fact that even moderate alcohol consumption and low levels of tobacco use increase cancer risk, pose challenges to researchers and clinicians. Increased public awareness of the cancer risks associated with tobacco and alcohol co-use will help reduce cancer incidence and mortality.

Research Opportunities

This monograph emphasizes the importance of understanding the effects of tobacco and alcohol co-use and reviews state of the science pertaining to co-use as of 2026. Throughout the monograph, each chapter details opportunities for future research efforts. This section highlights research needs by topics that are particularly important and timely as of 2026. These research needs serve as a starting point to develop informed hypotheses and future tobacco and alcohol co-use research projects.

Improving Measurement and Surveillance

Throughout the monograph, measurement is identified as an important area for improvement. Although co-use of tobacco and alcohol is fairly common, there remains a lack of complete and consistent measurement of tobacco and alcohol co-use in many nationally representative health surveys and large cohort studies, as well as a general lack of studies on co-use specifically. As the tobacco product landscape has changed and continues to change over time, it is important also to measure the use of tobacco products with varying characteristics and modes of delivery (e.g., e-cigarettes, heated tobacco products, nicotine pouches) and their co-use with alcohol. Similarly, new alcohol products continue to enter the marketplace, and efforts to measure the use of products such as ready-to-drink alcoholic beverages are needed. For both emerging tobacco and alcohol products, there is a dearth of research on shared determinants of use (such as mental health conditions, peer influences, marketing exposure), patterns of use, and health risks, including dependence, associated with each type of product and their co-use.

Most nationally representative health surveys and epidemiological studies do not include assessment of tobacco and alcohol co-use as well as detailed assessment of the use of these substances individually. This is likely because assessment of tobacco and alcohol use and co-use involves a large number of questions to adequately address the various types of tobacco and alcohol products, frequency, intensity, duration of consumption, and potentially different reference periods. Some studies have included measures of the use of multiple substances. For example, research focused on the effects of prevention interventions among adolescents has examined the use of cannabis alone or use of multiple substances; however, these broad measures are not specific to tobacco and alcohol co-use.

Studies are needed that focus on the duration of co-use patterns over the life course to assess cumulative exposures, which is especially relevant to understanding the risk of cancer. There is a clear need to develop harmonized measures to better capture the lifetime pattern of tobacco and alcohol co-use. For example, there are few cohorts that assess alcohol use beyond current or average use in the past month or year. Thus, the typical non-drinking referent group used in many studies includes both people who have never used alcohol and people who formerly used alcohol but have since quit alcohol use.[8,9] In addition, better measurement of the interpersonal and contextual factors (e.g., social norms and settings) that contribute to patterns of use of tobacco and alcohol, which is critical for prevention and treatment, are necessary.

Further complicating measurement and surveillance is the lack of tobacco and alcohol biomarkers that account for cumulative exposures. Such biomarkers would contribute to better estimates of lifetime cancer risk and cumulative effects on other health outcomes. For both tobacco and alcohol use, reliance on self-report limits accurate measurement due to both potential recall bias and stigma related to disclosing use patterns, especially for heavy tobacco and alcohol use. Improved assessment of co-use may provide new insights into the behaviors and risks associated with tobacco and alcohol co-use.

Improved measurement efforts should include better sampling, instrument designs, and measurement of important constructs for specific subpopulations, with an emphasis on the unique factors related to tobacco and alcohol co-use among subpopulations. More attention to demographic subgroups, known to have increased risk for co-use or its health effects and who are not always well-sampled in nationally representative surveys (e.g., American Indian

or Alaska Native individuals, sexual minority individuals, individuals who live in low-income communities, and individuals in rural communities), could elucidate unique concerns that could make interventions more effective.[10,11] Innovative approaches to overcome challenges in sampling and engagement with underrepresented populations could enhance the inclusivity and relevance of co-use research, ultimately helping to achieve better health outcomes. Better measurement of tobacco and alcohol co-use is essential to understand their health impacts, guide effective policies, target interventions, and reduce the health, social, and economic burdens associated with their co-use.

Assessing Shared Mechanisms of Tobacco and Alcohol Use and Cancer Risks

The genetic studies presented in this monograph have identified the thousands of genes associated with tobacco and alcohol use that may help inform future treatments.[12] There are specific underlying biologic contributions, including genetic, to smoking initiation, heaviness of use, cessation, and alcohol use. Substantial research is needed to determine the functional significance of genetic variants shown to be associated with tobacco and alcohol exposures[13] and whether research on these variants can help inform more effective use of existing therapies and contribute to the development of new ones.[13] An improved understanding of genetic associations and pathways that overlap between the two substances, enhanced brain imaging studies, and further investigation of influences of environmental exposures are additional avenues that could inform personalized medicine approaches to addressing tobacco and alcohol co-use and reducing cancer risk.

More research about the effects of co-use on the risk of other cancers is needed, especially for less common cancers where sample sizes have not always been sufficient and for cancers where associations with tobacco and alcohol have a more modest strength. Further, research is needed to assess the combined cancer risks for co-use of specific product types beyond cigarettes (e.g., cigars, e-cigarettes, smokeless tobacco) with alcohol. Risk assessments of different patterns of tobacco and alcohol use (e.g., nondaily smoking or episodic drinking) are also incomplete.

Assessing the potential biological underpinnings of different patterns of initiation, use, and co-use of tobacco and alcohol by subpopulation groups may further uncover important considerations. Many studies assess alcohol use and its health effects only in individuals with alcohol use disorders, and additional research is needed on the health effects, including cancer risk, of less frequent and intermittent alcohol use and tobacco and alcohol co-use.

Mechanistic understanding of the interactions between tobacco and alcohol is mixed at present, with a deeper understanding of how they interact in relation to acetaldehyde and aerodigestive cancers, but a more limited understanding of inflammation and hormonal pathways. Few studies assess variation in the exposure effects over time, how they are related to quitting, and testing for their reversibility once an individual has stopped use of one or both substances. Assessment of how changes in tobacco and alcohol use, separately and especially together, influence inflammation, other carcinogenic processes, and cancer risk, as well as how tobacco and alcohol cessation may result in metabolic change, repair of DNA damage, and changes in cancer risk, would be informative. Similarly, more can be learned about the role of the microbiome, specifically on how co-use of tobacco and alcohol (as well as cannabis and other products) influence carcinogenesis via effects on the oral and gut microbiomes. A better understanding

of these potential relationships may provide further opportunities for precision medicine. Notably, more research is also needed on factors modifying cancer risk reduction related to cessation of tobacco and alcohol use and especially co-use. Additional studies are needed to better understand the effect of reducing or stopping drinking for cancer risk in several organ sites, particularly for female breast cancer.[14]

Assessing Shared Genetic and Neurophysiological Mechanisms Associated With Trajectories of Tobacco and Alcohol Use and Co-Use

A growing body of genetic and neurophysiological studies, along with cohorts and birth cohorts addressing substance use patterns and cessation, are providing new targets for prevention of initiation and the potential development of new pharmacotherapies for cessation. These targets are based on genetic differences and on the identification and validation of additional phenotypes and endophenotypes associated with tobacco and alcohol use and co-use. Clinical trials incorporating genetic information may improve the effectiveness of treatments for tobacco cessation and alcohol use disorder. However, effective precision prevention and treatment approaches have yet to be developed.

Developing Interventions to Address Tobacco and Alcohol Use Concurrently

Existing evidence-based pharmacological treatments for tobacco cessation, such as varenicline, may prove to be successful for treating tobacco and alcohol co-use. However, other pharmacological treatments, such as glucagon-like peptide-1 (GLP-1) agonists, may be emerging treatments for tobacco and alcohol co-use and present additional options for concurrent treatment. Key research questions remain to identify, implement, and maintain effective tobacco use cessation treatments within the co-use context. Such research would inform efforts to integrate alcohol and tobacco environmental interventions to maximize efficiency and impact on prevention, use patterns, and health outcomes.

This monograph concludes that providing tobacco cessation treatment concurrent with alcohol treatment does not hinder the success of alcohol use disorder treatment. However, more research is needed to develop effective treatments for co-use of tobacco and alcohol for all levels of alcohol use. As of 2026, few interventions are designed to treat both substances; most address treatment of one in the context of use of the other. When people who engage in co-use have risky or harmful levels of alcohol consumption and/or alcohol use disorder, interventions ideally would address both tobacco cessation and alcohol reduction or cessation. Further studies could focus on determining how best to intervene with people who use tobacco and have low to moderate levels of alcohol consumption. More research is also needed on the efficacy of integrating treatment for both behaviors and how to best leverage behavioral support into treatment among people who co-use tobacco and alcohol.

Standardizing methods and reporting protocols for clinical trials focused on the integrated treatment of smoking and alcohol use is crucial, where definitions of co-use and inclusion criteria are reported. Including a broader range of reported levels of drinking in smoking cessation studies will increase the generalizability of findings and could yield increased treatment success outcomes.

Improving Prevention Interventions for Tobacco and Alcohol Use

The risks associated with the use of tobacco and alcohol underscore the importance of identifying effective prevention programs and policy responses to prevent and reduce the use of tobacco and alcohol. Among youth and young adults, preventing initiation and escalation to regular use are important goals for reducing the cancer risks associated with tobacco and alcohol and their co-use. Typically, tobacco prevention programs focus on measuring effects on tobacco use without specifically examining effects on alcohol use, despite shared contexts for use and shared determinants of use. Future research to evaluate the effectiveness of tobacco prevention programs would benefit from examining the effects of the programs on alcohol use and vice versa. Moreover, improved measurement of the effects of prevention programs on the co-use of tobacco and alcohol specifically are needed. Importantly, such studies should measure the use of tobacco, alcohol, and co-use before and after prevention program participation or policy implementation.

Additional research is needed to achieve an optimal balance between providing tailored prevention programs that address both tobacco and alcohol use for individual population subgroups (e.g., American Indian or Alaska Native individuals, sexual minority individuals, individuals who live in low-income communities, and individuals in rural communities), and providing general programs that are effective for the overall population. The monograph highlights that programs integrating tobacco and alcohol prevention throughout adolescence and young adulthood are particularly needed. In addition, because the marketplaces for tobacco and alcohol are constantly evolving, prevention interventions for the various non-cigarette tobacco products used by young people (e.g., e-cigarettes) and co-use with alcohol are needed. In addition, the effects of these co-use patterns on cancer risk are not well understood.

Conducting Policy Research Related to Tobacco and Alcohol Co-Use

Despite existing in the same marketplace and social environment, few studies have investigated the cross-substance effects of tobacco- and alcohol-related economic, environmental, and policy factors. Additional research is needed on how the retail environments for tobacco and alcohol interact, especially regarding cross-promotion via flavors (e.g., wine-flavored cigars) and price promotions and other marketing strategies in shared retail environments. Secondary analyses and original research that investigate the cross-substance impacts of policies on alcohol availability, marketing and countermarketing, and retailer density on tobacco use or the impact of policies on tobacco product availability or use on alcohol consumption, would be useful. This research is needed to understand how alcohol policies could complement or enhance existing efforts to reduce tobacco use. Conceptual and empirical advances are also needed to better understand policy effects on the co-use of tobacco and alcohol.

There is a small body of literature regarding how alcohol pricing policies, such as taxation, influence tobacco use. More research, with attention to study design and quality, is needed to ascertain whether higher alcohol taxes or prices reduce or increase tobacco consumption. Notably, although existing research on cross-price elasticities has shed light on this topic, much of it is outdated and primarily focused on beer and cigarettes. To better understand how

alcohol-related economic factors affect tobacco use, future studies should leverage recent data that reflect the evolving landscape of tobacco and alcohol products Also, given changes in policies and use patterns related to cannabis, studies may benefit from also including cannabis products in their analyses. There is also a significant gap in research on other alcohol policies, such as minimum age of purchase or warning labels, on tobacco use and co-use.

Additional research is needed to determine whether greater retail availability of alcohol products increases the likelihood of tobacco use and co-use, as well as to understand the impact of online alcohol sales on tobacco initiation, use, and cessation. Studies should also examine the characteristics and locations of retail outlets selling both substances, as the number and distribution of such outlets in the United States is not available. Further analysis of the spatial distribution of tobacco and alcohol retailers in relation to neighborhood demographics, such as socioeconomic status and race and/or ethnicity, is critical to addressing health disparities caused by tobacco. Research should investigate subgroup differences based on race and/or ethnicity and use geolocation and purchase data to investigate disparities in use and access. Additionally, studies on place-based alcohol pricing and its comparison to tobacco pricing across retail environments could highlight environmental inequities and guide future policies.

Addressing Risk Perception of Tobacco and Alcohol Co-Use

The scientific evidence presented in this monograph indicates that greater efforts to address tobacco and alcohol co-use could significantly advance progress in tobacco control and reduce the burden of alcohol use. Addressing tobacco and alcohol co-use is complicated by the fact that researchers, clinicians, practitioners, and people who use tobacco and alcohol may have differing perceptions of the risk that tobacco and alcohol co-use poses related to cancer. The harms of heavy tobacco or alcohol use are generally well-appreciated by the public. In contrast, the harms of less frequent tobacco use (e.g., nondaily cigarette smoking) or alcohol use (e.g., one drink per day) are generally poorly understood by the public.[15] Additionally, data from the 2022 Health Information National Trends Survey (HINTS) found that only 40.6% of U.S. adults recognized that alcohol consumption increases cancer risk.[16]

Although guidelines exist to advise people about the harms of both tobacco and alcohol use (e.g., the 2008 Public Health Service Clinical Practice Guideline[17] and the 2020–2025[18] and 2025–2030[19] U.S. Dietary Guidelines for Americans), these guidelines are distinct for each substance and do not reflect the synergistic risk of co-use specifically. Professional societies that inform and use such guidelines for clinical decision-making tend to publish substance-specific journals and sponsor substance-specific conferences, reflecting the separate domain preferences of researchers and practitioners themselves. For clinical practice to change and improve health outcomes, a stronger and more consistent focus on the cancer risk posed by tobacco and alcohol co-use is needed.

Tobacco use and alcohol use are risk factors for cancer and may contribute to worse outcomes following a cancer diagnosis. These behaviors and their co-use are important objectives for education and intervention in the cancer survivor population. In 2022, the National Cancer Institute concluded that compared to continued smoking after a cancer diagnosis, smoking cessation improves outcomes and should be a high priority for patients and their clinicians.[20] The 2022 National Comprehensive Cancer Network Guidelines for Survivorship recommend

limiting the intake of alcohol to reduce cancer risk and stress avoidance of alcohol for head and neck cancer survivors due to the risk of recurrence.[21] Other organizations that have guidelines for the care of cancer survivors, including the American Cancer Society[22] and the American Society of Clinical Oncology,[23] make similar recommendations regarding tobacco and alcohol use in this population. Further research to understand optimal dissemination of guidelines to improve screening and counseling by clinicians for tobacco and alcohol use and co-use patterns is clearly needed, as is further research on how to equip health professionals to refer people to treatment for tobacco and alcohol co-use.

Conclusion

This monograph, the 24th volume in the National Cancer Institute's Tobacco Control Monograph Series, provides a comprehensive examination of the state of the science on tobacco and alcohol co-use and the influence of co-use on cancer risk. It examines co-use of tobacco and alcohol with the primary goal of reducing cancer incidence and mortality by better understanding the interplay of tobacco use with alcohol use. As the literature on co-use continues to evolve, it presents opportunities to broaden and deepen the evidence base concerning tobacco and alcohol co-use. The volume concludes that further research on tobacco and alcohol co-use will accelerate progress in preventing tobacco use initiation, improving rates of cessation, and avoiding relapse to smoking and may contribute to the reduction of alcohol use. An important next step in addressing tobacco and alcohol co-use is developing interventions that address co-use at both the individual and population levels. Advancing research on tobacco and alcohol co-use will help shape effective interventions, clinical practice, and policies to mitigate the combined impact of these addictive products on cancer incidence and mortality.

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