The alcohol and cancer webinar will begin shortly.

**Engagement:** Submit questions at any time using the Chat Panel and select All Participants. You may need to activate the appropriate box using the floating navigation panel, found on the lower right hand corner of your screen.

**Recording:** This Webinar will be recorded and be available soon.

**Technical Issues:** If you have any technical issues, please contact Vanessa Torres, the Host of the webinar via the Chat Panel.
Alcohol as a Target for Cancer Prevention and Control: Research Challenges.

Virtual webinar
December 18, 2020
Acknowledgements

• Co-Chairs
  • Dr. Susan Gapstur, Consultant
  • Dr. William Klein, NCI

• Steering Committee
  • Dr. Elisa Bandera, Rutgers Cancer Institute of New Jersey
  • Dr. David Jernigan, Boston University School of Public Health
  • Dr. Noelle LoConte, University of Wisconsin School of Medicine and Public Health
  • Dr. Brian Southwell, RTI International and Duke University
  • Dr. Vasilis Vasiliou, Yale School of Public Health

• NCI DCCPS Organizers
  • Dr. Tanya Agurs-Collins, NCI
  • Dr. David Berrigan, NCI

• Special Thanks
  • Drs. Joanne Elena, Somdat Mahabir, Kate Castro and Alycia Boutte; Ms. Mimi Lising, NCI
  • Ms. Jennifer Schaefer and Ms. Vanessa Torres, ICF
Webinar Goals and Agenda

• Presentations regarding what is known and identify critical gaps in four key areas.
  1. the epidemiology and biology of alcohol and cancer risk.
  2. the effects of alcohol use during and after cancer treatment.
  3. individual and policy level interventions focused on reducing alcohol consumption.
  4. the public awareness of and communications about the alcohol and cancer link.

• Following each presenter, there will be open discussion. Please submit comments and questions using the chat box.
Disclosure Statement

Susan Gapstur’s efforts to chair this activity and to draft of an executive summary and white paper are supported by the National Cancer Institute.
Alcohol Produced as Early as 7000–6600 BC in China*

McGovern et al., Proc Natl Acad Sci, 101(51), 17593–8; 2004

• Cultural
• Social
• Celebratory

• Religious
• Relaxation
Alcohol Drinking and Health

- Ethanol:
  - Principal alcohol in alcoholic beverages
  - Psychoactive agent that has dependence-producing properties
- Worldwide, 3 million deaths every year (5.3% of all deaths) result from harmful use of alcohol.
- Harmful use of alcohol is a causal factor in more than 200 disease and injury conditions.
- Overall, 5.1% of the global burden of disease and injury is attributable to alcohol, as measured in disability-adjusted life years (DALYs).
- Beyond health consequences, the harmful use of alcohol brings significant social and economic losses to individuals and society at large.

https://www.who.int/news-room/fact-sheets/detail/alcohol
Accessed Dec. 6, 2020
## IARC Monograph Program

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td><strong>Sufficient Evidence of Carcinogenicity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcoholic Beverages</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>EtOH in Alcohol Beverages</td>
<td></td>
<td>X*</td>
<td>X</td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td></td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

### WCRF/AICR Continuous Update Project

[Image of WCRF/AICR Continuous Update Project]

[Image of Third Expert Report 1997 and 2007]

[Image of World Cancer Research Fund and American Institute for Cancer Research]
Understanding the Role of Alcohol Consumption in Cancer Etiology

Kevin Shield, PhD
Head, Collaborating Centre in Addiction and Mental Health
Pan American Health Organization
Assistant Professor, Division of Epidemiology
Dalla Lana School of Public Health
University of Toronto
Table 1. Summary of the evidence for a causal relationship between alcohol consumption and the risk of various cancer subtypes [7, 12, 15, 59–67]

<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Causally related to alcohol consumption</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral cavity (C02–06)</td>
<td>Sufficient evidence</td>
<td>Convincing [59]</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Oropharynx (C01, C09–10)</td>
<td>Sufficient evidence</td>
<td>Convincing [59]</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Hypopharynx (C12–13)</td>
<td>Sufficient evidence</td>
<td>Convincing [59]</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Oesophagus (C16)</td>
<td>Sufficient evidence</td>
<td>–</td>
<td>Limited – no conclusion [60]</td>
<td>Insufficient evidence</td>
</tr>
<tr>
<td>Oesophagus – adenocarcinoma</td>
<td>–</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oesophagus – squamous cell carcinoma</td>
<td>–</td>
<td>Convincing [60]</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Colon (C18)</td>
<td>Sufficient evidence</td>
<td>Convincing (men)/probable (women) [61]</td>
<td>Convincing (men)/probable (women) [61]</td>
<td>Convincing</td>
</tr>
<tr>
<td>Rectum (C19–20)</td>
<td>Sufficient evidence</td>
<td>Convincing [26]</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Liver (C22)</td>
<td>Sufficient evidence</td>
<td>Convincing</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Larynx (C32)</td>
<td>Sufficient evidence</td>
<td>Convincing</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td>Breast (female) (C50)</td>
<td>Sufficient evidence</td>
<td>Convincing</td>
<td></td>
<td>Convincing</td>
</tr>
<tr>
<td><strong>Causality not established</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stomach (C16)</td>
<td>–</td>
<td>Probable [63]</td>
<td>Controversial results</td>
<td>Not established</td>
</tr>
<tr>
<td>Gallbladder (C23)</td>
<td>–</td>
<td>Limited – no conclusion [64]</td>
<td>Controversial results</td>
<td>Not established</td>
</tr>
<tr>
<td>Pancreas (C25)</td>
<td>Observed association</td>
<td>Limited – suggestive (heavy consumption) [65]</td>
<td>Nevertheless controversial</td>
<td>Not established</td>
</tr>
<tr>
<td>Prostate (C61)</td>
<td>–</td>
<td>Limited – no conclusion [66]</td>
<td>Not established – may be associated at higher alcohol consumption levels</td>
<td>Insufficient data</td>
</tr>
<tr>
<td>Kidney (C64–65)</td>
<td>Evidence suggesting lack of carcinogenicity</td>
<td>Probable (for alcohol intake up to 30 g/day) [67]</td>
<td>Insufficient data</td>
<td></td>
</tr>
</tbody>
</table>

(Rehm and Shield, Eur Addict Res 2020)
Alcohol and Cancer

We thank Dr. Samir Zakhari for the use of this slide.
Oxidative Pathways of Alcohol Metabolism

We thank Dr. Samir Zakhari for the use of this slide.
We thank Dr. Samir Zakhari for the use of this slide.
Acetaldehyde: DNA Adduct Formation

We thank Dr. Samir Zakhari for the use of this slide.
Acetate Dependence of Tumors

Microbiome → Acetaldehyde → Ingestion → Ethanol → ADHs, Catalase, CYP2E1 → Acetaldehyde → ALDH1B1 (ALDH2) → Acetate → ATP, Co-A, AMP, PP → ACSS2 → Ac-CoA → TCA cycle
Alcohol Consumption and Cancer Risk

- Alcohol consumption increases cancer risk based on ethanol content (grams per day)

- Low dose alcohol consumption increases cancer risk

- Resveratrol does not meaningfully offset cancer risk (a std. drink of wine contains 1/100 000 of a meaningful dose)

Cao et al., 2016, BMJ
Alcohol Consumption and Heavy Episodic Drinking

• Heavy episodic drinking has been observed to increase the risk of breast cancer [Sarich, 2020].
Figure. Risk decline of laryngeal and pharyngeal cancers over a forty-year period after drinking cessation

Kiadaliri et al., 2013
Interactions with Other Risk Factors: Smoking

Cao et al., 2016, BMJ
Populations at Elevated Risk: Indigenous Populations

Moore et al., 2015
Contribution to the Overall Burden of Disease: United States 2014

Islami et al., CA Cancer J Clin 2018
<table>
<thead>
<tr>
<th>CANCER</th>
<th>MEN</th>
<th>WOMEN</th>
<th>BOTH SEXES COMBINED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ATTRIBUTABLE DEATHS, NO.</td>
<td>PAF (95% CI), %</td>
<td>ATTRIBUTABLE DEATHS, NO.</td>
</tr>
<tr>
<td>Alcohol intake</td>
<td>(95% CI)</td>
<td></td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Oral cavity, pharynx</td>
<td>3000 (2830-3180)</td>
<td>44.4 (41.9-47.2)</td>
<td>650 (590-710)</td>
</tr>
<tr>
<td>Larynx</td>
<td>750 (660-830)</td>
<td>24.5 (21.7-27.3)</td>
<td>90 (80-110)</td>
</tr>
<tr>
<td>Liver</td>
<td>3270 (1970-4840)</td>
<td>24.0 (14.5-35.6)</td>
<td>570 (340-860)</td>
</tr>
<tr>
<td>Esophagus</td>
<td>1900 (1620-2130)</td>
<td>15.9 (13.6-17.8)</td>
<td>610 (450-750)</td>
</tr>
<tr>
<td>Breast</td>
<td>—</td>
<td>15.4 (12.8-18.4)</td>
<td>6350 (5250-7570)</td>
</tr>
<tr>
<td>Colorectum</td>
<td>4460 (2870-6150)</td>
<td>16.3 (10.5-22.4)</td>
<td>1810 (1160-2660)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6290 (4590-8100)</td>
</tr>
</tbody>
</table>

Islami et al., CA Cancer J Clin 2018
Acknowledgements:
Dr. Elisa V. Bandera
Professor and Chief, Cancer Epidemiology and Health Outcomes, Rutgers Cancer Institute of New Jersey

Dr. Kevin Shield
Independent Scientist, Head WHO/PAHO Collaborating Centre Centre for Addiction and Mental Health, Toronto, Canada

Dr. Vasilis Vasiliou
Department Chair and Susan Dwight Bliss Professor of Epidemiology (Environmental Health Sciences) and of Ophthalmology and Visual Science and of Environment Yale School of Public Health
Etiology Discussion Topics

1. How do different patterns of consumption (i.e., binge vs. daily of lower amounts; liver “holidays”) or reducing/ceasing drinking affect cancer risk (including early onsets of liver and colon cancer)? How can biologic studies help inform why binge vs. low level consistent consumption affect risk?

2. What is the impact of alcohol consumption at different times of life on cancer risk (including early onsets of liver and colorectal cancer); How does pre-gravid consumption affect risk over time?

3. What is unknown about interactions of alcohol and genetic, lifestyle, environmental and sociodemographic characteristics on cancer? To what extent are the NCI Cohort Consortium, other collaborative efforts, large cohorts or other novel data sources covering these issues?

4. What is the impact of methodological issues in assessing alcohol consumption (underreporting in certain populations, dimensions of alcohol), reverse causation, residual confounding on alcohol-cancer associations? Are there corrections that can be applied to improve measurement?

5. Is there heterogeneity of alcohol-cancer associations by tumor subtype (breast cancer intrinsic subtypes) and tumor location (e.g., for colorectal cancer).

6. How can we better understand the role of alcohol in cancer etiology based on studies of alcohol effects on the immune system, metabolome, epigenome, and microbiome?
Health Effects of Alcohol During and After Treatment

Noelle LoConte, MD
Associate Professor
Division of Hematology, Medical Oncology & Palliative Care
Department of Medicine
University of Wisconsin, Madison
Overview

• Alcohol and its association with cancer outcomes

• Alcohol and its impact on cancer treatment
## Effects on breast cancer recurrence and mortality

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Special populations</th>
<th>Effect size recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collaborative Breast Cancer Study</td>
<td>22,980</td>
<td>None</td>
<td>0.85</td>
</tr>
<tr>
<td>Danish (Holm)</td>
<td>1,052</td>
<td>&gt;2 drinks/d</td>
<td>1.65 ($p=0.04$)</td>
</tr>
<tr>
<td>After Breast Cancer Pooling Project</td>
<td>9,329</td>
<td>None</td>
<td>0.83 (NS)</td>
</tr>
<tr>
<td>After BrCa Pooling Project</td>
<td>7,027</td>
<td>ER+, postmeno</td>
<td>1.19</td>
</tr>
</tbody>
</table>

Breast cancer specific recurrence (mortality)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>&lt;6 g/d</th>
<th>&gt;= 6 g/d</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premenopausal</td>
<td>1.01</td>
<td>1.25</td>
<td>0.52 (0.61)</td>
</tr>
<tr>
<td>Postmenopausal</td>
<td>1.12</td>
<td>1.51</td>
<td>0.03 (0.04)</td>
</tr>
<tr>
<td>Normal BMI 1 year predx</td>
<td>0.81</td>
<td>1.09</td>
<td>0.47 (0.50)</td>
</tr>
<tr>
<td>Overweight/obese</td>
<td>1.27</td>
<td>1.60</td>
<td>0.03 (0.04)</td>
</tr>
<tr>
<td>ER positive</td>
<td>1.00 (1.04)</td>
<td>1.23 (1.48)</td>
<td>0.19 (0.08)</td>
</tr>
<tr>
<td>ER negative</td>
<td>1.29 (1.38)</td>
<td>2.00 (1.62)</td>
<td>0.07 (0.43)</td>
</tr>
</tbody>
</table>

### Table 1. Joint Effects of Alcohol Consumption and Smoking on Risk of Contralateral Breast Cancer

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Controls (n = 567)</th>
<th>Patients With Contralateral Breast Cancer (n = 263)</th>
<th>Odds Ratio*</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Alcohol consumption and smoking at first breast cancer diagnosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-6.9 drinks/week and never/former smoker</td>
<td>416</td>
<td>73.4</td>
<td>185</td>
<td>70.3</td>
</tr>
<tr>
<td>0-6.9 drinks/week and current smoker</td>
<td>70</td>
<td>12.4</td>
<td>35</td>
<td>13.3</td>
</tr>
<tr>
<td>≥ 7 drinks/week and never/former smoker</td>
<td>65</td>
<td>11.5</td>
<td>27</td>
<td>10.3</td>
</tr>
<tr>
<td>≥ 7 drinks/week and current smoker</td>
<td>16</td>
<td>2.8</td>
<td>16</td>
<td>6.1</td>
</tr>
<tr>
<td>*P for interaction</td>
<td>.078</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption and smoking at reference date</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-6.9 drinks/week and never/former smoker</td>
<td>445</td>
<td>78.5</td>
<td>197</td>
<td>74.9</td>
</tr>
<tr>
<td>0-6.9 drinks/week and current smoker</td>
<td>49</td>
<td>8.6</td>
<td>23</td>
<td>8.8</td>
</tr>
<tr>
<td>≥ 7 drinks/week and never/former smoker</td>
<td>64</td>
<td>11.3</td>
<td>29</td>
<td>11.0</td>
</tr>
<tr>
<td>≥ 7 drinks/week and current smoker</td>
<td>9</td>
<td>1.6</td>
<td>14</td>
<td>5.3</td>
</tr>
<tr>
<td>*P for interaction</td>
<td>.047</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Odds ratios and 95% CIs were estimated using conditional logistic regression and are implicitly adjusted for each of the matching variables (age and year of first breast cancer diagnosis, county, race/ethnicity, stage, and survival time). Risk estimates are additionally adjusted for use of adjuvant hormone therapy, chemotherapy, body mass index at reference date, and first degree family history of breast cancer.

1P < .05.

# Colorectal cancer

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>N</th>
<th>DFS with heavy drinking</th>
</tr>
</thead>
<tbody>
<tr>
<td>German</td>
<td>Cohort, interview</td>
<td>511 v 248</td>
<td>1.32 (1.05-1.66)</td>
</tr>
<tr>
<td>Seattle Colon Cancer Family Registry</td>
<td>Telephone interview to incident cases in tumor registry</td>
<td>2264</td>
<td>1.02 (0.78-1.32)</td>
</tr>
<tr>
<td>Seattle Colon Cancer Family Registry</td>
<td>Telephone interview to incident cases in tumor registry</td>
<td>4966</td>
<td>Wine 0.90 (0.68-1.22)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Beer 1.01 (0.84-1.22)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Liquor 0.94 (0.73-1.21)</td>
</tr>
<tr>
<td>Schwedhelm et al</td>
<td>Meta-analysis</td>
<td>209,597</td>
<td>1.17 (1.05-1.31)</td>
</tr>
<tr>
<td>N0147</td>
<td>Randomized phase III trial (FOLFOX vs FOLFOX/cetuximab), food questionnaire prior to treatment</td>
<td>1,984</td>
<td>Wine 0.68 (0.45-1.04)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Beer 0.81 (0.60-1.09)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Liquor 1.00 (0.66-1.52)</td>
</tr>
</tbody>
</table>

Alcohol and the cancer patient

• 2007-2017 National Health Interview Survey of adults with cancer: 56% reported using alcohol and 34% exceeded moderate limits

• Alcohol abuse is associated with comorbid psychiatric conditions which affects cancer treatment adherence and quality of life

• Heavy alcohol use is predictive of malnutrition and increased susceptibility to bacterial infections with poorer outcomes

Sanford NN, J Natl Compr Cancer Network, 2020
Lundberg JC, psycho-oncology. 6:253-266, 1997
Alcohol and impact on cancer treatment

- Heavy alcohol use is associated with post operative complications, poorer surgical outcomes and longer hospitalizations

- Heavy drinkers have increased comorbidities e.g. cardiovascular risk, liver dysfunction that can complicate systemic treatment choices and guideline adherence

- Smoking and alcohol use during and after radiation for oropharyngeal cancer have been associated with increased risk of osteoradionecrosis of jaw

Owosho AA et al. Oral Oncol 64:44-51, 2017
Is there a benefit to alcohol cessation on cancer outcomes?

Meta Analysis of Alcohol Cessation and Risk of Laryngeal and Pharyngeal Cancers

Kiadaliri et al. Plos One, 2013
What is the oncologist’s role in counselling patients about heavy alcohol use?

• A cancer diagnosis is a teachable moment for risk-reduction health behaviors – 30% of participants of head and neck 5000 clinical cohort lowered alcohol use post diagnosis

• Patients’ perceptions of negative effects of continued alcohol use and receipt of counseling on alcohol use are associated with increased chance of decreased use after diagnosis

• Interventions for heavy drinking in the primary care setting have been effective at decreasing alcohol use

• Heavy alcohol use associated with health outcome risks e.g. cardiovascular disease, liver disease, accidents which affect non-cancer related mortality

Penfold CM et al. Head Neck. 40: 1389-1399, 2018
Eng L et al. European Journal of Cancer Care, 28, 2018
Reid MC et al. Arch Intern Med.159:1681-1689, 1999
During/after Treatment Discussion Topics

1. Characterize pre- and post-diagnosis drinking (and change in drinking from pre-to post) among survivors (i.e., impact of a cancer diagnosis on alcohol consumption)

2. Further characterize associations of pre- and post-diagnosis (and change in drinking from pre-to post) on prognosis (disease specific mortality vs. overall mortality) and patient-reported outcomes (e.g., quality of life, sleep, fatigue, neuropathy)

3. How does ongoing alcohol use affect chemotherapy tolerance, side effects, treatment efficacy, and guideline concordant treatment? What is the effect on radiation and oncologic surgery treatment?

4. What is the optimal way for physicians and other providers to ask about alcohol use? What is the optimal electronic health record-based screening tool? What are the weaknesses for assessing alcohol use in currently available datasets?

5. What is best practice about helping cancer patients cut down on their drinking?

6. Cross cutting issue: COVID-19 effects of alcohol use? Highlight for the effect on women?
Effective Policies Relevant to Reducing the Health Effects of Alcohol Consumption

Timothy Naimi, MD, MPH
Director
Canadian Institute for Substance Use Research
University of Victoria
Alcohol Policies and Cancer

National Cancer Institute: Alcohol and Cancer Webinar
December 8-10, 2020

Timothy S. Naimi M.D., M.P.H.
Director, Canadian Institute for Substance Use Research (CISUR)
Professor, Department of Public Health and Social Policy
University of Victoria, Victoria, BC, Canada
Alcohol Policies and Cancer: Conceptual Framework

Alcohol Policies
- Overall policy environment
- Policy subgroups
- Individual Policies

Alcohol Consumption
- Total amount per person
- Patterns (how consumed?)

Cancer
- Alcohol-related cancers
  - In aggregate
  - By individual cancer types

Other Factors
- Socio-demog. factors
- Smoking
- Obesity
- Physical Inactivity

Reproductive factors
- HPV infection
- HCV & HBV infections
- Screening
Effective Alcohol Policies: Community Guide Recommendations

- Increase alcohol taxes
- Regulate alcohol outlet density
- Dram shop (commercial host) liability
- Avoid privatization of alcohol sales
- Maintain limits on days of sale
- Maintain limits on hours of sale
- Enhance enforcement of laws prohibiting alcohol sales to minors
What works: WHO list of most effective and cost-effective interventions

- Alcohol taxes and other price controls
- Regulate physical availability through restrictions on time, place, and density of alcohol outlets
- Regulate alcohol advertising and other marketing
Alcohol Consumption and Risk of Cancers:
Meta-analysis of Individual-level Risk

Bagnardi et al, 2015
Alcohol Consumption and Cancer: Population-level Data

- Individual-level consumption risk $\rightarrow$ alcohol-attributable cancers in population, using population attributable fraction (indirect) methods

- Population-level alcohol consumption $\rightarrow$ alcohol-related cancer mortality in population (less evidence)


Alcohol Policies and Cancer

• If alcohol policies affect consumption, and consumption can affect cancer, alcohol policies can affect certain cancers

• Modeling studies quantify this for taxes

• What about aggregate measures of alcohol policy?


Alcohol Policies and Alcohol-attributable Cancer Mortality in the United States

Maha Alattas, Craig S. Ross, Elizabeth R. Henehan, Timothy S. Naimi

*Clinical Addiction Research and Education Unit, Section of General Internal Medicine, Boston Medical Center, 801 Massachusetts Ave, Boston, 02118, MA, USA

Department of Epidemiology, Boston University School of Public Health, 715 Albany St, Boston, 02118, MA, USA

Department of Community Health Sciences, Boston University School of Public Health, 715 Albany St, Boston, 02118, MA, USA
Study:

• Alcohol Policy Scales (APS) scores, U.S. states (29 policies weighted by efficacy, implementation)

• Related state APS scores to annual alcohol-attributable deaths for 6 cancer types in US states


Alcohol Policies and Alcohol-Attributable Cancer Mortality
Associations between 10% difference in APS score and relative difference in alcohol-attributable cancer mortality rates

<table>
<thead>
<tr>
<th>Cancer Types</th>
<th>Total</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>Six Types Combined</td>
<td>-8.5%*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Breast Cancer</td>
<td>n/a</td>
<td>-7.3%</td>
<td>n/a</td>
</tr>
<tr>
<td>Esophageal Cancer</td>
<td>-4.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laryngeal Cancer</td>
<td>-9.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver Cancer</td>
<td>-7.7%*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Oropharyngeal Cancer</td>
<td>-8.3%*</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Prostate Cancer</td>
<td>n/a</td>
<td>n/a</td>
<td>-8.5%*</td>
</tr>
</tbody>
</table>

* significant alpha 0.05

Figure 3. Change in median scores for state alcohol policy subgroups, 1999–2018. Lines consist of two sets of
Change in State Alcohol Taxes, U.S.

Figure 1. Average U.S. state alcohol specific excise tax rates per standard drink, inflation adjusted to 2015 dollars, by beverage type, 1991–2015. The usage of substances.
Summary

- Alcohol policies affect alcohol consumption
- Alcohol consumption affects cancers
- Changing policies is the cornerstone of a public health approach to cancer prevention
- Changing alcohol policies is difficult
- Additional research about policy-cancer relationships would be helpful for science, policy development
Acknowledgements

- NIH Grant support: RO1AA018377, R01AA023376

- Co-authors:
  - Maha Alattas
  - Elizabeth Henehan
  - Craig Ross

- Presentation doesn’t represent views of NCI, NIAAA or NIH
Policy Discussion Topics

1. What more do we need to know?
   • Do we need longitudinal research on the relationship between specific policies and policies in combination on cancer incidence, prevalence and survivorship?
   • What types of modeling studies are needed to estimate policy effects on cancer?

2. What are the translational science needs – cost studies, economic effects, policy coherence within larger non-communicable disease (NCD) framework, role of women and low and middle income (LMI) communities and countries to enhance policies to reduce alcohol consumption?

3. What are the research gaps that, if filled, would be helpful to the efforts of the non-governmental organization (NGO) community?
Designing Public Communication Efforts to Address Alcohol and Cancer Risk

Courtney Scherr, PhD
Assistant Professor
Center for Communication and Health
Department of Communication Studies
Northwestern University
Awareness of the Association between Alcohol Consumption and Cancer in the U.S.

# Correlates of Awareness

<table>
<thead>
<tr>
<th>Previous Correlates</th>
<th>Possible Correlates</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Personal cancer history</td>
<td>• Employment Status</td>
</tr>
<tr>
<td>• Family cancer history</td>
<td>• Race/Ethnicity</td>
</tr>
<tr>
<td>• Sex</td>
<td>• Health self-efficacy</td>
</tr>
<tr>
<td>• Smoking Status</td>
<td>• Cancer Worry</td>
</tr>
<tr>
<td>• Age</td>
<td>• Cause ambiguity</td>
</tr>
<tr>
<td>• Education</td>
<td>• Cancer fatalism</td>
</tr>
<tr>
<td></td>
<td>• Information seeking</td>
</tr>
</tbody>
</table>

Wiseman, KP & Klein, WMP (2019) Cancer Epidemiology, Biomarkers & Prevention
Correlates of Awareness

Previous Correlates
Age: “don’t know”
- 18 – 39 years; OR = 0.47 (0.23 – 0.95)
- 40 – 49 years; OR = 0.63 (0.40 - 0.97)

Possible Correlates
Self efficacy: “don’t know”
- Somewhat/not; OR = 2.32 (1.30-4.14)
- Very; OR = 2.07 (1.37-3.14)

Cause ambiguity: “yes”
- OR = 1.61 (1.08 – 2.42)

Information seeking: “yes”
- OR = 1.80 (1.27-2.57)

*Referent outcome group was “No”
International Efforts to Increase Awareness

Adapting, implementing & evaluating strategies from anti-tobacco campaigns

Testing WHO recommended strategy of container labeling

Gain support for policies

Change beliefs, attitudes & behavior

World Health Organization, 2017
"Alcohol does something to us"

<table>
<thead>
<tr>
<th></th>
<th>Pre-campaign (n = 3000)</th>
<th>Post-campaign (n = 3000)</th>
<th>(p)-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unprompted awareness</td>
<td>22.2%</td>
<td>27.0%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Prompted awareness</td>
<td>44.8%</td>
<td>49.7%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Support min. unit pricing</td>
<td>25.7%</td>
<td>31.0%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Support ad. bans</td>
<td>40.7%</td>
<td>44.1%</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Support nutrition labeling</td>
<td>43.9%</td>
<td>47.5%</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

**Males only \(n = 1500\) \(n = 1514\)**

<table>
<thead>
<tr>
<th></th>
<th>(n = 1500)</th>
<th>(n = 1514)</th>
<th>(p)-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>18 year age limit</td>
<td>42.5%</td>
<td>51.8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age limit on schools</td>
<td>44.2%</td>
<td>53.8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Enforcement of age limits</td>
<td>62.9%</td>
<td>67.4%</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Warning Labels

Warning Labels

Cancer Warning

National Drinking Guidelines

Example Standard Drink Information

c. Thought about labels

- Intervention
- Comparison

Wave 1 Wave 2 Wave 3

25.4% 33.2% 33.6%
22.0% 23.9% 19.1%

d. Talked with others about labels

- Intervention
- Comparison

Wave 1 Wave 2 Wave 3

11.0% 22.4% 20.5%
10.9% 12.9% 7.6%

e. Self-reported drinking less due to labels

- Intervention
- Comparison

Wave 1 Wave 2 Wave 3

14.9% 11.5% 9.2%
6.2% 9.9% 6.9%

Fig. 3. (a–e). Impact of alcohol warning labels on label outcomes in intervention and comparison sites.
Multiple Source condition:

- More believable, convincing & personally relevant

- More likely to report they should & would:
  - Reduce current alcohol consumption
  - Reduce intentions to consume 5+ drinks in a single sitting
Interpersonal Communication

• Family communication:
  – Interpersonal influence can shape decisions and health behaviors

• Clinicians:
  – Raising awareness
  – Personalization
  – Clear & consistent messaging

Alcohol Industry Strategies

Parallel tobacco industry strategies:

1) **denying, omitting, disputing** – evidence that alcohol consumption increases cancer risk

2) **distorting** – mentioning cancer, but misrepresenting risk

3) **distracting** – focusing discussion away from independent effects of alcohol on common cancers (breast & colorectal)
Contextual Challenges

Mixed Messages

I drink every day, but not very much. Is that risky?

Some studies have shown that those who drink moderate amounts of alcohol have lower rates of heart disease than nondrinkers. But drinking alcohol every day to excess can lead to serious cardiovascular disease risks including high blood pressure, obesity and stroke. If you find yourself drinking more and more over time, consider cutting back.


Sociocultural Aspects

Guideline Conflicts/Changes

www.airc.org
Psychosocial Challenges

- Cognitive Dissonance
- Reactance
- Information Overload
- Cause Ambiguity
- Fatalistic Beliefs
What do we need to know in order to design public communication efforts to address alcohol and cancer risk?
Communication Discussion Topics

1. What roles could public communication campaigns play to affect alcohol use?
2. How should we think about misinformation circulating about relationship of alcohol and cancer as a topic to investigate?
3. How can we mitigate health disparities through communication?
4. How can we best support health care professionals as they discuss alcohol and cancer with patients and their families?
5. How should we counsel cancer patients about the utility of alcohol reduction?
Closing Remarks

William P. Klein, PhD
Associate Director
Behavioral Research Program
Division of Cancer Control & Population Sciences
National Cancer Institute
IF I HAVE SEEN FURTHER, IT IS BY STANDING ON THE SHOULDERS OF GIANTS

- SIR ISAAC NEWTON

.... and many more!
Behavioral Research Program Selected Alcohol-Related Activities and Resources

- Panels and Sessions at Conferences: e.g. SBM, SPR, ASCO, APHA
- Consultation with SMEs: e.g. Dr. David Jernigan
- Webinars: e.g. Dr. Noelle LoConte, Alcohol and Cancer
- Data Resources: Health Information National Trends Survey
- Funding for Alcohol and Tobacco Supplements, 2020
- 2020 Notice of Special Interest (NOSI) on Alcohol and Cancer (w/NIAAA)
- Fellows Training and Research: J. Scheideler, K. Wiseman, R. Eck, Á. Budenz, H. Platter, A. Siedenberg, M. Mayer etc.
- Workshop Dec. 8-10th, 2020: Alcohol and Cancer: Identifying Evidence Gaps and Research Challenges Across the Cancer Control Continuum

Alcohol as a Target for Cancer Prevention and Control: Research Challenges. Public Webinar, 2:00-3:30 pm EST Dec. 18th 2020
THANK YOU