Alcohol Marketing and Youth Drinking: Is There a Causal Relationship, and Why Does It Matter?

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It has long been known that addressing behavioral risk factors (Lim et al., 2012) is key to preventing or delaying chronic diseases and unintentional injuries. Cigarette smoking, overeating, and alcohol consumption are the three behaviors implicated as direct or indirect causes of cancer, heart disease, chronic lung disease, liver cirrhosis, and diabetes, the five diseases that result in the lion’s share of chronic disease mortality and morbidity. Alcohol is implicated in 4% of global deaths and in 10% of deaths among 15- to 49-year-olds; it is implicated in 9% of global disability-adjusted life years among men and 2% among women (GBD 2016 Alcohol Collaborators, 2018). Alcohol costs amount to more than 1% of the gross national product of high- and middle-income economies (Rehm et al., 2009).

In contrast to cigarette smoking, which begins to exert an influence on mortality after age 40 (Centers for Disease Control and Prevention [CDC], 1993), alcohol begins its influence in childhood and adolescence because of its contribution to injury, deaths, and suicide (Rehm et al., 2009). The most recent data available indicate that between 1990 and 2013, alcohol use was the leading risk factor for death among 15- to 19-year-olds and 20- to 24-year-olds (Mokdad et al., 2016).

This supplement to the Journal of Studies on Alcohol and Drugs contains eight articles that summarize the current scientific literature that has assessed the scope and extent of alcohol marketing, the scientific evidence that exposure to alcohol marketing influences how young persons perceive alcohol, and whether exposure influences youths’ decisions to engage in underage drinking. The focus on alcohol marketing is warranted not only because it has been implicated as a potential contributor to the global burden of disease (Esser & Jernigan, 2018) but also because it represents a modifiable risk factor that could have enormous implications for prevention policies.

Although the medical system typically treats behaviors as risk factors addressable at the individual level, public health scientists focus on the basis for the behaviors, sometimes referred to as the commercial determinants of health (Kickbusch et al., 2016), which encompass the actions of multinational corporations in the development, marketing, and worldwide distribution of cigarettes, ultraprocessed foods, and alcohol (Moodie et al., 2013). Corporate marketing of ultraprocessed food begins with the marketing of fast food (Emond et al., 2016) and sugar-sweetened cereals (Emond et al., 2019) to preschoolers. Tobacco and alcohol companies have always claimed that they avoid targeting the underage market, but internal tobacco company documents obtained from court proceedings have proved otherwise (Cummings et al., 2002), and this evidence was sufficient for a U.S. federal judge to convict cigarette companies of conspiring to hide the harms of smoking and market to underage youth (Vernick et al., 2007). No such company documents exist for alcohol, except for a troubling investigation conducted by the United Kingdom Home Office suggesting that some of the large multinational producers in the United Kingdom were targeting children and adolescents with their marketing campaigns (Hastings, 2009).

It is not necessary to show that companies intended to market a product such as alcohol to youth to justify public health policies to constrain marketing practices. The basis for action to constrain marketing of these products to youth is scientific evidence that exposure to marketing among youth is associated with use of the product and subsequent harm to health. For tobacco, years of observational research were summarized in three successive Surgeon General Reports. The first, published in 1994 (U.S. Department of Health and Human Services, 1994), included a chapter titled “Tobacco Advertising and Promotional Activities.” This report did not conclude that tobacco marketing was one cause of underage smoking but had five carefully worded conclusions:

1. Young people continue to be a strategically important market for the tobacco industry.

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2. Young people are currently exposed to cigarette messages through print media (including outdoor billboards) and through promotional activities, such as sponsorship of sporting events and public entertainment, point-of-sale displays, and distribution of specialty items.

3. Cigarette advertising uses images rather than information to portray the attractiveness and function of smoking. Human models and cartoon characters in cigarette advertising convey independence, healthfulness, adventure-seeking, and youthful activities—themes correlated with psychosocial factors that appeal to young people.

4. Cigarette advertisements capitalize on the disparity between the ideal and actual self-image and imply that smoking may close the gap.

5. Cigarette advertising appears to affect young people’s perceptions of the pervasiveness, image, and function of smoking. Since misperceptions in these areas constitute psychosocial risk factors for the initiation of smoking, cigarette advertising appears to increase young people’s risk of smoking. (U.S. Department of Health and Human Services, 1994, p. 10)

Tobacco marketing has been heavily regulated in most countries since this was written—there are no more billboards, cigarette ads do not appear on television, and cigarette image advertising has been greatly constrained. But alcohol marketing has not. As career scientists who have studied both cigarette and alcohol marketing, it is not hard for us to see how one could just replace “cigarette” with “alcohol” and “smoking” with “drinking” and have a set of conclusions that would accurately describe the current landscape for alcohol marketing.

As research accumulated, subsequent Surgeon General Reports included a clearer statement about marketing implications for youth smoking. The 2012 Surgeon General Report (National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2012) included a chapter titled, “The Tobacco Industry’s Influences on the Use of Tobacco Among Youth,” which stated the following: “The evidence is sufficient to conclude that there is a causal relationship between advertising and promotional efforts of the tobacco companies and the initiation and progression of tobacco use among young people” (p. 8).

This supplement was written with the recognition that the U.S. Surgeon General and CDC have not sought to summarize the scientific literature on alcohol and that this neglect has left gaps in our understanding of the relation between alcohol and disease, and, more specifically, the relation between alcohol marketing and underage drinking.

Basis for the supplement

This supplement was funded, in part, by a research grant from the National Institute on Alcohol Abuse and Alcoholism (R01 AA021347), titled Alcohol Marketing and Underage Drinking. On the first review of the proposal, a study section reviewer noted the lack of a plan to integrate various studies in this area, and our response promised a review article. In thinking about the review, we noted multiple reviews of tobacco marketing and underage smoking conducted by the office of the Surgeon General, and how that summary work benefitted the field by enabling scientists to review the literature and determine if the evidence supported a causal statement. We noted also that, although the adverse effects of alcohol consumption on disease and well-being among youth were clear, and although the literature on the relation between alcohol marketing exposure and underage drinking is extensive, no similar process had ever been initiated for alcohol at the Office of the Surgeon General.

In 2016, Rutger Engels sponsored a meeting of behavioral scientists in Utrecht, The Netherlands, in which we decided to conduct a Cochrane review of all longitudinal and experimental studies of alcohol marketing and drinking among youth. A team of investigators obtained sponsorship from the appropriate Cochrane review committees and published a protocol (Cukier et al., 2018), which guided subsequent and ongoing efforts both to finish the Cochrane review and to also commission other articles that we deemed important for a scientific summary but out of the scope of the Cochrane review. Those articles were commissioned and discussed at a subsequent 2017 meeting in Atlanta, GA; they include systematic and narrative reviews. All systematic reviews are based on the comprehensive literature search that serves as the basis for the Cochrane review.

Method

Two information specialists from the project coordination team developed a general search strategy that would serve to populate four unique systematic reviews on the topic of alcohol marketing and underage drinking. The four systematic reviews are described briefly below:

(a) Cochrane review of longitudinal and experimental studies, titled “Impact of exposure to alcohol marketing and subsequent drinking patterns among youth and young adults.” The published protocol (Cukier et al., 2018) includes a summary of previous reviews; a definition of marketing comprising paid, earned, and owned media that aims to cultivate brand allegiance and increase demand for a product; and a theoretical discussion of how marketing could influence underage drinkers; as well as objectives and methods.

(b) Cross-sectional studies of exposure to alcohol marketing and its influence on alcohol attitudes (Henehan & Ross, 2020, this supplement).

(c) Cross-sectional studies of exposure to alcohol marketing and its influence on alcohol behaviors (Finan et al., 2020, this supplement).
Figure 1 shows the PRISMA search results. The identification of studies was completed in sequence, as follows.

Step 1: To identify potentially relevant studies, information specialists from the project coordination team at Dartmouth College searched the following databases: Cochrane Central Register of Controlled Trials (CENTRAL) (1992 to February 14, 2017); MEDLINE via Ovid (1946 to February 14, 2017); Embase via Elsevier (1947 to February 14, 2017); Web of Science (1900 to February 14, 2017) and CINAHL via EBSCO (1981 to February 14, 2017); PsycINFO via EBSCO (1806 to February 14, 2017); Communication & Mass Media Complete via EBSCO (1918 to February 14, 2017); Econlit via Proquest (1969 to February 14, 2017). The search included subject headings and text words to capture the concepts of alcohol and marketing. The search strategy was adjusted for the syntax appropriate for each database. This initial search yielded 27,351 records.

Step 2: Information specialists de-duplicated records and provided results to the project coordination team (18,997 records). Research assistant members of the project coordination team conducted a second round of de-duplication and a title screen resulting in 11,126 records retained.

Step 3: Two investigators (Samantha Cukier, Ashley Wettlaufer) screened titles and abstracts from the 11,126 records identified in step 2 into the following five categories, using Rayyan online software (http://rayyan.qcri.org): (a) longitudinal studies (n = 126), (b) experimental studies (n = 145), (c) cross-sectional studies (n = 1,346), (d) descriptive studies (n = 1,173), and (e) influence of digital marketing (n = 119). Full text records were provided to each of the four author groups. The longitudinal and experimental studies were screened by the author group conducting the Cochrane review. The cross-sectional studies were screened by two author groups to capture studies that assessed the influence of exposure on behavior (Finan et al., 2020) and attitudes (Henahan et al., 2020). Some studies were screened by both author groups because they address both behaviors and attitudes. The studies on digital marketing were screened by the author group conducting the review on the influence of digital marketing (Noel et al., 2020). Each author group then went on to complete full-text screening, using their own inclusion and exclusion criteria for their respective categories of articles. For reasons of feasibility, the review of descriptive studies was not completed and so is not included in the PRISMA flow diagram.

Scientific framework—The Bradford Hill Criteria

The proposition that exposure to marketing affects drinking behavior is not easily tested using the “gold standard” research tool—the randomized trial. Randomization of the exposure would be ideal because exposure to marketing communications depends to a certain extent on individual preferences. Large-scale rollouts of marketing campaigns would preclude cluster randomized trials, in which entire communities were shielded from alcohol marketing campaigns. Randomized studies on underage drinking behavior at the individual level would be unethical to perform. However, a handful of studies have been conducted in young adults of legal age, primarily from Europe (e.g., Koordeman et al., 2012). These experiments have studied short-term responses to alcohol marketing, such as effects on immediate drinking (Koordeman et al., 2012). To the extent that most commercial marketing of alcohol now involves a repeated-exposures process of consumer socialization (Harris et al., 2015) that leads over a period of months to years to brand preference and consumption (McClure et al., 2013), short-term randomized experiments necessarily underestimate marketing influence. Because of these constraints, policy decisions about alcohol marketing must rely on our interpretation of observational studies.

There are many observational studies that address alcohol marketing, its thematic content, and its association with underage drinking. There have also been experimental studies that have investigated different aspects of alcohol marketing exposures, primarily in terms of psychological and neurobiological responses to alcohol-related stimuli or cues (Courtney et al., 2020; Finan et al., 2020; Jackson & Barthlow, 2020). Our approach to summarizing this literature is organized around the Bradford Hill criteria for causality.

The Bradford Hill criteria were developed by an environmental epidemiologist who studied disease outcomes. In the context of a workplace epidemic of, for example, a certain type of cancer, such as liver cancer in workers exposed to vinyl chloride (Waxweiler et al., 1976), Hill (1965) developed criteria that could be applied to determine if a causal statement was warranted from the observational epidemiological evidence. From his perspective, causation was not something that could be proved by any one study or research design but was based on a summary judgment, typically by a panel of scientists, after reviewing all the evidence on a given association. This supplement is an attempt to gather that evidence to support a scientific determination of whether the association between exposure to alcohol marketing and underage drinking is causal.

In his classic 1965 President’s Address to the Royal Society of Medicine, Hill laid out nine criteria for causation: (a) strength of association, (b) consistency, (c) specificity of association, (d) temporality, (e) biological gradient (dose-response relationship), (f) biological plausibility, (g) coherence, (h) experimental evidence, and (i) analogy. These have subsequently been used to judge whether the association between marketing exposures and behavioral outcomes is causal for tobacco marketing and underage smoking (National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2012). This litera-
Figure 1. PRISMA 2009 flow diagram. Source: Moher et al. (2009). Used with permission.
ture suggests the need first to understand the exposure in its broader context. Jernigan and Ross (2020) begin our supplement with an overview of the alcohol marketing landscape in order to map the scope of alcohol marketing globally, along with the different communications venues through which it is deployed.

Hill suggested that an exposure–disease association must be plausible; that is, there has to be some hypothesized mechanism of action through which an agent embedded in the exposure exerts a biological influence that results in disease. In this supplement there are three contributions that address plausibility (Courtney et al., 2020; Henehan et al., 2020; Jackson & Bartholow, 2020). Psychologists develop, measure, and test psychological models in which exposures affect thoughts and cognitions or attitudes, thereby changing the likelihood that an individual will engage in a behavior. Theoretical approaches that come to mind when thinking about alcohol exposure and youth drinking include social cognitive theory (Bandura, 1989), problem behavior theory (Jessor, 1987), the theory of reasoned action (Sheppard et al., 1988), and the related prototype-willingness model (Gerrard et al., 2008). Jackson and Bartholow (2020) provide a narrative summary of psychological plausibility by examining the level of scientific support for these and other behavioral models. The review by Henehan et al. (2020) addresses the association between alcohol marketing exposure and cognitions/attitudes toward alcohol among underage persons.

Biological plausibility is an area where functional magnetic resonance imaging and other neurobiological methods are now being used to test how young persons respond to alcohol marketing compared to other marketing inputs, and whether those responses relate to their own real-world drinking. The supplement article by Courtney et al. (2020) provides a narrative summary of the available scientific evidence to support the notion that brain responses to alcohol advertising relate to underage drinking behavior.

Hill (1965) also suggested that an argument for causality could be supported by analogy—“In some circumstances it would be fair to judge by analogy. With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy” (p. 11). It is not unreasonable to consider analogy given the widespread acceptance that tobacco marketing is one cause of youth smoking, the similar age at onset of smoking and drinking, that alcohol companies look to the same advertising firms that successfully marketed cigarettes to youth, and that the scope of the advertising investment is as similar for alcohol as it was for tobacco during the 1960s, 1970s, and 1980s, when cigarettes were widely marketed to youth across multiple media channels. The article by Weitzman et al. (2020) makes the case for analogy, this by a scientist who provided evidence on tobacco marketing and youth smoking for the tobacco Racketeer Influenced and Corrupt Organizations trial (U.S. v. Philip Morris, 2006).

The most important Bradford Hill criterion is the strength of the association. A strong association is easier to act on because a strong association often has big public health implications. Hill emphasized that it is not necessary to subject a strong association (for example, mortality from scrotal cancer was 200 times more common among chimney sweeps, among whom exposure to tar and mineral oils were an occupational hazard) to covariate controls or p-value determination, because the association is unlikely to be undermined. One of the first attempts to document an association between alcohol marketing and youth drinking came from econometric studies, an area where the strength of evidence has been positive but weak. This issue is revisited in the present supplement in a new review by Henry Saffer (2020).

For weaker associations, it is important to show that the association is independent—that it remains significant after controlling for covariates that are associated with both the exposure and the outcome. In behavioral modeling, it is important to think carefully about confounders and mediators, which are statistically indistinguishable. A confounding covariate is a variable that is associated with both the exposure and the outcome but is independent of the causal pathway from exposure to behavior. A mediator is a variable that is set in motion by the exposure and contributes indirectly to the effect of the exposure on behavior. Psychological constructs such as alcohol expectancies are often modeled as mediators to create mental models that shed light on mechanisms. Specificity is present when the exposure is associated with only one adverse outcome. The exposure–disease relationship does not have to be specific for a causal interpretation. For example, that smoking is thought to be a cause of heart disease does not decrease our certainty that it does not also cause lung cancer. Cigarette smoke contains many agents that could affect many disease processes. With respect to alcohol marketing, if it could be shown that exposure to alcohol advertising was associated with drinking but exposure to food marketing was not (Tanski et al., 2015), then it could be concluded that there was evidence of advertising message specificity.

Hill also considered it helpful when there was a dose–response association, such that higher doses of the exposure could be demonstrated to result in progressively higher risk. Another important aspect of determining causality is to show that the exposure precedes the outcome. This criterion places a premium on longitudinal research in which marketing exposures are measured before the onset of the behavior—these studies, along with studies in which marketing exposures were randomly assigned, will be covered in the Cochrane review on the impact of exposure to alcohol marketing (Cukier et al., 2018), the basis for this supplement. Longitudinal observational studies of
ten assess marketing exposure in a cohort of adolescents, starting with never-drinkers and then show the association between exposure at baseline and onset of drinking. Other transitions can be modeled, and some studies begin with adolescents who have not engaged in hazardous drinking (including the experimenters) and examine the transition to hazardous or binge drinking. That is not to say that cross-sectional or retrospective research is not valuable, especially if the results of these studies are consistent with the longitudinal studies.

This leads to Hill’s consistency criterion, an important one for relatively weaker associations in which confounding plays a significant role. According to Hill, if a modest association is consistently present in multivariate models despite having been tested in multiple settings and using multiple arrays of covariates, then that consistency bolsters the case for causality. Reviews by Henehan et al. (2020), Finan et al. (2020), and Noel et al. (2020) involve studies that examine the association between exposure to alcohol marketing and drinking outcomes from the standpoint of strength, dose-response, and consistency. The review by Noel et al. addresses alcohol marketing through digital media.

If alcohol marketing were responsible for a large share of youth drinking, and if such marketing were not adequately regulated, youth drinking would show increases over time. In other words, the increases would be coherent with the notion that the relation between alcohol marketing and youth drinking is a causal and important effect from a public health perspective. However, in the United States, youth drinking has been declining for three decades. For example, the Monitoring the Future surveys have assessed the proportion of 12th graders who have had 5+ drinks in a row since 1979, and it has dropped from more than 40% in the early 1980s to less than 20% in the past 3 years of the survey (Miech et al., 2018). Given the extent of marketing that will be documented in this supplement, the decline in youth drinking is not coherent with a strong causal association. But neither is it inconsistent with a modest association that is only one of many factors that influence youth drinking.

**Conclusion: Why does causality matter?**

To many readers, the present attempt to answer such a complex question as to whether alcohol marketing “causes” alcohol-related harm may be considered a frivolous academic exercise for which there is no easy answer. We disagree for two reasons. First, policymakers and public health professionals have a duty to make informed decisions about “life and death” matters like the drivers of alcohol-related mortality on the basis of the best available evidence. Second, the emerging scientific literature on alcohol marketing has matured to the point where the same causal criteria used to guide policy decisions about cancer prevention or climate change can now be applied to alcohol marketing. And just as there are those who attempt to undermine effective policy actions by pointing to apparent inconsistencies, ambiguities, or contradictions in the science base, so too has the alcohol industry attempted to cast doubt on the fundamental question: Does alcohol marketing cause the initiation of alcohol use and the development of alcohol-related problems in youth and other vulnerable populations?

This question is becoming increasingly important to answer because of the growth of alcohol marketing, promotion, and sponsorships throughout the world (Institute of Alcohol Studies, 2017), the use of new digital technologies linked to popular social media, and the introduction of new alcohol products targeted at youth populations, particularly in the emerging economies of Africa, Asia, and Latin America. Although the alcohol industry claims it does not target youth and only advertise to compete with rival brands for increased market share (European Centre for Monitoring Alcohol Marketing, 2014), internal industry documents (Hastings et al., 2009) and recent scientific research (Jernigan et al., 2017) suggest otherwise. Despite evidence to the contrary, the global alcohol producers have argued that the research is “very weak in real terms and does not make a compelling case that advertising causes harmful drinking” (International Alliance for Responsible Drinking, 2014, p. 2).

If a definitive answer could be found for the perennial question of whether alcohol marketing contributes in a causal way to alcohol consumption and drinking problems at a crucial stage of adolescent development, it could have enormous implications for public health policy designed to prevent the onset of numerous noncommunicable diseases well as premature mortality. This is why the articles in this supplement are so important.

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**Conflict of Interest Statement**

The authors have no competing interests to declare.

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