The Relationship Between Exposure to Alcohol Marketing and Underage Drinking Is Causal

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ABSTRACT. Objective: This article summarizes the findings of narrative and systematic literature reviews focused on the relationship between exposure to alcohol marketing and youth drinking, viewed in context of criteria for causality. We also consider the implications of this proposition for alcohol policy and public health.

Method: Our descriptive synthesis of findings is from 11 narrative and systematic reviews using the nine Bradford Hill causality criteria: (a) strength of association, (b) consistency, (c) specificity of association, (d) temporality, (e) biological gradient, (f) biological plausibility, (g) coherence, (h) experimental evidence, and (i) analogy. Results: Evidence of causality for all nine of the Bradford Hill criteria was found across the review articles commissioned for this supplement and in other previously published reviews. In some reviews, multiple Bradford Hill criteria were met. The reviews document that a substantial amount of empirical research has been conducted in a variety of countries using different but complementary research designs. Conclusions: The research literature available today is consistent with the judgment that the association between alcohol marketing and drinking among young persons is causal. (J. Stud. Alcohol Drugs, Supplement 19, 113–124, 2020)

In the introduction to this supplemental issue to the Journal of Studies on Alcohol and Drugs, we described how eight manuscripts were commissioned to address different Bradford Hill criteria (Hill, 1965) to assess whether the association between alcohol marketing and the onset and severity alcohol consumption by youth is causal. Causality is not a conclusion that derives from any one scientific study: It is a judgment call—a summary statement that describes how a scientist (or group of scientists) views the evidence as a whole. In Hill’s words, “No formal tests of significance can answer those questions” (p. 299). Neither can one study, regardless of how compelling it might be, answer the question. Moreover, a judgment of causality is always subject to revision as the science underlying the judgment progresses. In this concluding article, we provide our own scientific judgment of findings presented in this supplement and beyond, along with suggesting implications for public health policy and further action.

The articles, commissioned as part of a larger Cochrane review that will address longitudinal and experimental studies of alcohol marketing, cover a broad variety of approaches that have been taken to answer the causality question. The approaches comprise different research designs (cross-sectional, longitudinal, experimental), measurement techniques (survey studies, econometric research, randomized trials, laboratory studies), national contexts of exposure (high-, middle-, and low-income countries), and media/communication channels (e.g., print, television, digital, films).

Alcohol marketing research has matured since the days when exposures were measured across a collection of high-income countries in terms of aggregate amounts of industry spending on traditional advertising and when effects were measured in terms of cross-sectional association with per capita alcohol consumption at the national level (Saffer, 2020). With the addition of more sophisticated longitudinal designs, new ways to address confounding, and more representative samples responding over time to better measures of exposure to different types of advertising, confidence in the directionality of the associations and the validity of the findings has increased.

For example, a recent systematic review of research on the association between alcohol marketing and youth drinking (Jernigan et al., 2017), based on studies published since 2009, using sophisticated longitudinal designs with more than 35,000 persons, reported a significant association between youth exposure to alcohol marketing and subsequent drinking behavior. One deficiency with this method of summarizing the literature, which the present supplement was designed to correct, is that the research literature had never been organized to address multiple causal criteria in a way that would satisfy both the scientist and the policy maker. The articles in this supplement go beyond the evidence from prior reviews of the alcohol marketing literature and attempt at integration (e.g., Babor et al., 2017) by critically evaluating a variety of observational and experimental research of putative mechanisms that covers laboratory-based neurobiological studies (Courtney et al., 2020), psychological studies (Jackson & Bartholow, 2020), and econometric studies (Saffer, 2020).

In the remainder of this article, we explain how the research findings assembled in this project provide clear an-
swers to the questions posed at the beginning: Does exposure to alcohol marketing have a causal influence, and, if so, what are the implications for alcohol policy and public health?

Does exposure to alcohol marketing have a causal influence on youth drinking?

Sir Austin Bradford Hill (1965) was an environmental epidemiologist who focused on workplace hazards. He is well known for his contributions to our understanding of the relationship between smoking and disease. The basis for the widely held notion that smoking is one cause of cancer and other conditions is purely observational science—science that has been periodically summarized by panels of experts using a Hill causality framework. That is the process whereby causal statements about smoking and disease came to be.

In 1965, Hill identified a set of criteria used to assess causality for associations between environment and disease. The Bradford Hill criteria have been widely used in establishing consensus judgments about causality in medicine and public health, playing an important role in justifying evidence-based public health regulations (Doll, 2002; Hill, 1965; McDonald & Strang, 2016). These criteria may also be applied to research involving behavioral outcomes. For example, a causal statement on tobacco marketing and adolescent smoking was made by the U.S. Department of Health and Human Services (National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2012) by applying Bradford Hill criteria to that literature.

Causality is most convincingly demonstrated by randomized clinical trials, and there are some examples in the alcohol marketing literature of the use of this “gold-standard” research design to evaluate short-term relationships at the psychological, neurobiological, and behavioral levels of analysis (e.g., Jackson & Bartholow, 2020; Courtney et al., 2020; Noel et al., 2020). However, most of the Bradford Hill criteria apply to results from multiple observational studies, especially when randomized clinical trials are difficult to conduct for practical or ethical reasons (e.g., it would be unethical to assign persons to smoke cigarettes as a test of the smoking–lung cancer association). In addition, most of the studies conducted on the alcohol marketing–drinking association were based on nonexperimental observational studies in which the independent variable (exposure/receptivity to alcohol marketing) was not under the control of the researcher because of ethical or logistical constraints.

Hill’s causality criteria comprised the following: (a) strength of association, (b) consistency, (c) specificity of association, (d) temporality, (e) biological gradient (dose–response relationship), (f) biological plausibility (to this we would add plausibility regarding psychological theory), (g) coherence, (h) experimental evidence (e.g., reproducibility in animal models; experiments involving randomization), and (i) analogy. In the following analysis of the findings presented in available literature reviews, along with those presented in this supplement, we have adapted the Bradford Hill criteria to the hypothesized causal association between alcohol marketing exposures and adolescent drinking. The criteria, their definitions and their application to alcohol marketing research are shown in Table 1.

As noted in the introductory article to this supplement (Sargent et al., 2020, Figure 1), the Cochrane review project contributing to these reviews screened 18,997 articles, finding 11,126 of them to be relevant to alcohol marketing research. Of these, 1,736 were eligible for consideration of causality, and 163 of these studies were included in the quantitative synthesis. The extent of this literature indicates that a significant amount of empirical research has been conducted on this topic. Table 2 summarizes the evidence for each Bradford Hill criterion as described in the narrative and systematic reviews presented in this supplement. The table also adds ratings of the evidence from three recent reviews not included (Smith & Foxcroft, 2009; Jernigan et al., 2017; Stautz et al., 2016) because of their focus on longitudinal and experimental studies. A review of these studies will also be covered in the forthcoming Cochrane review. Because the recent reviews of longitudinal and experimental studies are highly relevant to the criteria of strength of association, temporal precedence, and consistency, they are included here in our concluding causality assessment.

The table gives evidence of causality for all nine of the Bradford Hill criteria. In some reviews, multiple criteria have been met. The table also shows that these review articles are based on substantial empirical research that has been conducted in a variety of countries.

Strength of the association, dose–response, temporal precedence, and consistency

One of the most important Bradford Hill criteria is the strength of the association, which can be measured statistically in terms of relative risk (for dichotomous outcomes) or Cohen’s d (for continuous ones). In contrast to the p value, in which there is general agreement on the .05 cutoff level, there are no widely agreed-upon categories for strength of relative risk; most scientists would agree that the association between smoking and lung cancer is strong (adjusted relative risk > 10; i.e., smokers are at more than 10 times greater risk for lung cancer than non-smokers) and the association with breast cancer (adjusted relative risk = 1.3) is modest or weak. We would consider a relative risk that is less than 2.0 to be modest and a relative risk of 2 to less than 10 to be moderately strong. For continuous outcomes, Cohen recommended a rule of thumb: .2 was considered modest, .5 moderate, and .8 strong.

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
for the outcome. Another important aspect of causality is to demonstrate that the exposure precedes the outcome. This criterion places a premium on longitudinal research in which marketing exposures are measured before the onset of alcohol use. Longitudinal observational studies typically assess marketing exposure in a cohort of adolescents, starting with never drinkers during childhood or early adolescence who are followed over time to evaluate the association between exposure at baseline and onset of drinking. Some studies begin with adolescents who have not engaged in hazardous drinking but who may be experimenting with alcohol and examine the transition to hazardous or binge drinking.

Smith and Foxcroft (2009) published a review of seven longitudinal studies that followed 13,000 young people ages 10–26 years and evaluated a range of alcohol advertising and marketing exposures. Most of the studies suffered from attrition bias, but all demonstrated “significant effects across a range of different exposure variables and outcome measures” (p. 7). Reviews such as these speak to consistency of effect across a heterogeneous group of studies, an important Bradford Hill criterion. The authors concluded the data from these studies suggested a modest association between exposure and subsequent alcohol consumption in young persons.

Jernigan et al. (2017) conducted a systematic review of longitudinal studies published after the Smith and Foxcroft (2009) review that examined exposure to advertising and drinking among underage persons. All 12 studies found a positive association between marketing exposure and one or more alcohol consumption outcomes. For initiation of alcohol use the odds ratios for different marketing exposures ranged from 1.00 to 1.69, and for subsequent hazardous or binge drinking, the range was somewhat higher: 1.38 to 2.15. Thus, a review of longitudinal studies published after 2009 offered the same conclusion—evidence of a modest effect of alcohol marketing, with consistency across heterogeneous measures of marketing exposure and drinking.

The review of cross-sectional studies by Finan et al. (2020) presents mixed results. In general, the authors report more evidence for a positive relationship between alcohol marketing exposure and alcohol use behavior among adolescents and young adults than negative or null evidence. For example, of the 38 studies reviewed comprising 32 differ-

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**Table 1. Bradford Hill criteria: Definitions and application to alcohol marketing research**

| Criterion                    | Definition                                                                 | Application to alcohol marketing exposures                                                                 |
|------------------------------|---------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------|
| Strength of association      | The stronger the association between the exposure and the clinical outcome, the less likely it is influenced by an external variable or confounded by a variable associated with the exposure and outcome of interest. | How strong is the association between exposure to alcohol marketing and changes in alcohol consumption compared to other neurobiological, psychological, and behavioral correlates of drinking behavior? |
| Dose–response relationship   | If a dose–response relationship can be observed for the cause-and-effect hypothesis, increased exposure will proportionally impact the clinical outcome. | Does risk of alcohol consumption increase monotonically with higher levels of exposure to marketing or marketing receptivity? |
| Temporal association         | Is there evidence that the presumed cause precedes the effect in time?      | Did the exposure to alcohol marketing precede early onset of drinking and progression to binge drinking? Is the association between exposure to alcohol marketing and drinking reciprocal? |
| Consistency                  | The credibility of a finding increases if different investigators can replicate it across different locations, with different populations, and under different circumstances. | Have there been multiple observations of alcohol marketing effects across multiple media, in multiple countries, as reported by different investigators using a variety of exposure measures and covariate controls? |
| Specificity                  | Causality can be established when one type of exposure leads to one specific outcome. | Is the association between alcohol marketing exposure and substance use confined only to drinking, or does it also include other behaviors like smoking? Is exposure to other marketing inputs (e.g., food) associated with higher risk of drinking? |
| Plausibility                 | There is stronger support for causality if there is a likely biological and/or psychological mechanism that can explain the association between exposure and the outcome. | Is it biologically plausible that changes in neurobiological responses and psychological processes can account for the association? Do mediational analyses confirm psychological theory? |
| Experimental evidence        | If experimental manipulation of the exposure-outcome association impacts the outcome, this represents very strong support for causation. | Is there experimental evidence that relies on randomization of marketing exposure or instrumental variables to rule out third variable explanations? |
| Coherence                    | Causality between an exposure and a health outcome is supported when the association is coherent with current knowledge of the health condition. Conflicting or lack of supporting evidence would count against coherence. | Are there documented examples of youth alcohol use increasing without marketing exposures or decreasing with them? If so, does this empirical evidence conflict with a causal interpretation? |
| Analogy                      | If an exposure factor similar to A leads to a clinical outcome similar to B, then this analogy counts as evidence in support of our hypothesis that A causes B. | Are the effects of exposure to alcohol marketing similar the results of research on exposure to tobacco marketing? |

*Adapted from McDonald and Strang (2016).*
Table 2. Bradford Hill criteria addressed mainly and secondarily in 11 review articles

| Author/review topic | Jernigan & Ross/ alcohol marketing landscape | Weitzman & Lee/ alcohol and tobacco similarities | Jackson & Bartholow/ psychological processes | Courtney et al./ neuropsychological studies | Henehan et al./ youth cognitive responses | Finan et al./ cross-sectional studies | Noel et al./ digital alcohol marketing | Saffer/ econometric studies | Smith & Foxcroft (2009)/ longitudinal studies | Jernigan & Ross (2017)/ longitudinal studies | Stautz et al. (2016)/ experimental studies |
|---------------------|--------------------------------------------|-----------------------------------------------|---------------------------------------------|------------------------------------------|----------------------------------------|-------------------------------------|-----------------------------------------|--------------------------------|------------------------------------------|--------------------------------|------------------------------------------|
| Number of studies or (references) | (70) | (97) | (120) | (133) | 22 | 38 | 25 | 17 | OECD countries |
| Number of countries | N.A. | N.A. | N.A. | N.A. | 6 | 15 | 8 | 7 | 3 |
| Number of subjects | 13,255 | 35,219 | |
| Bradford Hill criterion | | | | | | | | | |
| Strength of association | M+ | M+ | M+ | M+ | M+ | M+ | M+ | M+ | M+ |
| Dose–response relationship | S+ | S+ | S+ | S+ | S+ | S+ | S+ | S+ | S+ |
| Temporal precedence | S+ | M+ | S+ | M+ | S+ | M+ | S+ | M+ | M+ |
| Consistency of association | S- | S- | M+ | S+ | S+ | S- | S+ | S- | |
| Specificity of association | S+ | | | | | | | | |
| Plausibility | M+ | M+ | M+ | M+ | |
| Experimental evidence | S+ | S+ | S+ | S+ | |
| Coherence | S+ | M+ | |
| Analogy | M+ | |

Notes: Supported (+); not supported (-). M refers to the main conclusion of a systematic review that supports (+) or does not support (-) one of the Bradford Hill principles; S refers to a secondary conclusion of the review that is supported by citations and the description of evidence from other scientific research that supports (+) or does not support (-) one of the Bradford Hill principles. N.A. = not applicable; OECD = Organisation for Economic Co-Operation and Development. This review used statistical significance and whether the association was positive or negative. For most drinking outcomes, the association, if significant was positive. In only two studies was it negative; but in one third, it was null.

ent associations between marketing exposures and lifetime drinking outcomes, the authors found 21 positive relationships compared with 11 null association relationships and only a handful of negative relationships—more evidence for a modest association. They also found that relationships for alcohol promotion and owning alcohol-related merchandise exposures were more consistently positive than for other advertising exposures. As the authors note, methodological issues make it difficult to review, evaluate, and summarize cross-sectional findings.

Similarly, in Noel et al.’s (2020) review of the literature on digital marketing, which comprises studies using cross-sectional, longitudinal, and experimental designs, the strength of associations across the 25 studies was mixed. Nevertheless, the findings support the conclusion of a modest positive association between engagement with digital alcohol marketing and increased alcohol consumption as well as increased binge or hazardous drinking behavior. In addition, their review showed that liking or sharing an advertisement on social media or downloading alcohol-branded content was positively associated with alcohol use, whereas the effects of simple exposure to digital alcohol advertising were inconclusive.

Based on the cumulative evidence, we conclude that the strength of association, temporal precedence, and consistency criteria have been met but that more research is needed to establish dose–response relationships.

Specificity and independence

Even when an association is moderately strong and statistically significant, the ability to draw causal inferences is limited if the study does not adjust for confounding, which occurs when exposure and drinking behavior are both influenced by an unmeasured third variable that accounts for the association. It is important to distinguish between confounders and mediators, which are statistically identical. A confounder is a variable associated with both the exposure and the outcome but independent of the causal pathway from exposure to behavior. A mediator is on the causal pathway—a variable that is set in motion by the exposure and contributes indirectly to the effect of the exposure on behavior. Psychological constructs that lie on the theoretical pathway between advertising exposure and drinking (e.g., alcohol expectancies) should not generally be modeled as covariates but as mediators in order to test theoretical models that shed light on mechanisms.

Parenting styles, peer and family drinking, and personality traits such as sensation seeking have all been found to increase the risk of underage drinking. To the extent that they also increase exposure to advertising, not accounting for them could cause us to reach a spurious conclusion. Fortunately, alcohol advertising has also been shown to be independent of many of these confounders in the cross-sectional and longitudinal studies cited above. For example, one study
(Stoolmiller et al., 2012) found a longitudinal association between ownership of alcohol-branded merchandise (marketing receptivity) and drinking onset as well as binge drinking after accounting for age, sex, race/ethnicity, parental education, family income, alcohol use by parents and peers, poor school performance, sensation seeking, rebelliousness, parenting effectiveness, weekly spending allowance, television viewing, and exposure to alcohol in movies.

Jackson and Bartholow (2020) note that much of the support for an association between alcohol-related marketing and youth drinking is based on prospective cohort studies that adjust for potential interpersonal-level (parent, peer influence) and individual-level (sociodemographics, sensation seeking) confounders. These studies lend credence to the argument that marketing exposure is a causal factor in drinking behavior. Not only does this review make a strong case for independence of association, but it also supports several plausible psychological mechanisms that could be responsible for causal associations between alcohol-related marketing and youth drinking. The fact that modest associations are found across multiple studies, each of which adjusts for a somewhat different set of covariates, is a very strong indicator of the robustness and consistency of the association.

The review of econometric studies by Saffer (2020) points out the limitations of older research (Nelson, 1999) that could not adequately rule out a third unmeasured causal variable, with a focus on minimizing the possibility of a third variable effect. In econometric language, endogeneity is the unmeasured third factor. Endogeneity could involve reverse causality (demand for alcohol can prompt more advertising) or factors that work at the individual level. Endogeneity is a concern with any marketing assessment that goes in the direction from product consumption to heightened awareness of advertising for the product. For example, engagement in online marketing is not only a measure of “exposure” to such advertising, but it also is often a measure of the degree to which an individual is engaged in the consumption. Saffer highlights several recent econometric studies (e.g., Molloy, 2016) that used instrumental variables analysis to ensure the exogeneity of the marketing exposure measure and found a modest association. These studies go a long way to assure us that the modest strength associations for cross-sectional and longitudinal studies listed above are real.

Few of the studies tested explicitly for specificity of the marketing message. This would be demonstrated if alcohol marketing exposure predicted binge drinking but not other risk behaviors, such as smoking. To the extent risk behaviors cluster, this could be difficult to demonstrate. Hill emphasized that many exposures cause multiple diseases, such that this type of specificity should not be seen as an absolute prerequisite to causality, and the same is probably also true with marketing effects on risk behaviors.

However, it has been possible to show that alcohol marketing is associated with drinking, independent of exposure to other marketing inputs. For example, Morgenstern et al. (2011) showed that alcohol marketing receptivity was associated with drinking independent of receptivity to marketing for other products (e.g., candy and mobile phones), and Tanski et al. (2015) showed that the association between receptivity to television alcohol advertising and onset of drinking and binge drinking was independent of receptivity to fast food advertising.

**Plausibility**

One indicator of the maturity of an area of research (e.g., marketing research) is its ability to articulate plausible conceptual models and theoretical explanations based on cumulative findings. Plausibility refers to whether an association has a credible empirical or theoretical basis in terms of biological, psychological, or social mechanisms. Plausibility was explored directly in two of the reviews in this supplement, and indirectly in several others, on the psychological and the biological levels of analysis.

In recent years, psychologists have developed and tested theoretical models in which marketing exposures are hypothesized to affect psychological mediators relating to thoughts, cognitions and attitudes. These marketing-induced changes are hypothesized to predict whether an individual will engage in drinking behavior. Jackson and Bartholow (2020) provide a narrative summary of psychological plausibility using an integrated conceptual model that depicts relevant psychological processes as they work together in a complex chain of influence. The evidence suggests that perceptions of others’ behaviors and attitudes in relation to alcohol (social norms) may be a more potent driver of youth drinking than evaluations of drinking outcomes (expectancies). Their review suggests how the mechanisms of action work and helps explain the different strategies used by the alcohol industry either intentionally or indirectly in the complex world of alcohol marketing, such as frequent exposure to promote familiarity and evaluative conditioning, a tactic that pairs a more familiar object with a less familiar product to influence the perception of the new product.

Henehan et al. (2020) review research on youth cognitive responses to advertising to evaluate underlying theories that might explain the association between marketing exposures and alcohol use by youth. Cognitive responses to alcohol advertising were found to be complex, with many factors modifying the association, including differences by age, experience with alcohol, and alcohol advertising content. This explains the need for segmentation and specification of different types of alcohol advertising.

These authors also point out developmental considerations. For example, younger populations tend to be less skeptical of advertising claims than are older adolescents. In addition, youth who reported prior alcohol use liked alcohol advertisements more than their peers with less or no alcohol
use history. Not surprisingly, youth preferred image advertisements that portrayed the appeal of drinking lifestyles rather than quality advertisements that featured product attributes such as ingredients or taste.

The authors also describe how the research supports a threshold effect, which is one of five “empirical generalizations” advertisers consider both highly valid and important. The greatest response to advertising is from the initial exposures. As the number of cumulative exposures increases, the strength of association between further exposure and behavior declines.

Biological plausibility is another area of research that has advanced significantly because of functional magnetic resonance imaging, a technique for measuring brain activity. Functional magnetic resonance imaging and other neurobiological methods have been used to test how young people respond to alcohol marketing compared with other marketing inputs and whether those neural responses relate to their own real-world drinking. Courtney et al. (2020) reviewed the marketing literature from the perspective of adolescent brain development, reward sensitization and risk taking. The results are remarkable from a plausibility perspective and provide a neurobiological basis for the neurological mechanisms identified by Jackson and Bartholow (2020) and Henehan et al. (2020). The evidence suggests that responses in prefrontal–reward circuitry establish alcohol advertisements as reward-predictive cues that may reinforce alcohol consumption upon exposure.

This circuitry is especially important during adolescent development, when youth are particularly susceptible to social and reward cues, which are defining characteristics of many alcohol advertisements. Courtney et al. (2020) suggest that early drinking is influenced by an imbalance in the relative maturation of brain circuits that may modulate susceptibility to alcohol advertisements and risky behaviors associated with drinking. In addition, social motivation and peer influence exacerbate cue sensitivity and reinforce drinking behaviors via the rewarding properties of the action itself (e.g., drinking and pleasure) and conformity to social norms of peer groups. Not only does the neuroscience help to explain the initiation of alcohol use in adolescents, but the authors also suggest that the experience of intoxication reinforces continued drinking as reward circuitry becomes sensitized to alcohol advertising cues, which may ultimately contribute to patterns of risky alcohol use (e.g., binge drinking episodes), reinforcing the notion that the relation between marketing exposure and drinking behavior is bidirectional.

Despite the inability of cross-sectional studies to prove causality, Finan et al. (2020) note the importance of this research for theory development, which is amply illustrated in several of the other supplement reviews in terms of plausible explanatory models that are supported by substantial research. Perhaps the greatest evidence for plausibility, which is circumstantial rather than scientific in nature, is the information presented by Jernigan and Ross (2020) describing the modern development of the alcoholic beverage industry and its concentration into a small number of large producers on a global level. These transnational corporations have increased their marketing expenditures, especially in the low- and middle-income countries, and in digital and social media. Both digitally and geographically, they are exposing and in some cases targeting the large population of young consumers, as well as women. Although the industry does not share its marketing research, huge investments are being made on the basis of informed expectations of a significant return on investment.

We conclude that there is now overwhelming evidence—from multiple studies that have explored mechanisms of action on the level of psychological processes, neurobiological influences, and adolescent development—that the association between exposure to alcohol marketing and youth drinking is plausible.

**Experimental evidence**

A recent meta-analysis (Stautz et al., 2016) of 24 experimental studies (involving 1,363 young persons) has addressed the immediate effects of alcohol marketing communications and media portrayals on alcohol consumption and cognition. This review concluded that there was a modest effect of alcohol marketing on immediate drinking (Cohen’s $d = 0.20$, 95% CI [0.05, 0.34]). The authors found no immediate effect for exposure to alcohol in films (Cohen’s $d = 0.16$). Thus, experimental evidence using randomized designs also supports a modest relation between exposure to alcohol advertising inputs and immediate drinking, consistent with cross-sectional and longitudinal observational studies.

In this supplement, the review by Noel et al. (2020) describes research from randomized controlled trials as well as eight prospective cohort studies of digital marketing strategies. Other reviews also reported the results of experimental studies, although many of these were focused on more narrow outcomes such as the cognitive (Henehan et al., 2020), psychological (Jackson & Bartholow, 2020), and neurobiological (Courtney et al., 2020) correlates of alcohol marketing effects.

**Coherence**

Coherence with current knowledge of the health risk condition (i.e., youth alcohol consumption and binge drinking), is difficult to estimate from the studies that have been conducted. Conflicting or lack of supporting evidence would count against coherence.

If alcohol marketing were responsible for a large share of youth drinking, it is argued that youth drinking would show increases over time. In other words, increases in youth
drinking would be coherent with the notion that the relation between alcohol marketing and youth drinking is causal. However, in the United States, youth drinking has been declining for three decades. In fact, as marketing expenditures have increased globally, alcohol consumption by youth has declined in high-income countries (de Looze et al., 2015). Although the decline in youth drinking is not coherent with the evidence of a large causal association, it is not inconsistent with a modest association, along with competing influences from regulatory factors (e.g., the United States during this period raised the alcohol purchase age from 18 to 21 years) as well as macroeconomic factors and changes in parental practices that influence youth drinking (Vashishtha et al., 2019). Because most of the research has been conducted in high-income countries, it is possible that the effects could be much stronger in low- and middle-income countries, where controls are weak and exposures are increasing.

**Analogy**

The analogy criterion applies when an analogous causal relationship exists between a similar agent or exposure, such as tobacco, and a similar behavior or disorder, such as early initiation of smoking. The main support for the analogy criterion comes from the review article by Weitzman & Lee (2020), which evaluates the similarities between alcohol and tobacco advertising and their associations with adolescent attitudes and behaviors. Dr. Weitzman participated as an expert witness on tobacco marketing and youth smoking in the Racketeer Influenced and Corrupt Organizations trial (U.S. v. Philip Morris, 2006), which found that tobacco companies conspired to hide the adverse health effects of their products and targeted adolescents with their marketing campaigns.

The similarities are numerous, for example, the positive perceptions of tobacco and alcohol that develop after children are exposed to cartoon characters and animal mascots (e.g., Joe Camel and Budweiser frogs), and the impact of brand-specific image advertising on youth. The association between tobacco marketing and youth smoking has been tested with numerous cross-sectional and longitudinal studies, and the association is modest but consistent across studies, just as it is with alcohol.

Analogy is not typically considered a strong criterion for causal inference, but the similarities between tobacco and alcohol marketing provide a clear case for invoking analogy in this case. If one believes the U.S. Centers for Disease Control and Prevention’s Surgeon General Statement (National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2012) that tobacco marketing is one cause of adolescent smoking, why would this not apply to billion-dollar image advertising campaigns developed by the same creative talents that developed cigarette image advertising campaigns, involving similar themes, communicated through the same media channels that reach millions of adolescents, and directed at a different risk behavior (alcohol use) with a similar time of onset in the life course?

*Why is the association modest and not strong?*

There are many reasons for the general finding that all marketing effects are modest in strength. Studies of marketing are not designed to measure the cumulative effects of marketing over the 10 years an individual spends in adolescence. Marketing inputs are ubiquitous; they begin in early childhood. Typical middle school students are exposed to two to four alcohol advertisements per day, and some are exposed many more times than that (Collins et al., 2016). Moreover, as discussed by Saffer (2020), the relation between marketing exposure and behavior is bidirectional—it is a reciprocal process that would be very difficult to capture and piece out in its entirety. Alcohol marketing exposure prompts a cognitive response, which changes how much the individual notices and responds to subsequent exposure. As pointed out by Courtney, et al. (2020), it is also likely that initiation of alcohol use changes the way the individual responds to marketing. Ultimately, marketers expect this bidirectional process to lead to an affective response (liking) and, thence, to the choice of a favorite brand—the marketing “holy grail.” Marketing studies pick up only bits and pieces of this process; therefore, it is not surprising that the effects are modest. But one can conceive how the cumulative impact on behavior over the entire period of adolescence might be very large indeed.

Effect size is often limited by how the marketing exposure is modeled. Observational studies of marketing tend to focus on only one or two media channels, not all the channels available to modern marketing campaigns. Take, for example, a Heineken campaign built around use of the product in a James Bond movie. The integrated campaign rolls out with the release of the movie, which shows the lead actor using the product. This is paired with a television advertising campaign that promotes both the product and the movie (and perhaps the other products placed in the movie). There would also typically be billboard, point-of-purchase, and social media components to the campaign. A study of social network marketing would rarely measure exposure to television advertising at the same time, thus missing the scope of the modern integrated marketing campaign. Studies that do measure multiple channels often make the mistake of having these exposures compete with each other in a multivariable analysis, which causes correlated exposures to cancel each other out. Instead, marketing studies should work toward measures that treat exposure from separate channels as measured components of a latent construct, an approach that would likely show a larger association with behavior.

Last, in many countries marketing is neither directed at nor expected to affect the entire population, which may
dilute the overall relationship between marketing and population drinking.

Summary

Our judgment is that when marketing research is assembled and evaluated according to the Bradford Hill criteria, there is persuasive evidence that exposure to alcohol marketing is one cause of drinking onset during adolescence and also one cause of binge drinking. Our judgment is based on the best available evidence, which—although international in scope and drawn from almost all of the world’s geographic regions—is largely the result of a small group of U.S.-based investigators. To the extent that this is the case, there is a need for the same literature to be reviewed by a larger panel of public health experts in order to reach a broader consensus at the level of national and international organizations, such as the U.S. National Academies of Sciences, Engineering, and Medicine and the World Health Organization. It should be noted that the World Health Organization (2017) already considers restrictions on alcohol advertising to be an effective and cost-effective intervention for reducing harmful use of alcohol.

Why Does Causality Matter? Implications of a Scientific Consensus on Causal Associations

Why does causality matter?

Causality matters because of the scope of the exposure. As reported in the scoping review by Jernigan and Ross (2020), global alcohol sales totaled more than $1.5 trillion in 2017, with increased spending in parts of the world (Asia, Africa, Latin America) with the least controls on marketing and the highest proportions of children and adolescents who are likely to be exposed. Because alcoholic beverage production and marketing is concentrated in a small number of firms with huge economic power, the industry has been successful in resisting statutory controls on advertising in favor of industry self-regulation (Jernigan & Ross, 2020). The sheer amount of spending on advertising makes it impossible in most countries to shield vulnerable populations from this level of marketing activity.

Causality matters because of the need to protect vulnerable populations. Recently, a broader concept of vulnerability has been applied by public health professionals (Babor et al., 2017) to the analysis of marketing effects. Developmental theory and marketing research suggest that groups defined by younger age, incomplete neurocognitive development and a history of personality disorder may be particularly vulnerable because of their increased susceptibility to alcohol marketing and the disproportionate harm they experience from alcohol. For example, children may be more susceptible to media imagery because they do not have the ability to compensate for biases in advertising portrayals and glamorized media imagery.

To the extent that both theory and empirical research suggest that populations defined by age and developmental history may be particularly susceptible to alcohol marketing, there are grounds for strengthening the protections used in many countries that limit exposure to potentially harmful marketing content.

Causality matters because this conclusion forms the basis for action in real life. In Hill’s (1965) words, “in passing from association to causation I believe in ‘real life’ we shall have to consider what flows from that decision. On scientific grounds we should do no such thing. The evidence is there to be judged on its merits and the judgment (in that sense) should be utterly independent of what hangs upon it—or who hangs because of it . . . . [O]ur object is usually to take action. If this be operative cause and that be deleterious effect, then we shall wish to intervene to abolish or reduce [its impact on] death or disease” (p. 300). Causality matters because it becomes the basis for common sense governmental restrictions on alcohol marketing, restrictions that should aim to reduce the exposure to (or its impact on) vulnerable populations through effective implementation.

How do corporations respond to causality statements?

It has become increasingly clear that there is a corporate strategy to oppose scientific studies that link a corporate product with disease and that could undermine a successful business model (Oreskes & Conway, 2010). The strategy was developed by the tobacco industry over the past 50 years and involves treating the scientific findings as a public relations problem: emphasize scientific uncertainty, hire scientists to counter the claim, hire lobbyists to harass the real scientists, create dark-money organizations to influence public opinion, and lobby against government regulation at all costs (World Health Organization, 2018). For evidence of the ultimate harm of such a strategy, just look at the inability of the world to address climate change in the face of overwhelming evidence that fossil-fuel consumption is causing it.

The main response of the alcohol industry to the mounting evidence of the impact of alcohol advertising has been to use the same strategy as the tobacco industry—to question the validity of the evidence, promote the implementation of industry self-regulation measures, and promote “responsible-drinking” campaigns as a way to prevent or reduce harmful drinking. Just as the tobacco industry did two decades ago, the alcohol industry alleges that its marketing efforts direct consumer attention toward particular brands but do not encourage more drinking in any segment (Beer Institute, 2015).

The alcohol industry can be expected to continue to advocate for its own ability to regulate its marketing communications. These so-called self-regulation programs are based on voluntary codes developed and enforced by the
alcohol industry, which violates its own self-regulation codes; evaluations of these programs have indicated they are ineffective (Noel et al., 2017; Noel & Babor, 2017). Responsible-drinking messages and campaigns in the United States are primarily directed by the major alcohol producers, trade associations, and industry-funded social aspects/public relations organizations. They fail to take into account the considerable scientific literature pointing to the risks associated with different levels of alcohol consumption; further, they do not define the limits of lower risk drinking or the health benefits of abstinence from alcohol.

What are the policy implications of a causal statement?

Government agencies—indeed, independent from industry—should restrict alcohol marketing exposures in the adolescent population. Policy on national and international levels should take into account the influence of alcohol marketing on the developing brain, adolescent cognitive development and alcohol use itself. Courtney et al. (2020) indicate that because adolescence is a sensitive window of brain development, alcohol consumption during this period may interfere with normative neuro-maturational processes, which may result in a cascade of neurocognitive impairments and increase the likelihood of later alcohol and substance dependence. Their model suggests that alcohol advertising may inordinately appeal to adolescent consumers. Enacting policies that shield youth from alcohol advertising until they emerge from this sensitive window of development (around age 21 years) could be a particularly effective strategy for preventing early alcohol abuse. Although statutory bans can be circumvented (Gallopel-Morvan et al., 2017), research suggests they are far more effective than voluntary codes (Pantani et al., 2017) for reasons indicated above.

Legislation restricting or banning alcohol advertising is a well-established policy measure used throughout the world to protect children and adolescents, despite opposition from the alcohol industry (World Health Organization, 2017). However, in most countries, advertising restrictions are piecemeal in nature, applying only to certain beverages (e.g., distilled spirits), certain hours of television broadcasting, or specific media (e.g., television, radio, print, and outdoor billboards). With the increasing amounts of marketing now conducted through digital formats and social media, which are even less regulated, youth exposure to alcohol marketing is increasing in many parts of the world, and it is virtually impossible to control within a nation’s borders because of the global reach of alcohol marketers. This situation speaks to the need for an international agreement along the lines of the United Nations’ Framework Convention on Tobacco Control (World Health Organization, 2005). In its Regional Plan to Reduce Harmful Use of Alcohol, the Pan American Health Organization (2011), a regional office of the World Health Organization, recommended that countries encourage statutory regulation to restrict or ban the marketing of alcoholic beverages to youth and other vulnerable groups and designate a government agency to enforce marketing regulation. A subsequent “Technical Note” (Pan American Health Organization, 2017) referred to the Framework Convention on Tobacco Control’s total ban on tobacco marketing across all signatory countries as a model for a legally binding international Framework Convention on Alcohol Control, as did The Lancet (2007) editors a decade earlier.

Government bodies should track alcohol use and alcohol harms in the population at large and respond to unhealthy trends in other vulnerable groups. As an example, recent increases in all-cause mortality have been particularly striking among middle-age women, and increasing mortality from alcoholic cirrhosis of the liver is contributing to this increase (Woolf & Shoomaker, 2019). It would be reasonable to ask whether the renewal of the marketing of distilled spirits in the mid-1990s and the industry’s targeting of women have contributed to this trend in alcoholic cirrhosis.

Global implications for research and theory

The Centers for Disease Control and Prevention or the Office of the Surgeon General should sponsor a series of reports on alcohol and health, similar to the ones that have been published on tobacco. Given that alcohol is responsible for almost 100,000 deaths a year in the United States alone and more than $200 billion in costs—80% of which are borne by individual families and government (Sacks et al., 2015)—it is unfortunate that there have been few reports on underage drinking and none on alcohol marketing. As mentioned in the introduction to this supplement, the Surgeon General reports provide a forum for scientists who study alcohol to come together, collect the scientific literature, and offer judgments on the relation between alcohol consumption and disease as well as on the relation between corporate practices and harmful drinking behavior. The Surgeon General reports have been enormously helpful in guiding tobacco-control policies, and they would be similarly helpful in guiding American efforts in alcohol control. Another vehicle for U.S. policy would be to commission a study of alcohol marketing to be conducted by the National Academies of Sciences, Engineering, and Medicine, along the lines of its 2018 report on alcohol-impaired driving (National Academies of Sciences, Engineering, and Medicine, 2018).

The U.S. National Institute on Alcohol Abuse and Alcoholism (NIAAA) should resurrect its program to fund research on alcohol marketing and vulnerable populations. Just as the Centers for Disease Control and Prevention’s Surgeon General reports helped form the basis for public health policy, efforts by the National Cancer Institute to fund research into tobacco and disease and tobacco marketing and youth smoking formed the bases for some of the judgments within those
Henehan et al. (2020) suggest reasons why some research was funded, in part, through an NIAAA program of research that was active up to May 2014 (PA-11-015; https://grants.nih.gov/grants/guide/pa-files/pa-11-015.html). The articles in this supplement provide a valuable chronology of the evolution of research methods, the strengths and weaknesses of different research designs, and the need for improved theory and continued study. In light of this, it is disappointing that alcohol marketing research is no longer a programmatic priority at NIAAA (Helman, 2018). In fact, NIAAA staff have been reported to actively discourage investigators from submitting proposals on alcohol marketing (Begley, 2018). In a conversation one of us (JS) had in 2015, he was told that even if his application was to score in the second percentile (top 2% of scores), it would have a poor chance of being funded because of altered institutional research priorities.

The following points suggest an agenda for the next generation of marketing studies, as drawn from the various reviews.

- Henehan et al. (2020), in their evaluation of psychological theories, suggest that future studies use nonlinear methods to assess the association between advertising and cognitions, avoid the tendency to measure alcohol advertising as a uniform and dose–response exposure, use theoretical frameworks, and control more carefully for confounding bias.
- Results presented in the review by Jackson and Bartholow (2020) suggest that cultural orientation influences adolescent drinking and this influence is mediated partially through cultural orientation influences on adolescent drinking expectancies and self-efficacy. Future research should be conducted in non-Western countries, such as China and Vietnam, where the impact of alcohol advertising on cultural orientation can be evaluated as a mediating factor.
- Courtney et al. (2020) propose directions for future research that would extend previous neurobiological studies and test specific hypotheses targeting characteristics of alcohol advertisements that appeal to adolescents and factors promoting sensitivity to alcohol cues.
- Henehan et al. (2020) suggest reasons why some research has demonstrated small or marginal effects of alcohol advertising. On the basis of the evidence from psychological research, they hypothesize that advertising effects should be strongest in populations that are primed for the first time with positive images about alcohol use. Once these positive expectations have been created, additional exposures will have a marginal impact. This suggests that studies need to be conducted in younger populations that have little exposure to prior alcohol use and in study populations in countries in which advertising is just beginning to increase and should take into consideration the nonlinear association between advertising exposure and alcohol expectancies.
- Jackson and Bartholow (2020) propose that careful consideration of how alcohol exposure is operationalized (marketing vs. entertainment media, traditional vs. digital media, industry sponsored vs. user generated, in vivo vs. cumulative exposure, simple dosage effects vs. stages of personal involvement) is crucial for future research, as is precision regarding the outcomes under investigation (intention, initiation, consumption, heavier use, problems).
- Given the increasing importance of digital marketing, and the results of initial studies that it is associated with youth drinking, there is a need to conduct multiwave prospective cohort studies that can apply path models to test the link between marketing receptivity, attitudes and drinking behavior onset within a hypothesized causal chain.
- Given the tendency of marketing campaign messages to be integrated across many media channels, studies of exposure and behavior should capture as many channels as possible and determine if these can be analyzed in an integrated way. One recent study suggested it may be possible to integrate these exposures into a latent construct (Gabrielli et al., 2019).

Ethical implications

Another area of concern is the ethical training of psychologists and other behavioral and social scientists who are often hired by the tobacco and alcohol industries to design their marketing campaigns. In addition, advertisers and marketers routinely use evidence generated in basic psychological research (e.g., on attitude formation and consumer behavior) to design campaigns. If the intention of advertising and marketing is to instill positive evaluations of advertised products, thereby encouraging intentions to purchase and ultimately consume or use those products, the evidence presented in this supplement suggests that there is a strong need for more rigorous ethical training in our graduate programs for psychologists, social scientists, and business majors.

Conclusion

We judge as scientists that the evidence presented in this supplement is sufficient to conclude that alcohol consumption by youth is affected by exposure to alcohol marketing. In arriving at this conclusion, we have used the Bradford Hill criteria as a heuristic device for assessing causation in a broad epidemiological framework that draws evidence from many disciplines, many countries and many methods. Modest positive effects have been found in a range of countries, and plausible psychological, neurobiological, and other mechanisms have been identified as possible mediating factors, thus giving further support for a causal association. We hope that the results will serve to generate thoughtful discourse among researchers, effective prevention measures among policymakers, and an effort to reach consensus on...
this issue among a larger and more representative body of scientists.

Conflict of Interest Statement

The authors have no competing interests to declare.

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