

COMMENTARY

Thirty years of research show alcohol to be a cause of intimate partner violence: Future research needs to identify who to treat and how to treat them

ABSTRACT

Research over the past 30 years has demonstrated that excessive alcohol use meets all of the epidemiological criteria for causality. While neither a necessary nor a sufficient cause, excessive alcohol use does contribute to the occurrence of partner violence and that contribution is approximately equal to other contributing causes such as gender roles, anger and marital functioning. Current theories of how excessive drinking results in partner violence provide a potentially valuable framework with respect to who should be targeted for interventions with respect to alcohol-related partner violence and what those interventions should address.

Key words: alcohol, intimate partner violence.

Is excessive alcohol consumption a cause of intimate partner violence (IPV)? Thirty years ago, Leonard and Jacob [1] reviewed the relatively scant research and described the many methodological weaknesses and gaps in the literature that precluded a clear answer to this question. While there were still some gaps in the literature, in 2005 we asked ‘when can we say that alcohol is cause of aggression?’ [2]. Our argument was that the answer involved a judgment using the epidemiological criteria of causality. Based on the breadth and consistency of methodologically diverse studies, our judgment was that alcohol was a contributing cause. In contrast, some argued that there was not sufficient evidence to attribute a causal role to alcohol [3].

Over the past 10 years, increasingly sophisticated studies have continued to support excessive drinking as a contributing cause of violence and of partner violence. The cross-sectional relationship has been demonstrated on every continent [4,5]. It has been reported in health-care settings [6], alcoholism treatment samples [7] and batterer samples [8]. Meta-analyses focused solely on studies of IPV have found a significant moderate association [9], an association that is nearly as strong as attitudes condoning violence or traditional sex roles [10].

Prospective studies have found that male drinking patterns, either as a main effect or in an interaction, are predictive of subsequent IPV over a 1- to 2-year period among newlyweds [11], new parents [12], young married couples (e.g. [13,14]), domestic violence offenders [15] and men in treatment for alcohol use disorders [16]. Studies examining the relationship between drinking and

violence at the daily or event level have found that alcohol use is more common in severely aggressive versus less severely aggressive events [17] and that perpetrator alcohol use in the preceding 4 h predicts both verbal and physical aggressions [18]. Laboratory studies have consistently found an effect of intoxication on analogues of human aggression, [19] although the majority of these studies have only examined male to male aggression. Crane, Godleski, Przybyla, Schlauch and Testa [20], however, found a significant effect of alcohol in a meta-analysis of all alcohol-aggression experiments in which alcohol was administered to men and aggression toward a woman was assessed.

Does alcohol contribute to the occurrence and severity of IPV? In our opinion, the answer is an unequivocal ‘yes’. Whether alcohol is framed as a trigger, a contributing cause or a factor that increases severity, it is difficult to argue that excessive alcohol use has no impact on violence. We need to refocus our research on how to reduce alcohol-related IPV. The questions that we should be asking are ‘what are the processes that we should direct our interventions toward’, and ‘to whom should we direct these interventions?’. Although the answers to these questions have not yet been empirically studied, our current theoretical understanding of the mechanisms underlying the causal influence of alcohol provides guidance with respect to the development of interventions.

Theories of how excessive drinking might cause aggressive behaviour have, from the beginning, been theories of moderated causation. Any causal influence must be moderated by person and situation variables

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because it is patently obvious that excessive drinking does not result in aggression in all people under all circumstances. Cognitive disruption theories called attention to the balance of instigation and inhibition as a critical issue. Taylor and Leonard [21] posited that alcohol intoxication's impact on aggression resulted from an impaired capacity to attend to the instigative and inhibitory cues in a situation and that 'when instigative cues are dominant, an intoxicated person would be likely to focus on these dominant cues and therefore, be more likely to react aggressively' (p. 96), while aggression would not be expected when inhibitory cues are dominant. Steele and Josephs [22] formalised this position as 'alcohol myopia' and observed that alcohol would have its largest impact under situations of 'inhibition conflict, in which a response provoked by salient, strong cues is also inhibited by other strong cues that require further processing to grasp (p. 923)'. While these theories focused on instigatory and inhibitory cues, the interpretation of these cues occurs within an individual, and therefore, individual differences in the perception, interpretation and reaction to such cues would also play a role. This concept is apparent in Fals-Stewart, Leonard and Birchler's [23] position that 'IPV occurs when an individual's aggression threshold is exceeded; that is, when the strength of the aggressive motivations exceeds the strength of the inhibitions', that 'there are multiple thresholds because there is assumed to be a higher threshold for severe aggression than nonsevere aggression' and that 'alcohol is likely to have its greatest impact on those who are slightly below an aggression threshold when sober' (p. 240). This approach to 'aggressive motivations' and 'inhibitions' was viewed as a person-environmental cue perspective.

Taken together, these positions suggest that alcohol's impact on IPV differs according to the balance of instigating and inhibiting forces. In the case of individuals with very low levels of instigating characteristics (e.g. trait anger and neuroticism) and high levels of inhibition (e.g. empathy and self-regulation), episodes of excessive drinking are unlikely to lead to aggression except perhaps in the presence of strong provocation. As a result, any policy or intervention with respect to alcohol is unlikely to impact the very low aggression rates in such individuals, although excessive drinking may have other adverse effects. At the other end of the spectrum, individuals with high levels of instigating characteristics and low levels of inhibition are likely to exhibit aggression frequently whether they are drinking or not, and alcohol's impact is likely to be one of increasing the severity of the aggression. Interventions directed as reducing alcohol use, therefore, may be useful in impacting severity and the potential for injury. However, without addressing the instigatory and inhibitory factors, reducing or eliminating excessive drinking would be

unlikely to eliminate the occurrence of aggression, and may not impact the frequency of aggression overall among such individuals. From this perspective, alcohol's greatest impact would be among individuals with moderate levels of instigatory and moderate levels of inhibitory factors. To be clear, such individuals are not at the greatest risk for violence or severe violence, but alcohol is more likely to be a significant factor in predicting the frequency and severity of violence in this group. Addressing excessive drinking in this group would be a key component of treatment. However, because such individuals have other factors that lead to violence, and because the chance for relapse to heavy drinking is significant, developing interventions to decrease the instigating and increase the inhibiting factors is also of importance.

This brief exposition cannot address a number of key issues that are raised by this approach to intervention. First, there are many potential instigatory and inhibitory factors, although anger/hostility and inhibitory control are two with empirical support [24,25]. Research that helps to identify the critical and most potent instigating and inhibiting factors is needed. Second, while there are well-developed interventions for anger, effective interventions for other instigatory and inhibitory factors need to be developed and tested. Third, while the role of alcohol is fairly clear, the potential impact of other drugs and the combination of alcohol with other drugs are less clear. Finally, although our focus has been on the psychopharmacological impact of alcohol, we recognise that emerging research suggests that alcohol may also serve as a cue that activates aggressive thoughts [26]. Understanding how this process may interact with the psychopharmacological impact of alcohol and other instigating and inhibiting factors may suggest additional interventions.

Fundamentally, however, we believe that we need to move beyond the argument as to whether alcohol is or is not a cause of IPV. We should be examining the processes by which alcohol facilitates the occurrence or severity of IPV and these examinations would be most fruitful in the context of interventions addressing both heavy drinking and instigating/inhibiting factors. Testing interventions derived from these cognitive disruption theories should not be viewed as enshrining these theories as the final explanation for the alcohol-violence effect. However, we have reached a point in the field in which we should be examining the potential causal processes in the context of interventions to reduce and eliminate partner violence.

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