Acute Alcohol Effects on Inhibitory Control and Implicit Cognition: Implications for Loss of Control Over Drinking

Matt Field, **Reinout W. Wiers**, **Paul Christiansen**, **Mark T. Fillmore**, and **Joris C. Verster**
School of Psychology (MF, PC), University of Liverpool, Liverpool, UK

Department of Psychology (RWW), University of Amsterdam, Amsterdam, The Netherlands

Department of Psychology (MTF), University of Kentucky, Lexington, Kentucky

Faculty of Science (JCV), Section Psychopharmacology, Utrecht University, Utrecht, The Netherlands.

Abstract

Alcohol impairs inhibitory control, and it alters implicit alcohol cognitions including attentional bias and implicit associations. These effects are seen after doses of alcohol which do not lead to global impairments in cognitive performance. We review studies which demonstrate that the effects of alcohol on inhibitory control are associated with the ability of alcohol to prime alcohol-seeking behavior. We also hypothesize that alcohol-induced changes in implicit alcohol cognitions may partially mediate alcohol-induced priming of the motivation to drink. Based on contemporary theoretical models and conceptualizations of executive function, impulsivity, and the motivational salience of alcohol-related cues, we speculate on other aspects of cognition that may underlie alcohol's effects on alcohol seeking. Inconsistencies in existing research and priorities for future research are highlighted, including dose effects and the potential interactions between chronic heavy drinking and the acute effects of alcohol on these cognitive processes.

Keywords

Inhibitory Control; Attentional Bias; Automatic Alcohol Associations; Priming

ACUTE ALCOHOL INTOXICATION PRODUCES PSYCHOLOGICAL CHANGES THAT HAVE IMPORTANT CONSEQUENCES

Alcohol intoxication is associated with a number of well-characterized changes in various aspects of psychological function. These include changes in subjective mood states and feelings of intoxication as well as impairments in psychomotor performance and cognitive processes such as memory, divided attention, and planning (Eckardt et al., 1998; Holloway, 1995; Hull and Bond, 1986; Weissenborn and Duka, 2003). Alcohol intoxication also has negative consequences outside of the laboratory. For example, alcohol increases sexual risk-taking (George and Stoner, 2000; Halpern-Felsher et al., 1996), aggressive behavior (Hull and Bond, 1986; Ito et al., 1996), and the risk of motor vehicle accidents (Koelega, 1995). This review is focused on a specific and well-known consequence of alcohol intoxication: increased consumption of alcohol or "loss of control" over alcohol-seeking behavior, known

as the alcohol priming effect (de Wit, 1996). We aim to demonstrate how alcohol-induced impairment in a few fairly specific aspects of cognitive processing may underlie the alcohol priming effect.

ACUTE ALCOHOL EFFECTS IN THE LABORATORY

Broadly, alcohol impairs cognitive and psychomotor functions (see Eckardt et al., 1998; Fillmore, 2007; Holloway, 1995; Hull and Bond, 1986). In this section, we argue that inhibitory control, a specific aspect of cognitive function, is impaired at doses that do not lead to a global impairment in cognitive performance. Similarly, alcohol may increase certain types of automatic alcohol-related cognitions at doses which do not lead to a general slowing or impairment of cognitive performance.

Although performance on most cognitive and psychomotor tasks is impaired at fairly high doses of alcohol, the dose of alcohol required to produce some degree of impairment varies for different psychological functions. Complex tasks, such as divided attention tasks, are impaired at fairly low doses (those producing a blood alcohol concentration, or BAC, around 0.01% or slightly higher; Moskowitz and Fiorentino, 2000), whereas much higher doses (BACs around 0.08%) are required to produce a slowing of simple reaction time (Fillmore, 2007; Holloway, 1995). As reviewed elsewhere (Field et al., 2008b; Fillmore, 2003, 2007), selective effects of alcohol administration have been reported at doses that do not lead to global cognitive impairment, as described later.

Firstly, alcohol impairs inhibitory control. Inhibitory control can be defined as the ability to inhibit a motor response that has already been initiated, and it represents a core component of executive functioning, alongside mental set shifting and working memory (Miyake et al., 2000). Deficient inhibitory control is also a component of "impulsivity," alongside delay discounting (Olmstead, 2006; de Wit, 2009). Various laboratory tasks have been developed to assess inhibitory control, including the Stop Signal and Cued Go/No-Go tasks. For example, in the Cued Go/No-Go task (e.g., Abroms et al., 2003), "Go" and "No-Go" visual targets are presented on a computer screen, and participants are instructed to rapidly respond to the former but inhibit responding to the latter. At the beginning of each trial, participants are presented with a cue that signals the probability of occurrence of the different targets, before the Go or No-Go targets are presented. For example, if a vertically oriented rectangular cue is presented, this signals an 80% likelihood that the subsequent target will be a Go target (and therefore only a 20% chance that the target will be a No-Go target), whereas if a horizontally oriented rectangular cue is presented, this signals an 80% likelihood that the subsequent target will be a No-Go target. The primary dependent measure in the task is the number of failures to inhibit responding to No-Go cues when those cues were preceded by an invalid cue, i.e., a cue that signaled an 80% probability that a Go target would be presented.

A number of studies have demonstrated that a moderate (0.4–0.45 g/kg) dose of alcohol, which produces BACs around 0.06%, reliably impairs inhibitory control on the Stop Signal and Cued Go/No-Go tasks (Marczinski et al., 2005; de Wit et al., 2000). Such doses also impair performance on other tasks that assess inhibitory control over attention (Abroms and Fillmore, 2004; Abroms et al., 2006). Importantly, the inhibitory control impairment at this BAC is not usually accompanied by impaired accuracy or speed of responding to "Go" targets in these tasks, which suggests that this disruption to inhibitory control is fairly selective, and it is not simply part of a global disruption of psychomotor performance.

Alcohol intoxication also increases the salience of alcohol-related cues. The extent to which alcohol-related cues are able to "grab the attention" of research participants (hereafter referred to as "attentional bias") can be assessed with a number of experimental tasks,

including the visual probe task. In this task, a pair of pictures is presented side by side on a computer monitor; one of the pictures depicts an alcohol-related scene (e.g., a close-up photo of an individual drinking beer), the other picture does not. Participants' eye movements can be recorded while pictures are presented, to measure how long participants spend attending to the two pictures. Immediately after pictures are removed from the screen, a small probe stimulus is presented in the spatial location that had been occupied by one of the pictures, and participants are instructed to make a rapid manual response whenever they see the probe. Probe detection latency can then be used as an additional measure of attention, because if participants are faster to respond to probes that replace alcohol-related pictures rather than control pictures, this implies that they were looking at the alcohol-related picture at the time of picture offset.

Studies using this task and other measures of attentional bias have demonstrated that heavy but not light drinkers have an attentional bias for alcohol-related cues (see Field and Cox, 2008; for a review). With regard to acute alcohol effects, two studies (Duka and Townshend, 2004; Schoenmakers et al., 2008) demonstrated that, among heavy social drinkers, a small dose of alcohol (0.3 g/kg; producing BACs around 0.04%) increased attentional bias for alcohol-related cues, relative to placebo. However, Duka and Townshend (2004) reported that a higher dose of alcohol (0.6 g/kg, producing BACs around 0.08%) did not increase attentional bias relative to placebo, which suggests that alcohol does not dose dependently increase attentional bias; instead, only low doses of alcohol seem to increase attentional bias. Furthermore, above a certain dose threshold, alcohol may actually *decrease* attentional bias. Schoenmakers and Wiers (2010) conducted a naturalistic study in a bar in the Netherlands, in which they examined the relationship between attentional bias and the amount of alcohol consumed in the same evening. They found that, in individuals who had been binge drinking, there was a negative correlation between self-reported drinks consumed and the magnitude of attentional bias.

Finally, experimental tasks have been used to probe the nature of automatic alcohol-related associations in heavy drinkers. For example, the Implicit Association Test is a speeded reaction time task that measures the strength of associations between alcohol-related words and concepts such as positive (vs. negative) valence, or arousal (vs. sedation). A conceptually similar task is the Stimulus-Response Compatibility task, which measures the speed at which research participants can direct symbolic approach (vs. avoidance) responses to alcohol-related pictorial cues. Results from studies utilizing these measures of implicit (or automatic) alcohol-related associations have demonstrated that presentation of alcohol-related pictorial cues or words tends to automatically activate concepts related to approach, arousal, and positive affect in heavy drinkers, but not in light drinkers (e.g., Field et al., 2008a; Palfai and Ostafin, 2003a; Wiers et al., 2002; see Wiers et al., 2007; for a review). Some studies have examined the effects of alcohol administration on these tasks. Palfai and Ostafin (2003b) reported that a priming dose of alcohol producing BACs around 0.04% increased the accessibility of positive, but not negative alcohol-related associations. A more recent study (Farris and Ostafin, 2008) demonstrated that alcohol increased the strength of associations between alcohol and approach concepts, relative to predrink baseline. However, there was no placebo control condition in this study, and participants chose how much alcohol to drink (rather than being given a set amount), so it is not possible to estimate average BACs from this study. A further study, which did include a placebo control condition (Schoenmakers et al., 2008), found that alcohol (0.3 g/kg; BACs around 0.04%) had no effect on alcohol-approach associations in a sample of heavy drinkers, despite this dose increasing attentional bias for alcohol cues.

We stress this as an important topic for future research because, theoretically, alcohol should increase the accessibility of appetitive alcohol associations. According to a recent model

(Wiers et al., 2007), automatic appetitive alcohol cognitions develop after chronic alcohol exposure through a sensitization process. This sensitization process should lead to strengthening of automatic appetitive alcohol cognitions among heavy drinkers, and the degree to which these associations are strengthened should be dependent on the number of experiences with alcohol over the lifetime. Therefore, even relatively light or moderate drinkers should show some strengthening of appetitive alcohol associations, but the associations should be much stronger in heavier drinkers. This effect is likely to be genetically moderated, as indicated, for example, in a recent study which found that heavy drinking students carrying a g-allele in the OPRM1-gene showed stronger alcohol-approach tendencies than equally heavy drinking students without a g-allele in the same gene (Wiers et al., 2009). Importantly, these cognitive changes should be apparent in heavy drinkers, even when sober, but acute alcohol intoxication should increase their strength even further, and this effect of acute alcohol should be disproportionately large in heavy drinkers because of the underlying long-term sensitization process. Clearly, additional research is required to clarify the acute effects of alcohol on measures of automatic alcohol associations.

SPECIFIC COGNITIVE PROCESSES MAY UNDERLIE ALCOHOL-SEEKING/ LOSS OF CONTROL DURING INTOXICATION

Alcohol administration leads to an increase in alcohol-seeking behavior. This "priming effect" was initially demonstrated in alcohol-dependent patients (Bigelow et al., 1977; Hodgson et al., 1979; Ludwig and Wikler, 1974), but it has been demonstrated in nondependent social drinkers as well (Rose and Duka, 2006; de Wit and Chutuape, 1993), even after very low doses of alcohol (0.25 g/kg, yielding BACs below 0.04%; de Wit and Chutuape, 1993). Subjective alcohol craving is similarly affected by administration of alcohol. Participants' beliefs that they are consuming alcohol (or not) may be a more important determinant of the priming effect than the alcohol content of the drinks consumed (Marlatt et al., 1973). However, the effects of an alcohol priming dose on both alcohol-seeking behavior and subjective craving tend to be dose dependent (Duka et al., 1999; Rose and Duka, 2006; de Wit and Chutuape, 1993). In this section, we argue that the priming effect is at least in part a consequence of alcohol-induced impairments in inhibitory control and changes in automatic alcohol cognitions.

According to several theoretical models (Goldstein and Volkow, 2002; Jentsch and Taylor, 1999; Wiers et al., 2007) two important causal factors in drug-seeking behavior are an increase in the salience of drug-related cues or approach tendencies elicited by those cues, and a failure or inability to exercise control over drug-seeking behaviors. The idea that alcohol abuse represents a loss of self-control has been a longstanding concept in the theory and treatment of addictions, particularly alcohol abuse disorders. E. M. Jellinek and Mark Keller, who pioneered research on alcoholism, did much to promote the control concept in alcohol abuse. Most notable was Jellinek's notion of "loss of control" whereby an alcoholic's initial consumption of alcohol triggers an uncontrollable urge to consume more alcohol, leading to a binge. Although the loss of control concept has been replaced by less extreme views of "reduced" or "impaired" control, many treatments for alcohol abuse today still advocate abstinence because of the suspicion that any alcohol consumption may result in a "loss of control" over drinking behavior (Jellinek, 1952; Keller, 1972).

In this section, we propose that impaired inhibitory control and changes in automatic alcohol cognitions play a key role in the alcohol priming effect. This proposal is similar in many ways to the myopia model of alcohol effects (Steele and Josephs, 1990), which proposes that alcohol-induced cognitive distortions can explain many of its effects, including changes in mood and aggressive behavior. Briefly, the myopia model proposes that alcohol leads to a narrowing of the attentional focus (myopia), such that dominant cues become the center of

attention and peripheral cues are ignored. This is particularly apparent when peripheral cues are present that encourage suppression of potentially undesirable behaviors; according to the myopia model, the individual fails to respond to these inhibiting cues when intoxicated. Although there is much empirical evidence in support of the myopia model (a review of which is beyond the scope of this study, but see Hull and Slone, 2004, 2006; Sher and Wood, 2005; Sher et al., 2005), several lines of evidence do not appear to support the model. For example, alcohol's effects on inhibition of peripheral cues may be more robust than alcohol-induced narrowing of attention for neutrally valenced visual cues (Bartholow et al., 2003). Furthermore, recent research on mind wandering suggests that acute alcohol can make people *less* able to focus on material that is the focus of attention, rather than more able (i.e. myopic), particularly if the to-be-focused material is not particularly compelling (Sayette et al., 2009). As detailed later, we specifically propose that alcohol consumption makes people myopic for alcohol cues, while simultaneously reducing their ability to inhibit their responding. Our proposal is that these distinct effects of acute alcohol consumption have additive and possibly interactive effects on alcohol-seeking behavior.

Results from one recent study suggest that alcohol-induced impairments in inhibitory control are related to alcohol-seeking behavior. Weafer and Fillmore (2008) examined the effects of alcohol (0.65 g/kg) on inhibitory control, and they then examined the relationship between the magnitude of alcohol-induced impairment and ad-lib alcohol consumption. As in previous studies (e.g. Abroms et al., 2003), they found that, relative to placebo, alcohol administration led to a significant increase in the number of failures to inhibit responding to invalidly cued No-Go cues during a Cued Go/No-Go task. Importantly, there was substantial between-subject variation in the magnitude of this impairment. In a subsequent testing session, participants were provided with access to four different types of beer and were instructed to consume as much beer as they wanted in the context of a bogus "taste-test." The central finding from the Weafer and Fillmore (2008) study was that the magnitude of alcohol-induced impairment in inhibitory control was significantly positively correlated with ad-lib beer consumption; the former explained 20% of the variance in the latter.

This is an important finding as it demonstrates a clear association between the effects of alcohol on inhibitory control, and alcohol-seeking behavior. To our knowledge, this is the only study of its kind, but it does suggest some interesting possibilities for future research. For example, the study did not investigate whether alcohol-induced failures of response inhibition are associated with *alcohol-induced increases* in alcohol-seeking behavior, because alcohol-seeking behavior was only tested when participants were sober, in the Weafer and Fillmore (2008) study. More broadly, the psychological concept of "impulsivity" encompasses both response inhibition and "delay discounting," or the preference for immediate gratification at the expense of longer-term gain (e.g., Olmstead, 2006). Acute alcohol effects on delay discounting have been investigated in several studies, but these studies have yielded inconsistent results. Two studies reported no differential effects of alcohol and placebo on delay discounting (Dougherty et al., 2008; Richards et al., 1999), one found increased delay discounting after alcohol compared to placebo (Reynolds et al., 2006), and one study found the opposite, i.e. increased delay discounting after placebo compared to alcohol (Ortner et al., 2003).

No previously published study has investigated ad-lib drinking/alcohol-seeking behavior in the context of an investigation into the effects of alcohol on delay discounting, but we suggest this as a fruitful area for future research: we would hypothesize that alcohol-induced increases in delay discounting should be positively correlated with alcohol-seeking behavior. Similarly, response inhibition is an important component of executive cognitive function, a set of abilities that also include working memory and mental set shifting (Miyake et al., 2000). Both working memory (Givens, 1995; Givens and McMahon, 1997; see Holloway,

1995; for a review) and mental set shifting (Lyvers and Maltzman, 1991) are impaired after alcohol administration, and more abstract aspects of executive function such as planning are similarly impaired (Weissenborn and Duka, 2003). We suggest that individual differences in alcohol-induced impairment of these cognitive functions might be correlated with ad-lib drinking or other indices of alcohol-seeking behavior after administration of a priming dose of alcohol.

Turning to measures of the motivational salience of alcohol-related cues, no published studies have investigated whether alcohol-induced increases in attentional bias or automatic alcohol associations are associated with the ability of alcohol to prime alcohol-seeking behavior. However, some of the evidence does suggest that there may be an association between the two. Firstly, as previously discussed, low doses of alcohol increase attentional bias for alcohol cues, and there is some evidence that alcohol also increases the strength of associations between alcohol and approach or positive valence. As with the previously discussed literature on alcohol effects on aspects of executive function and impulsivity, it is possible (and indeed likely) that there are marked individual differences in the effects of alcohol on measures of the motivational salience of alcohol cues. Given recent findings which suggest that attentional bias and automatic alcohol associations may have a causal influence on alcohol-seeking behavior (for reviews, see Field and Cox, 2008; Wiers et al., 2007), our tentative hypothesis is that those individuals who show a large increase in attentional bias/automatic alcohol associations after alcohol (relative to placebo) will also show the largest increase in alcohol-seeking behavior after administration of alcohol, relative to placebo.

Figure 1 illustrates the cognitive mechanisms that may underpin the alcohol priming effect and summarizes the arguments put forward in this section. To briefly recap, we suggest that administration of alcohol preloads increases the strength of automatic appetitive responses to alcohol cues, including attentional bias and the strength of associations between alcohol and concepts such as approach and positive valence. Alcohol preloads also impair response inhibition. Both of these effects may independently contribute to the alcohol priming effect (increased drinking, or loss of control over alcohol-seeking behavior). Moreover, these two processes may interact, such that alcohol-induced impairment of inhibitory control may make the individual less able to resist alcohol-induced increases in attentional bias and other automatic appetitive responses to alcohol cues. Although this latter interaction has not been studied in the context of acute alcohol effects, recent studies have demonstrated that (stable) individual differences in executive control (working memory or Stroop-interference) moderate the impact of automatic associations on drinking behavior over periods of weeks or months (Grenard et al., 2008; Houben and Wiers, 2009; Thush et al., 2008). Specifically, the association between drinking behavior and automatic alcohol associations is far stronger in individuals with relatively poor executive control abilities. If *trait* executive function moderates the impact of automatic alcohol associations on drinking behavior, then one might reasonably expect that a *state* manipulation of executive function (such as that produced by acute alcohol effects) might produce similar moderation effects.

Additional research findings suggest two potentially important factors that may moderate the effects of alcohol on the aforementioned aspects of cognitive processing. Firstly, the dose of alcohol administered seems to be important. In terms of impaired inhibitory control, reliable effects of alcohol administration are seen at doses ranging between 0.4 and 0.8 g/kg (Fillmore, 2007). However, in terms of alcohol effects on attentional bias, the only study to investigate effects of different doses of alcohol found that a low dose of alcohol (0.3 g/kg) led to increased attentional bias (relative to that seen after placebo), but a higher dose of alcohol (0.6 g/kg) led to no significant increase in attentional bias compared to after placebo; furthermore attentional bias after 0.6 g/kg was significantly lower than that seen after 0.3 g/

kg (Duka and Townshend, 2004; see also Schoenmakers and Wiers, 2010). These dose–response effects await confirmation in future research, but they may suggest that alcohol-induced increases in attentional bias could mediate alcohol priming effects on alcohol seeking at relatively low BACs, but not at higher BACs; conversely, alcohol-induced impairments in inhibitory control may mediate alcohol priming effects on alcohol seeking only at higher BACs. In other words, after low doses of alcohol, the incentive-motivational properties of alcohol may be bolstered, and this may partially account for increased alcohol seeking after alcohol; however, after higher doses of alcohol, the incentive-motivational properties of alcohol cues may be a less important determinant of alcohol-seeking behavior, and instead alcohol-induced impairment of inhibitory control might be the most important factor that determines subsequent alcohol-seeking behavior. Furthermore, one theoretical model suggests that impaired inhibitory control may only influence alcohol-seeking behavior if individuals are motivated to limit their drinking (Wiers et al., 2007), although the potential moderating role of the motivation to limit drinking has not yet been empirically investigated.

The second apparent moderator of alcohol effects on these distinct aspects of cognitive function is individual differences in drinking habits or alcohol abuse problems. One might expect that heavier drinkers, or those with a longer duration of alcohol exposure over the lifetime, should become tolerant to alcohol's effects on cognitive function, given that tolerance develops to a number of other effects, for example its anxiolytic effects (e.g., Lipscomb et al., 1980; see Sher and Wood, 2005; Sher et al., 2005 for reviews). However, with regard to alcohol's effects on inhibitory control, the reverse appears to be true: individuals who binge drink frequently (and who also consume more alcohol overall) are *more* sensitive to the effects of acute alcohol administration on response inhibition than participants who binge less frequently (Marczinski et al., 2007). This suggests a somewhat counterintuitive interaction between long-term drinking patterns and acute alcohol effects on response inhibition, with heavier drinkers perhaps hyper-sensitive to the impairing effects of alcohol intoxication. With regard to acute alcohol effects on the motivational salience of alcohol cues, no previous studies have investigated possible moderation of acute alcohol effects by previous experience with alcohol, although theoretically one might expect that long-term heavy drinking would render individuals hypersensitive (rather than tolerant) to the ability of acute alcohol administration to increase the salience of alcohol-related cues (Robinson and Berridge, 1993). Again, we highlight this as an important topic for future research.

CONCLUSIONS AND FUTURE DIRECTIONS

There is a need to clarify the psychological mechanisms that underlie loss of control over alcohol consumption as a consequence of acute intoxication. We reviewed evidence which shows that impairment of inhibitory control is seen after administration of moderate doses of alcohol and this impairment is associated with increased alcohol-seeking behavior. We have also shown that low doses of alcohol also lead to increases in the incentive-motivational properties of alcohol cues and according to several theoretical models, this could be an additional cognitive mechanism involved in loss of control over alcohol consumption when intoxicated. Finally, we highlight several important directions for future research, such as examining the role of alcohol-induced changes in other aspects of executive function and impulsivity in loss of control, the need to consider whether different aspects of cognition are differentially sensitive to different priming doses of alcohol, and the need to consider how individual differences in drinking history might moderate these effects.

Acknowledgments

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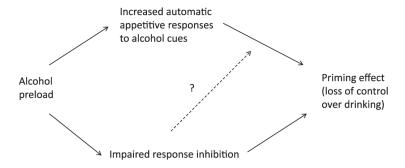
Fig. 1.
Schematic illustrating cognitive mechanisms that may underlie the alcohol priming effect. Administration of alcohol preloads increases the strength of automatic appetitive responses to alcohol cues, including attentional bias and the strength of associations between alcohol and concepts such as approach and positive valence. Alcohol preloads also impair response inhibition. Both of these effects may independently contribute to the alcohol priming effect (increased drinking or loss of control over alcohol-seeking behavior). In addition, the dashed line indicates a theoretically predicted interaction between these effects of alcohol preloads. Inhibitory control may normally act to reduce the influence of automatic appetitive reactions to alcohol cues on alcohol-seeking behavior; because inhibitory control is impaired by alcohol preloads, this moderating effect is diminished. As a consequence, automatic appetitive responses to alcohol cues have a greater influence on alcohol-seeking behavior after administration of an alcohol preload.