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Protracted impairment of impulse control under an acute dose of alcohol: A time-course analysis

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HIGHLIGHTS

- Inhibitory control was tested at 3 time points following a dose of alcohol.
- No acute tolerance to alcohol was found for inhibitory control.
- Inhibition was impaired even 5 h after drinking, when BACs were near zero.

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ABSTRACT

Alcohol is well-known for impairing impulse control as well as its disruptive effects on other aspects of behavioral functioning, such as motor control. Time-course analyses during a single dose show rapid development of acute tolerance to impairment of motor coordination, reaction time, and levels of subjective intoxication, but no acute tolerance to impairment of the ability to inhibit responses. Evidence for a possible lag in tolerance development to the impairing effects of alcohol on inhibitory control suggests that, as drinkers' blood alcohol concentration (BAC) declines, they might exhibit prolonged impulsivity despite having an unimpaired ability to initiate action. The present study extended the time-course analysis to examine the recovery of inhibitory control under a dose of alcohol as drinkers' BAC descended from a peak of 80 mg/100 ml to a zero level. Twenty-four healthy adults were tested following 0.65 g/kg alcohol and a placebo in a counterbalanced order. They performed a cued go/no-go task that measured response inhibition. They also performed tasks that assessed reaction time, motor coordination, and completed ratings of their subjective levels of intoxication. Alcohol initially impaired inhibitory control, response time, and motor coordination and increased subjective ratings of intoxication. However, acute tolerance to the impairing effects of alcohol was observed for measures of response time, motor coordination, and ratings of intoxication and these measures returned to sober (i.e., placebo) levels by the time BAC fell to near zero. By contrast, impairment of inhibitory control showed no acute tolerance and remained impaired even when drinkers' BAC returned to near zero. Taken together, these results indicate that the disinhibiting effects of alcohol are present even when the impairing effects of alcohol on other aspects of behavior have diminished under the dose. These findings could provide a greater understanding of impulsive behaviors during the descending limb of intoxication.

1. Introduction

The prevalence of alcohol abuse in the United States has increased over the past decade despite considerable concern over its social costs. Alcohol use is particularly prevalent among young adults, with over half of men and women between 18 and 25 years of age reporting frequent alcohol use (Substance Abuse and Mental Health Services Administration, 2004). Moreover, the typical pattern of alcohol use reported by this demographic is often characterized by periods of heavy alcohol consumption referred to as "binges," which are usually defined as consuming five or more drinks during a single occasion (Wechsler & Nelson, 2001). There is growing evidence that acute changes in fundamental mechanisms of impulse control contribute importantly to the transition from social drinking to abusive drinking (e.g., Fillmore, 2003, 2007; Lyvers, 2000). As such, researchers have sought to gain a better understanding of how mechanisms of impulsivity operate to promote the abuse of alcohol.

One fundamental component of impulsivity concerns the ability to inhibit inappropriate or maladaptive actions or behaviors. Inhibitory control refers to the ability to inhibit a response that has already been instigated (see Logan & Cowan, 1984). This mechanism of behavior affords an individual control over where and when responses

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are expressed. Thus, the inhibition of behavioral responses is a necessary function for situations in which an individual needs to exert self-restraint and regulation over behavior. As such, deficits in inhibitory control have been implicated in a wide array of impulsive behaviors including heavy, binge drinking (e.g., Goudriaan, Grekin, & Sher, 2007; Marczinski, Combs, & Fillmore, 2007). Human laboratory studies have employed stop-signal and cued go/no-go models to evaluate behavioral control as the ability to quickly activate and inhibit prepotent (i.e., instigated) responses (Logan, 1994; Miller, Schaffer, & Hackley, 1991). These models are based on reaction time tasks requiring individuals to quickly activate a response to a go-signal and inhibit a response to stop or no-go signals. Studies have shown that these mechanisms of behavioral control are sensitive to the disruptive effects of alcohol. Indeed, alcohol increases inhibitory failures and slows response activation in a dose-dependent manner (Fillmore, Marczinski, & Bowman, 2005; Fillmore & Weafer, 2004). However, studies provide evidence that inhibitory mechanisms are more sensitive to alcohol's impairing effects compared with response activation. For example, studies have consistently found that inhibitory control is impaired at relatively low blood alcohol concentrations (BAC) that fail to slow response times (e.g., De Wit, Crean, & Richards, 2000; Fillmore & Vogel-Sprott, 1999).

Studies examining the speed with which behaviors recover from alcohol's impairing effects have also provided evidence of the sensitivity of inhibitory mechanisms to the drug's effects (e.g., Fillmore & Weafer, 2012; Fillmore et al., 2005; Ostling & Fillmore, 2010). The term tolerance refers to the observation that the intensity of a behavioral response to a drug diminishes with repeated administrations of the drug (Kalant, Leblanc, & Gibbons, 1971). Although alcohol tolerance can develop as a function of chronic, heavy consumption, it can also be observed following a single dose of alcohol. Acute tolerance refers to the diminished response to alcohol during the time-course of a single dose. This effect was first documented early last century by Mellanby (1919), who compared the intensity of alcohol impairment at a given BAC on the ascending and descending limbs of the blood alcohol curve. He observed that alcohol-induced ataxia in dogs was less intense at a given BAC during the descending versus the ascending limb of the BAC curve. This acute tolerance might be due to an adaptive process occurring during physiological exposure to the drug over time (e.g., Kalant et al., 1971).

In humans, acute tolerance to the impairing effects of alcohol has been observed for several behaviors such as motor coordination, reaction time, and subjective ratings of intoxication (Bierness & Vogel-Sprott, 1984; Fillmore & Vogel-Sprott, 1996; Fillmore et al., 2005; Marczinski & Fillmore, 2009; Schewiezer et al., 2004; Schweizer, Jolicoeur, Vogel-Sprott, & Dixon, 2004). In the past, acute tolerance was thought to develop uniformly across behaviors. However, several laboratory studies have failed to observe the development of acute alcohol tolerance for measures of inhibitory control (e.g., Fillmore & Weafer, 2012; Fillmore et al., 2005; Ostling & Fillmore, 2010). In one such study, Fillmore et al. (2005) compared the development of acute tolerance to the impairing effects of alcohol on response activation to the impairing effects on response inhibition. Participants performed the cued go/no-go task twice: once on the ascending limb and once on the descending limb of the BAC curve following 0.65 mg/kg alcohol. Both tests were performed at comparable BACs of approximately 80 mg/100 ml. The study showed that alcohol impaired behavioral activation by slowing reaction time and impaired response inhibition by increasing failures to inhibit responses to no-go targets. With regard to acute tolerance, the study found rapid recovery of behavioral activation. That is, reaction times measured on the descending limb of the blood alcohol curve had returned to sober levels. However, inhibitory control remained as impaired on the descending limb as it was on the ascending limb of the blood alcohol curve. Such findings show that inhibitory mechanisms are especially slow to recover from the impairing effects of alcohol.

Evidence that inhibitory control fails to recover from alcohol's impairing effects at the same rate as other behaviors begs the question of when impaired inhibitory mechanisms return to sober levels. Prolonged impairment of inhibitory mechanisms along the descending limb of the BAC curve could play an important role in the development of alcohol abuse. Drinkers might be prone to engaging in continued impulsive action even as BACs decline, such as resuming alcohol consumption, resulting in excessive binge drinking in a situation. No work has systematically extended the time-course analysis of the disinhibiting effects of alcohol along the BAC curve to determine when behavioral impairment might show full recovery. Thus, the present study compared the recovery of alcohol-induced impairment of inhibitory control with the recovery of other behaviors that have demonstrated acute tolerance to alcohol. The study employed an extended time-course approach to examine the recovery of inhibitory control, reaction time, motor coordination, and ratings of subjective intoxication following a dose of 0.65 g/kg alcohol as drinkers' BAC descended from a peak of approximately 80 mg/100 ml to a near-zero level. As a control, performance was also tested following a placebo dose. Consistent with our previous research (e.g., Fillmore et al., 2005; Ostling & Fillmore, 2010), it was hypothesized that reaction time, motor coordination, and subjective intoxication would display acute tolerance to the effects of alcohol, and that complete recovery would also be evident once BACs returned to near-zero levels. However, we predicted that there would be no evidence of acute tolerance for inhibitory control, and that given this lag in recovery, we might fail to observe complete recovery of this impairment as BACs approach zero.

2. Method

2.1. Participants

Twenty-four individuals (12 men and 12 women) between the ages of 21 and 29 (mean age = 23.2, *SD* = 2.6) participated in this study. Volunteers were recruited by flyers, posters, and newspaper/online advertisements seeking adults for studies of the effects of alcohol on cognitive functions. Volunteers were screened using health questionnaires and a medical history interview. Volunteers who reported any contraindication to alcohol, impaired cardiovascular functioning, seizure, head trauma, or central nervous system (CNS) tumors, were excluded from participation. Volunteers were also asked about past histories or present diagnoses of psychiatric disorder (i.e., Axis I, *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV, American Psychiatric Association, 2000)). Participants who reported a diagnosis of a DSM-IV Axis I disorder, past or present use of psychotropic medication, and/or past or present participation in counseling or therapy were also excluded from participation.

Volunteers had to report drinking at least once per month in an amount of at least two drinks to participate. Volunteers who reported alcohol dependence, as determined by a score of 5 or higher on the Short-Michigan Alcoholism Screening Test (S-MAST; Selzer, Vinokur, & Rooijen, 1975) were excluded from the study. Any other high-risk indicators of alcohol dependence, including prior treatment for an alcohol use disorder or conviction for driving under the influence also precluded participation. With regard to other drug use, the majority of the samples reported using caffeine (n = 20). Thirteen participants reported smoking cigarettes in the amount of less than a pack of cigarettes a day. Nine reported occasional past month use of marijuana on a less-than-weekly basis. No other drug use in the past month, including stimulants, opiates, or cocaine, was reported. Participants were in good health with no contraindications to drinking. The University of Kentucky Medical Institutional Review Board approved the study, and participants received $85.