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Research Report

Alcohol consumption impairs stimulus- and error-related processing during a Go/No-Go Task

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Abstract

Alcohol consumption has been shown to increase the number of errors in tasks that require a high degree of cognitive control, such as a go/no-go task. The alcohol-related decline in performance may be related to difficulties in maintaining attention on the task at hand and/or deficits in inhibiting a prepotent response. To test these two accounts, we investigated the effects of alcohol on stimulus- and response-locked evoked potentials recorded during a go/no-go task that involved the withholding of key presses to rare targets. All participants performed the task prior to drinking and were then assigned randomly to either a control, low-dose, or moderate-dose treatment. Both doses of alcohol increased the number of errors relative to alcohol-free performance. Success in withholding a prepotent response was associated with an early-enhanced stimulus-locked negativity at inferior parietal sites, which was delayed when participants failed to inhibit the motor command. Moreover, low and moderate doses of alcohol reduced N170 and P3 amplitudes during go, no-go, and error trials. In comparison with the correct responses, errors generated large response-locked negative (Ne) and positive (Pe) waves at central sites. Both doses of alcohol reduced the Ne amplitude whereas the Pe amplitude decreased only after moderate doses of alcohol. These results are consistent with the interpretation that behavioral disinhibition following alcohol consumption involved alcohol-induced deficits in maintaining and allocating attention thereby affecting the processing of incoming stimuli and the recognition that an errant response has been made.

Theme: Neural basis of behavior
Topic: Cognition

Keywords: ERP; ERN; Alcohol; go/no-go; N2; Ne; Pe; Attention

Alcohol consumption has long been known to affect behavior and cognition [36,49]. A number of surveys suggest that some effects of alcohol on cognition and behavior, ranging from increased sociability to heightened aggression, are related to the effect of the drug on brain mechanisms supporting mood and behavioral control [6,45]. This hypothesis has also been tested using standard cognitive tasks of inhibitory control. For instance, it has been demonstrated that moderate doses of alcohol can impair performance on tasks requiring a high degree of cognitive control to overcome habitual or prepotent responses [8,9,12].

One possible explanation for the effects of alcohol on tasks of inhibitory control is that alcohol affects one's capacity to allocate attention to the incoming stimulus [44] (i.e., during stimulus encoding and categorization) thereby making stimulus evaluation and stimulus-response mapping less efficient. It is also possible that alcohol introduces "noise" in the sensory processing streams to the extent that infrequent targets are not easily recognized and discriminated from other task-irrelevant stimuli. Alternatively, effects of alcohol consumption on behavior could be related primarily to deficits in inhibiting a prepotent response

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[16,17]. Alcohol may also impair an individual's ability to detect the occurrence of an error [39] and to successfully implement corrective action in response to the detection of an error. Thus, behavioral effects of drinking could occur at various levels of processing including stimulus categorization, stimulus-response mapping, response inhibition, error detection and correction.

Recording human event-related brain potentials (ERPs) is a powerful technique for characterizing the level at which alcohol might affect performance in tasks that tap behavioral inhibition, such as go/no-go tasks. These tasks require participants to make a speeded button press to targets while withholding responses to infrequent no-go stimuli. The successful inhibition of a prepotent response in such tasks generates large N2 and P3 waves peaking at about 250 and 325 ms post-stimulus, respectively, at midline central sites [5,14,15,26,28,35,37]. The N2 wave may index top-down mechanisms needed to suppress an incorrect tendency to respond on the no-go trials [5,15,28] or detect a conflict [37] between activated and required responses. The P3 wave recorded in a go/no-go task is often referred to as the no-go P3 to distinguish it from the P3b wave elicited by infrequent target stimuli. The no-go P3 is more centrally distributed than the P3b and is thought to index inhibitory processes needed to withhold a prepotent response [5].

Evidence from studies employing either the oddball or the flanker tasks suggest that the N2 amplitude is little affected by low or moderate doses of alcohol [39,40], whereas its latency, like response time, increased after alcohol consumption [29,40]. In comparison, the P3 wave appears to be consistently reduced after alcohol consumption [3,40]. To our knowledge, there are no studies that have examined the effects of acute alcohol on the N2 and P3 elicited during a go/no-go task. Presumably, if alcohol affects response inhibition then one would expect to see alcohol-related changes in no-go N2 and P3 waves. In addition, the go/no-go task generates a relatively high proportion of false alarms (i.e., observers failed to inhibit the habitual response), making it ideal to examine the effects of alcohol on error processing.

Errors in forced choice response tasks are associated with an Ne and Pe wave [13,20]. The Ne, also referred to as the error-related negativity (ERN), peaks at about 100 ms after the response and is maximal in amplitude over the midline fronto-central scalp region. The Ne increases in amplitude with the probability of error correction and with the degree of confidence that an error has occurred [41]. Some evidence indicates that the amplitude of the Ne is greater when individuals are aware that an error has been made [32], suggesting that the Ne might provide an index of internal monitoring of goal-directed behavior or the activation of a neural system supporting the detection that an error has occurred. A recent study shows that low-to-moderate doses of alcohol reduce Ne amplitude [39]. Reduction in Ne amplitude have also been found following the administration of the benzodiazepine oxazepam [10,25].

Here, we examine ERPs generated during a go/no-go task to investigate the attention allocation and inhibitory control accounts of alcohol effects on behavioral inhibition. Participants were presented with a series of single digits in the center of the screen and required to occasionally withhold the response to a target. Behavioral and electrophysiological data were collected prior to and following beverage consumption in three groups of participants. Following drug-free performance, participants were assigned randomly to either a control group that received orange juice, or to a group that received a low or moderate dose of alcohol. Consistent with the notion that alcohol impairs behavioral inhibition, we expected the number of errors to increase after alcohol consumption relative to drug-free performance. A comparison of stimulus-locked ERPs before and after alcohol consumption was used to elucidate whether the effect of alcohol on impaired response inhibition can be attributed to the effect of the drug on the allocation of attention and/or the ability to monitor performance and detect errors. Given that the amplitude of visual sensory evoked responses is smaller for unattended than attended stimuli [23,24,30,33,50], and assuming that alcohol impairs the allocation of attention, one might expect a smaller amplitude of sensory evoked response under the influence of alcohol. Furthermore, we hypothesized that if alcohol impairs behavioral control and response monitoring then the N2, P3, and the Ne waves should decrease in amplitude after alcohol consumption and this effect should be greater during no-go than during go trials since only the former requires response inhibition.

1. Methods

1.1. Participants

Twenty-four right-handed adults took part in the study. They were recruited through the subject pool at the Rotman Research Institute in Toronto and were instructed to fast for 2 h, and did not drink alcohol or take any other drugs for 16 h, prior to the session. All volunteers provided informed consent according to Baycrest Centre for Geriatric Care guidelines and were paid for their time. Participants were divided into three groups: control (N = 8, Mean age = 27.75, SD = 5.50, 4 males), low dosage (N = 8, Mean age = 24.13, SD = 2.03, 4 males) and high dosage groups (N = 8, Mean age = 26.75, SD = 4.40, 4 males).

1.2. Drinking habit questionnaire

Participants completed the Personal Drinking History Questionnaire (PDHQ) [48]. The PDHQ provides self-report measures of dose (mL of alcohol/kg consumed on an average drinking occasion), weekly frequency of drinking, and duration (hours) of a typical drinking session. The PDHQ also asks whether or not participants have experi-