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ScienceDirect
Vision Research 47 (2007) 1821–1832
Vision Research
www.elsevier.com/locate/visres

Alcohol slows interhemispheric transmission, increases the flash-lag effect, and prolongs masking: Evidence for a slowing of neural processing and transmission

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Received 5 December 2006; received in revised form 14 March 2007

Abstract

While the alcohol literature is extensive, relatively little addresses the relationship between physiological effects and behavioural changes. Using the visual system as a model, we examined alcohol's influence on neural temporal processing as a potential means for alcohol's effects. We did this by using tasks that provided a measure of processing speed: Poffenberger paradigm, flash-lag, and backward masking. After moderate alcohol, participants showed longer interhemispheric transmission times, larger flash-lags, and prolonged masking. Our data are consistent with the view that alcohol slows neural processing, and provide support for a reduction in processing efficiency underlying alcohol-induced changes in temporal visual processing.
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Keywords: Alcohol; Processing efficiency; Transmission efficiency; Interhemispheric transfer time; Flash-lag effect; Backward masking

1. Introduction

Alcohol has widespread systemic effects on the body, including the central nervous system (CNS), where it influences various components involved in neuronal transmission. For instance, alcohol modulates the synthesis, storage, release, and inactivation of neurotransmitters. The physiological effects are reflected in a variety of changes to behaviour and cognitive performance; including deficits in sensory and perceptual task performance (Ogden & Moskowitz, 2004), impaired motor coordination (Draski & Deitrich, 1996; Mangold, Laubli, & Krueger, 1996), and difficulties in the encoding and retrieval of learned information (Browning, Schummers, & Bentz, 1999). Many of the observed deficits associated with alcohol seem to be related to a breakdown in the ability to integrate information adequately in a way that would allow skilled actions to occur. For example, the reduction in driving performance comes about because of impairment in a variety of perceptual and motor systems, and the failure to process information adequately. Much of the literature, while providing excellent description of deficits that may occur, has not addressed the question of the possible mechanisms that might mediate these deficits.

One route to a better understanding of alcohol's effects is to look at correlations between behavioural and neural effects. This is best accomplished by using a model system for which there is some understanding of the neural basis of the behaviour. Because of the existing knowledge to date of the neural bases for various visual behaviours, the visual system is ideal for this purpose. Thus, in the present context, findings demonstrating effects of alcohol on some visual functions while sparing others should provide a means to isolate any selective effects of alcohol on neural processing.

One hypothesis proposed to explain some of the effects of alcohol is that it preferentially affects the speed of neural processing. In the context of the present study, we will use speed of neural processing to include the amount of time

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doi:10.1016/j.visres.2007.03.008

necessary for synaptic transmission, post-synaptic effects leading to action potential, and transmission of the signal along the axons. The proposal of a preferential effect is based on data that show an apparent slowing in processing. For example, moderate levels of alcohol decreases critical flicker fusion (CFF) rate for both central and peripheral viewing (Enzer, Simonson, & Ballard, 1944; Hill, Powell, & Goodwin, 1973; Pearson & Timney, 1998; Virsu, Kykka, & Vahvelainen, 1974), decreases sensitivity to temporal contrast (Pearson & Timney, 1998), and increases the temporal range of masking in a visual masking task (Jones, Chronister, & Kennedy, 1998; Moskowitz & Murray, 1976). Data from electrophysiological studies demonstrate that the latency of specific waveforms depicting neural activity increases after alcohol, in both humans and animals. For example, Bernhard and Skoglund (1941) found a diminution of the *a*-wave and a rise in the *b*-wave in the amphibian electroretinogram after alcohol, Ikeda (1963) found reduction in the response amplitude and latency of the *b*-wave component to a rapidly flickering light in the human electroretinogram, and van Norren and Padmos (1977) demonstrated a prolongation in the recovery of sensitivity to glare in the monkey ERG. In humans, the latency of the early components of VEP has generally been found to be less affected by alcohol than the later components (Colrain et al., 1993; Rohrbaugh et al., 1987). In contrast, the latencies of both early and late components of the waveform have been shown to increase after alcohol in the rat, cat, and monkey (DiPerri, Dravid, Schweigerdt, & Himwich, 1968; Erickson, Joe Willey, Riley, Fuster, & Lawrence, 1982; Hetzler, Oaklay, Heilbronner, & Vestal, 1982).

Both the behavioural and electrophysiological data demonstrate a detrimental effect of alcohol on processing speed. A possible mechanism underlying these observed changes may be a slowing in speed of neural transmission, and/or an increase in latency of neural processing. Using the visual system as a model, participants were tested in visual tasks that would provide a measure of processing speed before and after alcohol consumption: a Poffenberger task, visual backward masking, and a flash-lag task. It was expected that alcohol-induced reductions in processing speed would be reflected in perceptual changes consistent with a slowing of responsivity.

2. General method

2.1. Observers

All participants gave written, informed consent prior to their inclusion in each experiment. All had normal or corrected to normal vision, and no previous history of alcohol abuse. The procedures in each experiment were approved by the University Research Ethics Board for Health Sciences Research and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

2.2. Apparatus and stimuli

All stimulus generation and data collection were controlled by a VSG2/5F graphics board (Cambridge Research Systems) installed in a Pentium III PC. Stimuli were presented on the face of a 19″ Sony Trinitron Multiscan 17 SeII display monitor, and all stimulus presentation and data tabulation were under computer control.

2.3. General procedure

Each participant completed each experiment under two conditions, alcohol or no-alcohol, on separate days (separated by at least 24 h). Participants expected that alcohol might be consumed in both conditions. The orders of the alcohol and no-alcohol conditions were counterbalanced across participants. All testing began at either 10 am or 2 pm, and participants were asked to consume a light, low-fat meal approximately 2 h before testing to avoid adverse effects from consuming alcohol on an empty stomach. It should be noted that the no-alcohol condition should not be considered as a placebo condition in the traditional sense since our study is designed to examine the putative physiological effect of alcohol and not the cognitive effects. Further, our aim in informing participants that they should expect that they might receive alcohol in both sessions was not to deceive, but rather to ensure that the participant would take the necessary steps to avoid the adverse effects of alcohol on an empty stomach.

In the alcohol condition, the participant was served an amount of alcohol (40% ethyl alcohol by volume) mixed with fruit juice in a 1:4 ratio. In the no-alcohol condition, participants received an amount of juice equal to that of the liquid volume in the alcohol condition.

The number of drinks to be consumed by each participant was calculated using the Computerized Blood Alcohol Calculator (CBAC, Addiction Research Foundation, 1992), based on the participant's sex, weight, height and age. Participants were asked to consume a number of drinks estimated to raise blood alcohol concentrations (BACs) in the alcohol condition to 0.08% within a period of 20 min. BACs, determined with a breath-measuring device (Alcometer 7410, Dræger, Inc.), were first measured 15 min after the 20-min drinking period, then every 15 min until the minimum BAC required for testing was reached. Data collection in the alcohol condition began upon reaching a BAC of 0.06% or greater. Following completion of all measurements, participants were asked to remain in the care of the experimenter until their BAC fell below 0.03%. Once this level was reached, participants were debriefed and released from the laboratory.

In the non-alcohol condition, data were obtained 15 min after the consumption of the juice. Measurements were obtained in an identical fashion to that described for the alcohol condition.

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