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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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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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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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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.

3. Experiment 1

As a first step, we were interested in obtaining data that addressed the issue of whether the alcohol-induced impairment in temporal processing is a consequence of an overall slowing of neural transmission. Generally, data from reaction time (RT) studies demonstrate an impairment of simple RT that is dose-dependant: moderate to higher amounts of alcohol consistently cause a greater increase (and thus a greater impairment) in reaction time for visual stimuli (Gustafson, 1986a, 1986b, 1986c; Hernández, Vogel-Sprott, Huchín-Ramirez, & Aké-Estrada, 2006; Lemon, Chesher, Fox, Greeley, & Nabke, 1993; Tzambazis & Stough, 2000; Wallgren & Barry, 1970; Young, 1970; see Jellinek & McFarland, 1940 for a review), while there is little evidence of an impairment at low doses (Wallgren & Barry; Mitchell, 1985). In contrast, tasks in which at least two different stimuli are presented that require different responses (i.e., choice RT) show impairment even at low doses of alcohol. The same pattern of impairments has also been shown for responses to auditory stimuli (Gustafson, 1986b; Hernández et al., 2006).

Because RT consists of the time it takes for stimulus detection *and* the time it takes to make the motor response (Luce, 1986), and since these early studies did not *directly* examine alcohol's effects on the sensory component separate from the motor component, it is not clear from the previous RT data on which of the two components alcohol is exerting its influence. Specifically, it might be that alcohol's effect is selective such that the increase in RT is due to an increase in latency at the sensory stage *or* at the motor stage. Alternatively, alcohol's influence may be a more global one, such that the increase in RT is due to increases in latency for both the sensory *and* motor stages.

To date, there are some data that support the possibility that each stage could be selectively affected by alcohol. For example, Krull, Smith, and Parsons (1994) correlated changes between simple RT and event-related potential (ERP) measures after alcohol consumption. They found that both moderate and high levels of alcohol suppressed the amplitude of the P100 portion of the waveform, as well as the N100 portion. Moreover, the suppression in amplitude was found to be correlated with the increased RT after alcohol. Because the P100 portion of the waveform is thought to reflect sensory processing (Picton, Hillyard, Krausz, & Galambos, 1974), Krull et al. (1994) concluded that the increase in RT was due to an increase in sensory processing time. Hernández et al. (2006) had their participants respond with a key press to the omission of a recurring stimulus. One key was depressed at the start of a trial until the cessation of the stimulus, at which point participants were required to release the first key and depress a second. Hernández et al. (2006) defined sensory RT as consisting of the time between the occurrence of the last stimulus and the release of the first key, while motor RT was the time between releasing the first key and pressing the second. Hernández et al. (2006) found that sensory reaction time for detection of the omission of a recurring stimulus increased after moderate doses of alcohol, while the RT to execute the response remained unchanged.

The available RT data indicate that alcohol causes an increase in sensory processing time. What should be noted, however, is that the kind of tasks employed in the past studies can only *suggest* that the increase in RT is due to an increase in sensory processing latency, because they did not take measures to control for the motor component of RT. Consequently, it is quite possible that an increase in motor processing time could also be contributing to the observed RT measures. A more informative approach would be to factor out the sensory and motor processing times and obtain data of alcohol's effects on transmission speed only. We did this by using a technique first developed by Poffenberger (1912). The technique involves presenting visual targets to either the left or the right visual field and recording manual reaction times to these presentations. When the hand of response and visual field of presentation are on the same side (ipsilateral; the uncrossed condition), the process underlying the response is contained entirely in one hemisphere and along a direct pathway. If, however, the hand of response is opposite to the visual field of presentation (contralateral; crossed condition), one hemisphere receives the visual signal and the other must execute the motor output, and as such the process underlying the response requires interhemispheric transfer.

Assuming that transmission of simple sensory information and the initiation of uncomplicated movements are conducted over fixed neuroanatomical pathways; an estimate of interhemispheric transfer time can be obtained simply by subtracting the reaction time for uncrossed conditions from the reaction time for crossed conditions (crossed-uncrossed difference; CUD). Since both the sensory stimulation and the required motor response are identical in both the crossed and uncrossed conditions, it can be assumed that both sensory and motor processing time are factored out in the calculated CUD and what is extracted is a measure of speed of neural transmission independent of the separate processing components.

To date, while there are some data on the effects of chronic alcohol consumption on interhemispheric transfer time (Schulte, Pfefferbaum, & Sullivan, 2004); there are no data on the effects of acute alcohol consumption. To that end, we used the Poffenberger paradigm in order to obtain a gross indication of alcohol's effects on transmission speed. If the apparent temporal disruption is due to an overall reduction in transmission speed, participants should demonstrate an increase in CUD.

3.1. Method

3.1.1. Observers

A group of 24 adults (19–31 years) participated in the experiment.

3.1.2. Apparatus and stimuli

Stimuli consisted of a rectangular bar 0.5°h by 0.1°w, which was viewed at a distance of 150 cm. Although no effect of retinal eccentricity on CUD has been reported, it was included as a main factor in order to explore any possible interactions between retinal eccentricity and alcohol on CUD. The target was presented 0.5°, 1.5°, 3°, or 5° to the left or right of a central fixation point, and all testing was conducted in a darkened room.

3.1.3. Procedure

The experimental design and procedures for administration of alcohol have already been described.

Participants were told that on each trial a target bar would be presented, either to the left or to the right of a fixation point. They were instructed to respond as quickly as possible to the onset of the target bar by button press of a response box with the index finger of their dominant hand (as determined by the Modified Edinburgh Handedness survey). The response box was located directly in front of the participant, aligned with the midline of his or her body. A single trial consisted of the onset of a centrally located fixation point, the duration of which was varied randomly between 1000 and 1500 ms from trial to trial in order to discourage anticipatory responses. Immediately following, the target was presented for 100 ms at one of the four positions: 0.5°, 1.5°, 3°, or 5° in each hemifield. Participants were required to respond within 800 ms, after which the next trial began. The starting position of their hand was aligned with the midline of his or her body. It was decided a priori to exclude trials in which participant's RT was less then 100 ms and greater then 700 ms, as these were assumed to reflect anticipatory and inattentive responses, respectively. Each of the four positions was presented 120 times per visual hemifield, for a total of 480 trials for each position per experimental session on each of the two test days. Nine of the 24 participants were tested on an earlier version of the task in which each of the four positions was presented 30 times per visual hemifield, for a total of 60 trials for each position, and 120 trials per experimental session on each of the two test days.

3.2. Results

Measurements were made in the window between 0.06% and the target of 0.08% on the rising and falling portion of the BAC curve. Mean BAC measures at the start and end of the experimental session were $M_{start} = 0.07\%$ (SD = 0.014), and $M_{end} = 0.07\%$ (SD = 0.019), respectively.

RT data in the alcohol-free and after alcohol conditions are shown in Fig. 1. Consistent with data from previous studies, a 2 (alcohol vs. no-alcohol) × 2 (cross vs. uncrossed) × 4 (target positions) repeated measures ANOVA show a main effect of alcohol: RT increased after alcohol consumption for both uncrossed and crossed conditions [$F(1, 23) = 6.83$, $p = 0.016$]. Within both the
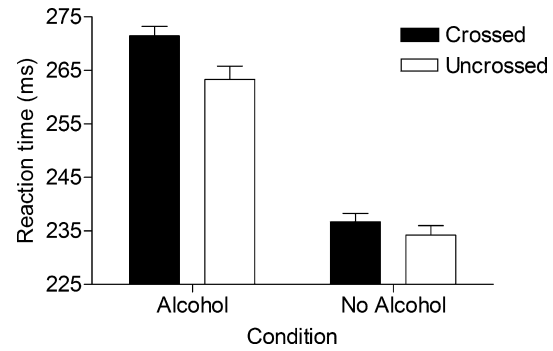

Fig. 1. Mean (± SEM) RT for all stimulus presentations plotted as a function of alcohol and no-alcohol.

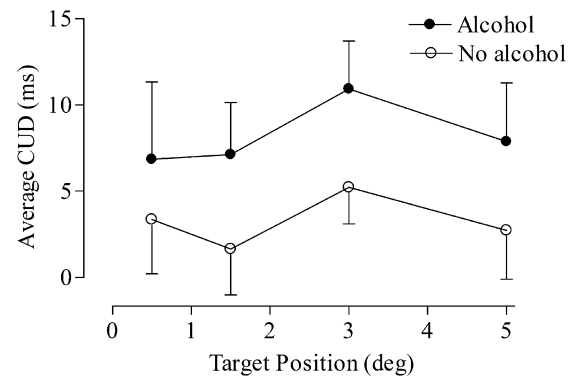
Fig. 2. Average CUD (± SEM) as a function of target position for both alcohol and no alcohol conditions.

alcohol and no alcohol condition, moreover, RT were longer for the longer, crossed path and shorter for the shorter, uncrossed path [$F(1, 23) = 18.74$, $p < 0.0001$].

Fig. 2 shows average CUD (crossed RT − uncrossed RT) before and after alcohol as a function of retinal eccentricity of stimulus presentation. A 2 (alcohol vs. no-alcohol) × 4 (target position) repeated measures ANOVA with CUD as the dependant variable confirmed that we found a main effect of alcohol [$F(1, 23) = 4.52$, $p = 0.044$], which did not differ across the four target positions, indicating a slowing of interhemispheric transmission when using an RT measure. Moreover, while presentation position had an effect on RT [$F(3, 69) = 3.51$, $p = 0.029$; alcohol and no alcohol at 0.5 and 3° (crossed condition), and alcohol at 0.5 and 3°, 1.5, and 3° (uncrossed condition)], it had no effect on CUD. The finding that RT increased with increasing retinal eccentricity is in agreement with the results of previous investigations on the effect of stimulus eccentricity and RT (e.g., Berlucchi, Heron, Hyman, Rizzolatti, & Umiltà, 1971).

4. Experiment 2

The data from our first experiment suggest a slowing in transmission speed. It is also quite possible that neuronal processing time itself is increased by alcohol. One way in

which this could be manifested is as an increase in neuronal latency: slower processing at the neuron and slower transmission of information along the axon. In order to address this possibility, a task is required that provides some measure of neuronal latencies. One example of such a task is the flash-lag effect (*FLE*), which is generally accepted as a psychophysical measure of neuronal latency.

In the flash-lag task, observers compare the position of a continuously moving stimulus to the position of a brief flash of light. When the moving stimulus and the flashed stimulus are physically aligned in space and time, observers nonetheless perceive the moving stimulus as being in front of the flash (MacKay, 1958; Metzger, 1932; Nijhawan, 1994; Walker & Irion, 1982). In order to perceive the two stimuli as being aligned, the flash must be delivered earlier, at a moment when the moving stimulus has not yet reached the flash's position. This illusory spatial offset has been demonstrated with different types of stimuli (Nijhawan & Khurana, 2000), for visual motion, and stimulus attribute changes (e.g., colour, contrast, entropy; for a recent reviews, see Krekelberg & Lappe, 2001; Nijhawan, 2002; Whitney, 2002).

Nijhawan (1994) was the first to also report on the linear speed dependence of the flash lag effect: the magnitude of the FLE increases as a linear function of the velocity of the moving stimulus. This linearity simply reflects the fact that the difference in latency is independent of speed (Krekelberg & Lappe, 2001), and that the slope of the speed dependence can be used to express spatial offsets as equivalent temporal delays. Thus, although the stationary stimulus appears to increasingly lag behind the progress of a moving stimulus increasing in speed, the temporal delay between the two is constant. Moreover, as the moving stimulus increases in speed, any temporal delays will be reflected in the lag between the two; the magnitude of the slope provides the magnitude of the temporal delay demonstrated by the spatial offset of the lag.

Although a variety of explanations have been proposed (e.g., motion extrapolation, sensory postdiction; Khurana & Nijhawan, 1995; Nijhawan, 1994, 1997, 2002; Eagleman & Sejnowski, 2000), a generally accepted view is that the perceived offset is caused by differential neuronal latencies for moving and flashed stimuli: moving stimuli are processed faster than stationary flashes (Brenner & Smeets, 2000; Eagleman & Sejnowski, 2000; Krekelberg & Lappe, 1999; Metzger, 1932; Purushothaman, Patel, Bedell, & Ogmen, 1998; Whitney & Cavanagh, 2000; Whitney, Cavanagh, & Murakami, 2000; Whitney & Murakami, 1998; Whitney, Murakami, & Cavanagh, 2000). With this difference in latency, the flash reaches awareness when the moving object is already farther along its trajectory. Therefore, the flash appears to lag behind the already processed, moving object.

Findings from some physiological studies support the notion that the FLE is a psychophysical manifestation of differential latencies. Intracellular recordings demonstrate that moving stimuli are processed within the visual system

at a different rate from stimuli presented for a very brief period of time. These differences in the rate of processing can be found at both the lateral geniculate nucleus (LGN) and the cortical level. For example, LGN neurons were found to respond with shorter delays to moving than for flashed light bars (Orban, Hoffmann, & Duysens, 1985). Measuring at the cortical level, Jancke, Erlhagen, Schöner, and Dinse (2004) presented moving and stationary flashed stimuli while recording from neurons in the primary visual cortex of the cat. Jancke et al. found that response latencies to moving light bars were significantly shorter than the response latencies from the same neurons for the presentation of stationary flashed stimuli. Their findings demonstrate differential processing, as well as providing indirect support for the differences in processing time that lead to a perceived spatial offset.

Given that the FLE is almost certainly an expression of neuronal latency of visual processing, examination of the FLE under alcohol and no-alcohol conditions should provide further understanding of alcohol's effects on processing speed. To our knowledge, no study has been conducted that examines the effects of alcohol on the FLE. If it is the case that alcohol acts to increase neuronal latency for both the flash and the moving stimulus, then participants should demonstrate an FLE increased in magnitude after alcohol.

4.1. Method

4.1.1. Observers
Participants were six volunteers, aged 21–34 years. Two additional male participants were disqualified due to difficulties in performing the task after alcohol consumption.

4.1.2. Apparatus and stimuli
A chin and headrest were used to maintain a constant viewing distance of 80 cm and to prevent excessive head movements. Participants viewed the display binocularly. The stimulus display employed in the present experiment is presented in Fig. 3. The background screen luminance was 1 cd m^2. The moving stimulus consisted of a pair of vertically aligned bars (40 cd m^2) that were translated horizontally 3° above a fixation cross. The distance between
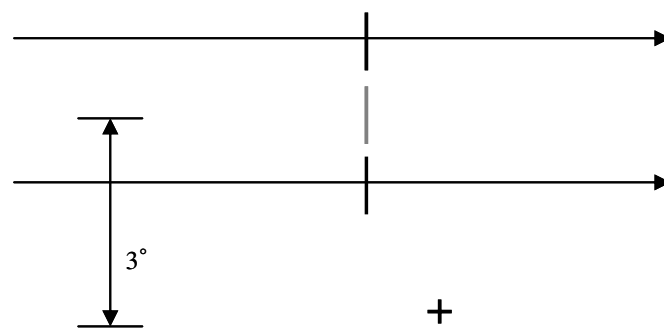

Fig. 3. Schematic of stimulus display used in the flash lag task (flashed target bar = grey, moving bars = black).

the bars was constant at 1°, and they moved concurrently at 6 different velocities (2.75, 4, 5.25, 6.5, 7.75, 9° s^{-1}), with only one velocity measured in a run. On each trial, the motion of the bars was randomly presented as either moving from the left to the right, or right to left. A bar equal in size to one of the pair of vertically aligned bars of the moving stimulus was flashed for one video frame (10 ms) between the moving bars.

4.1.3. Procedure

The experimental design and procedures for administration of alcohol have already been described.

Target spatial thresholds (the minimum amount of offset required in order for a percept of alignment between the moving bar and flash) were determined using a dual random interleaved staircase design. Participants indicated whether the flash appeared spatially offset to the left or right of the moving bar by pressing a button on a two-choice response box. Initial presentation of the flash was at centre, and a response of a perceived spatial offset shifted the bar in 0.4 min increments to the left or right of fixation (depending on the direction of motion of the moving bar). The flash was presented for 10 ms, and the velocity of the moving bar ranged from 2.75° s^{-1} to 9° s^{-1}, in increments of 1.25° s^{-1}. Each velocity was measured in a separate run. Testing was continued until seven response reversals had occurred on each staircase, and the spatial and temporal offset was taken as the average of the final six reversals on both staircases. Each velocity was tested three times, with the final average of the three runs making up the FLE for that particular bar velocity.

4.2. Results

Measurements were made in the window between 0.06% and the target of 0.08% on the rising and falling portion of the BAC curve. Mean BAC measures at the start and end of the experimental session were 0.0795% (SD = 0.019) at the start, and 0.0667% (SD = 0.011) at the end of the session.

Fig. 4 shows the data in units of spatial lag (min of arc) averaged across all six participants for both the alcohol and no-alcohol conditions. Spatial offset thresholds were plotted as a function of the six different bar velocities. Consistent with past findings, a 2 (alcohol vs. no-alcohol) × 6 (bar velocities) repeated measures ANOVA confirmed that participants demonstrated a typical flash-lag effect in which the amount of spatial offset increased as a linear function of bar velocity (Nijhawan, 1994) under both the alcohol and no-alcohol conditions [$F_{(5,25)} = 26.43$, $p < 0.0001$]. In addition, participants demonstrated a larger FLE after alcohol [$F_{(1,5)} = 9.51$, $p = 0.027$]. No significant alcohol × bar velocity interaction was found [$F_{(5,25)} = 1.56$, $p = 0.209$].

In order to determine the delay in processing time, the spatial lag data were converted into temporal units (see Fig. 5). On average, the flashed bar lagged the moving ones
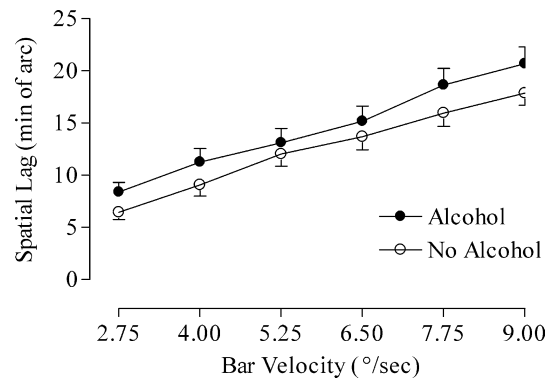

Fig. 4. Average spatial offset thresholds as a function of the six different bar velocities for both alcohol and no alcohol conditions.
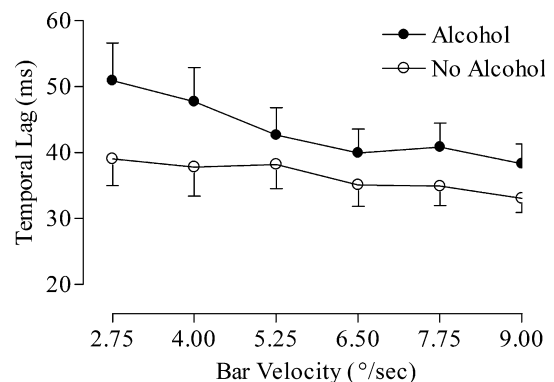

Fig. 5. Temporal offset plotted as a function of bar velocity for both alcohol and no alcohol conditions.

by 36.35 ms without alcohol and by 43.39 ms with alcohol [$F_{(1,5)} = 10.88$, $p = 0.021$]. Further inspection of the data suggests that alcohol seems to have a greater effect for targets that move at slower velocities. This apparent effect of bar velocity, however, was not significant [$F_{(5,25)} = 1.36$, $p = 0.272$], nor was there a significant alcohol × bar velocity interaction [$F_{(5,25)} = 1.92$, $p = 0.127$].

Taken together, these data demonstrate an increase in the magnitude of the FLE after alcohol, as manifested by a vertical shift in the flash-lag function, suggesting an alcohol-induced overall reduction in speed of processing for both the flash and the moving stimulus.

Given that the FLE is an expression of neuronal latency, the examination of the FLE under alcohol and no-alcohol conditions provides convergent evidence for a preferential effect of alcohol on processing speed. Specifically, these data show that alcohol seems to cause an increase in the neuronal latency to process visual information.

5. Experiment 3

Another way to investigate the dynamic properties of the visual system is to use visual masking. Masking occurs whenever the visibility of one stimulus, the target, is reduced by the presence of another stimulus, the mask.

The magnitude of the masking effect is dependent on the amount of time separating the onset of the target and the onset of the mask. By varying the onset asynchrony (i.e., SOA), the temporal relationship between the neural response to the target and the neural response to the mask can be manipulated. When the response to the mask interacts with the response to the target, the effect is measured psychophysically as masking. Given that masking provides a measure of temporal processing, examination of the masking effect under alcohol and no-alcohol conditions should provide further understanding of alcohol's effects on processing speed.

There is both behavioural and electrophysiological evidence for the efficacy of using a masking protocol. Neuronal recordings have found effects of visual masks at different levels in the visual pathway. Single unit recordings in V1 of the macaque revealed a reduction in the transient onset-response and after-discharges associated with the addition and removal, respectively, of a cycling stimulus (Macknik & Livingstone, 1998), and the effect was shown to be larger at later levels of visual processing: the duration, peak firing rate, and stimulus selectivity were reduced (Kovács, Vogels, & Orban, 1995; Rolls & Tovée, 1994; Rolls, Tovée, & Panzeri, 1999; Tovée & Rolls, 1995) as target visibility was reduced by the mask. These findings suggest an increasing interference of neural processing for the target by the mask, which corresponds to decreased target visibility shown behaviourally in humans (Macknik & Livingstone, 1998). In terms of behavioural evidence, there are many experiments that demonstrate the interaction between the mask and target on responses (Breitmeyer, 1984; Enns & Di Lollo, 2000), but the evidence on the associated changes in cerebral activity is sparse. The few studies done, however, provide converging evidence with the electrophysiological findings: both performance and cerebral activation decrease as the target and mask get temporally closer (Bacon-Macé, Macé, Fabre-Thorpe, & Thorpe, 2005; Grill-Spector, Kushnir, Hendler, & Malach, 2000).

To date, only two studies have used visual masking in an attempt to obtain a clearer understanding of alcohol's effects on the dynamics of visual processing. Jones et al. (1998), and Moskowitz and Murray (1976), using backward masking with noise or structure, respectively, found that an increase in temporal separation between the target and the mask was necessary after alcohol consumption, which they took as a demonstration of alcohol causing a reduction in the speed of processing. A closer examination of both the tasks show that the processing demands made on the participants could be construed as occurring at later levels. Specifically, the masking protocol also provides a means to indirectly explore alcohol's effects on the interaction between the processing demand of the task and the speed of processing. One way to manipulate the demands made on processing is to vary the type of response required from the participant. For example, the processing demands for *discriminating* a target are greater than the demands for

simply *detecting* a target: Jones et al. required that participants discriminate between two bars and identify which of the two had an extra feature, and Moskowitz and Murray had participants discriminate a letter from an array of four. Since the processing demands are generally greater for discrimination, it is not clear from these data whether early levels of processing also experience a decrease in the rate of processing, or even whether alcohol causes an overall slowing in total processing speed.

The existing literature suggests that overall processing efficiency is reduced after alcohol (Gustafson, 1986a, 1986b, 1986c; Hernández et al., 2006; Lemon et al., 1993; Maylor, Rabbitt, James, & Kerr, 1990, 1992; Tzambazis & Stough, 2000; Wallgren & Barry, 1970). Further, there are some data that show a reduction in processing efficiency for information with low processing demands (Maylor et al., 1990; Tzambazis & Stough, 2000). What is not apparent, however, is whether the demonstrated reduction of overall processing efficiency is a consequence of a chain reaction that commences with alcohol exerting its influence at the lowest levels of information processing, or whether the effects occur at a higher level.

In order to address this, participants were required to complete two types of masking tasks that differed with respect to the processing demands placed on the participant. In the low-level task (LLT), participants were required to simply detect the target; the higher-level task (HLT) required both detection and identification of the orientation of the target. The rationale was that if alcohol was affecting processing at the lowest levels, then performance on both tasks should be impaired. If only higher level processing was affected, then only the second task would show a deficit.

5.1. Method

5.1.1. Observers

Participants were twelve volunteers, aged 21–34 years. One participant was excluded because performance in the discrimination task hovered around chance (i.e., 25%).

5.1.2. Apparatus and stimuli

The screen luminance was 10 cd m^2, and four fixation crosses spaced 1° apart boxed in the central viewing area. The target stimulus was a disc with a diameter of 1°, containing a square-wave grating pattern with a spatial frequency of 5 cycles per degree (cpd). The target was presented for 10 ms, followed by a masking stimulus that was a 2° square. It contained a random pattern of black and white dots, with 100% contrast between the dots. The mean luminance of the masking stimulus was 50 cd m^{-2}. The mask was displayed on the screen for 100 ms.

5.1.3. Procedure

The experimental design and procedures for administration of alcohol were identical to that already described.

Low-level task. In the low-level task, participants were required to indicate whether they saw the vertically oriented target that was presented some time before the mask (see Fig. 6). Participants were tested at 10 SOA values of 10 ms to 100 ms in 10 ms increments, as well as a run in which only the target was presented without the mask. The order in which the SOA values were administered was random for each participant. Participants initiated each experimental run by pressing a button in response to a tone. Contrast thresholds were obtained using a dual random interleaved staircase procedure, with initial contrast set at 10%. The participant's contrast threshold was calculated as the average of the final eight reversals on both staircases. If the contrast in any experimental run was at 100% and the participant continually gave a negative response, the run was manually terminated after approximately eight negative responses in each staircase. The SOA value in which this occurred was re-tested once all other experimental runs were complete.

Higher-level task. For the second task, participants were required to make a decision regarding target orientation. There were four possible target orientations: vertical, horizontal, right-oblique, and left-oblique. A method of constant stimuli with a four-alternative forced choice procedure was used to measure the percentage of correct identifications as a function of SOA (see Fig. 7). In a run, each orientation was presented 25 times at a specific SOA, for a total of 100 presentations. In total, there were 10 experimental runs in the orientation task with SOA values of 10–100 ms in 10 ms increments. The SOA value for every run was chosen at random. The grating contrast of the target was held constant at 10%, with the expectation that at low SOA values it would be undetectable, and at high SOA values it would be easily detected. The participant initiated each run with a button press in response to a tone, causing the first stimulus to be presented on the screen. The participants were required to indicate the target orientation by pressing the appropriate button on a four-choice response box. At the end of each experimental run, the percent correct for each orientation was calculated, as well as an average percent correct across all orientations.
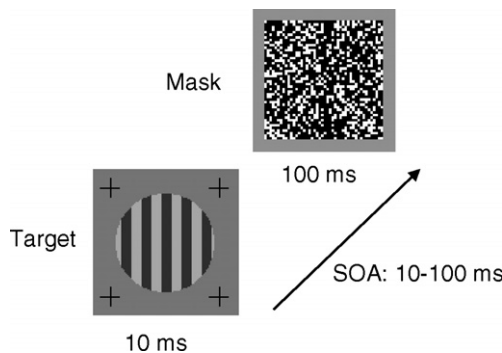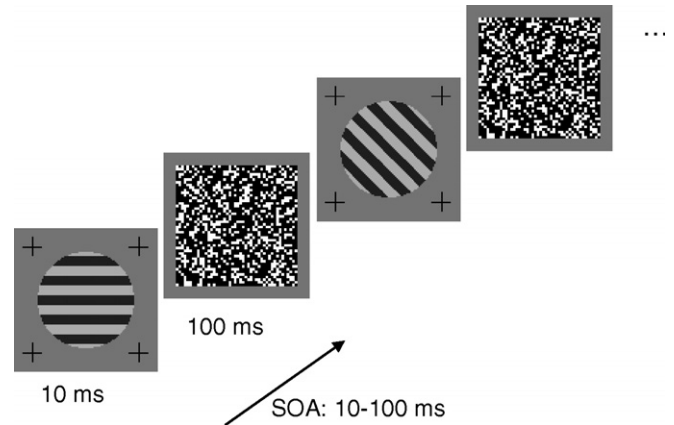

Fig. 7. Stimulus display for the higher-level task.

5.2. Results

Measurements were made in the window between 0.06% and the target of 0.08% on the rising and falling portion of the BAC curve. Mean BAC measures at the start and end of the experimental session were $M_{start} = 0.067\%$ (SD = 0.009), and $M_{end} = 0.07\%$ (SD = 0.014), respectively.

Initially, performance for each orientation in the HLT was assessed separately with a 4 (orientation) × 10 (SOA) repeated measures ANOVA. Because there were no differences (no alcohol: $p = 0.540$; alcohol: $p = 0.403$), data were collapsed across orientations in the HLT for both alcohol and no alcohol conditions. Frequency-of-seeing curves were plotted for the combined forced choice data for the HLT, and the threshold data for the LLT. Under both alcohol and no alcohol conditions, performance improved as SOA increased for both the LLT and HLT, consistent with other findings.

Low-level task. Fig. 8 shows average contrast required for target detection as a function of SOA. As can be seen from the group data, as SOA increased, contrast required for detection decreased. In order to determine whether there were any SOA values in which there was no effect of the mask, a single sample *t*-test comparing the average
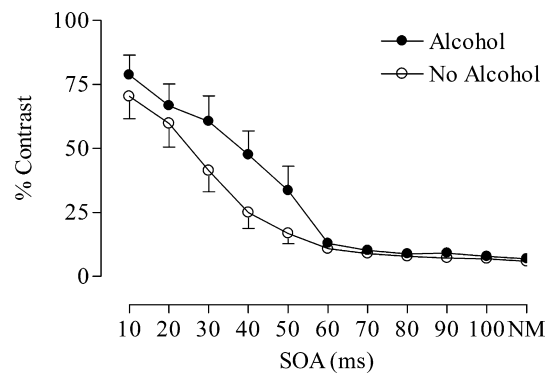

Fig. 6. Stimulus display for low level task.

Fig. 8. Percent contrast at threshold plotted as a function of SOA for both alcohol and no alcohol conditions in the low level task.

threshold for detection when no mask was presented with the average thresholds at each SOA was computed. Target visibility was greatest (and thus the effect of the mask on processing was the least) when the target was presented 100 ms before the mask; all other SOAs resulted in a significant masking effect. Further, participants demonstrated contrast thresholds that varied with SOA, indicating a masking effect $[F (8, 88) = 40.53, p < 0.001]$ overall with or without alcohol. There was no overall effect of alcohol on target detection ($p = 0.08$), likely due to the little or no effects at the higher SOAs, which is reflected in a significant interaction between SOA and alcohol $[F (8, 88) = 2.31, p = 0.027]$.

Alcohol had the greatest effect on masking SOA values of 30, 40, and 50 ms $[F (1, 11) = 4.92, p = 0.049]$. These data demonstrate alcohol induced changes in a detection task for short SOAs, suggesting that temporal processing is reduced after alcohol at lower levels of processing. This finding is consistent with findings from previous studies that indicate that early levels of processing are sensitive to the influence of alcohol.

Higher-level task. Fig. 9 shows the average percentage of correct identifications as a function of SOA for both alcohol and no alcohol conditions. The group data show a masking effect: as SOA increased, percentage of correct discriminations increased $[F (9, 99) = 52.82, p < 0.0001]$. In order to determine the extent to which complete masking occurred even in the absence of alcohol across SOAs, comparisons were made between chance performance (25%) and the average percentage of correct discriminations in the no alcohol conditions at each SOA using a single sample *t*-test. Complete masking (i.e., chance performance) occurred at SOA values of 10 ms and 20 ms, at all other SOA values participants' performance was above chance levels.

The effect of alcohol on the percentage of correct responses increased as SOA values increased from 30 to 100 ms $[F (7, 77) = 44.37, p < 0.001]$, indicating that a masking effect was present. Further, there was a main effect of alcohol $[F (1, 11) = 14.66, p = 0.003]$, showing that alcohol slowed the ability to discriminate target orientation as
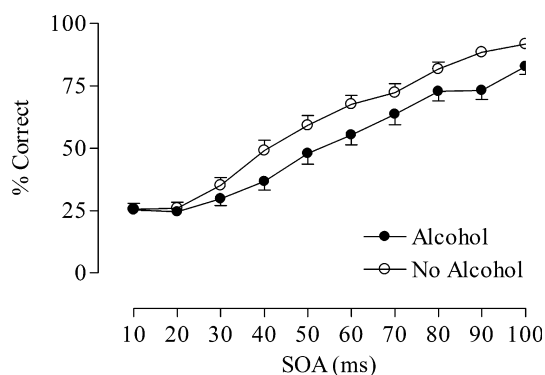
SOA increased by approximately 10 ms. That is to say, akin to the low-level task, the data show that SOA must be increased by 10 ms in order to produce the same level of performance with alcohol to the level of performance without alcohol; both the alcohol and no alcohol functions overlap if the no alcohol function is shifted by 10 ms to the right.

6. Discussion

Findings from previous studies have demonstrated the widespread systemic effects of alcohol on the body and CNS. These effects have been shown to occur both physiologically and behaviourally. The present series of experiments provides a demonstration of the use of the visual system as a model system to reveal a link between alcohol-induced changes in the physiological mechanisms underlying visual functions and alcohol-induced perceptual changes. While it is certain that alcohol's influences on visual functions are multiply determined, one candidate mechanism is alcohol-induced changes in the rate of neural processing. In tasks that provide a measure of processing and transmission speed, participants demonstrated larger CUDs, a larger FLE, and prolonged masking after alcohol consumption. It should be noted that because SOA values beyond 100 ms were not measured in our higher-level masking task, it is unclear whether or not performance after alcohol ever recovers to the no-alcohol level of performance, or whether the decrease in percent correct is due to a fundamental property of alcohol unrelated to time. Further investigation is required to address this issue. Nevertheless, our data are consistent with the view that alcohol impairs the speed of neural processing and transmission.

The data from the present series of experiments suggest that alcohol may exert its effects to a greater degree on functions largely reliant on processing speed. Support for this suggestion comes from two lines of evidence from the visual system. First, functions that the parvocellular pathway is responsible for seem to be affected very little, or not at all, by alcohol. For example, there are relatively small effects on acuity and spatial contrast sensitivity, and the ability to recognize objects is scarcely changed after alcohol. Second, there are data that show a greater effect of alcohol on *rapid* temporal processing; which is a property of the magnocellular pathway. For example, Pearson and Timney (1998) found that after alcohol, decreases in contrast sensitivity increase progressively as a function of temporal frequency, with small decreases at low temporal frequencies, and large decreases at high temporal frequencies. Given that it is more likely that the lower temporal frequencies are mediated by the parvocellular path, Pearson and Timney's finding suggest that with respect to speed of processing, alcohol has greater effects when rapid temporal processing is required, and that processing speed and transmission efficiency along the magnocellular pathways are more affected.

Fig. 9. Percent correct identifications plotted as a function of SOA for both alcohol and no alcohol conditions in the higher-level task.

Additional support for the view that alcohol may exert its effects to a greater degree on mechanisms of processing speed, and as such, on the magnocellular pathway, may be found from an examination of the "perception for action" component of the dorsal stream. Visuomotor processing, which is responsible for our ability to coordinate and use visual information in order to perform appropriate motor actions (i.e., perception for action), occurs in the magnocellular pathway. To date, there are some behavioural data demonstrating alcohol's effects on visuomotor processing (Timney & Johnston, 2003). For example, Timney & Johnston showed that in a simple manual prehension task, participants demonstrated changes to reaching behaviour after alcohol. Grasping, on the other hand, was not affected. Kirkpatrick (2005), Kade, Steeves, Goodale, and Timney (2005), and Johnston and Timney (unpublished data), moreover, demonstrated that alcohol adversely affects motor responses that are amended during execution due to changes in visual information.

The behavioural measures obtained in the present series of studies are not constrained to the visual modality. IHTT, the masking effect, and the flash-lag effect have all been demonstrated in the auditory and somatosensory domains, as well as cross-modally (Alais & Burr, 2003; Axelrod, Thompson, & Cohen, 1968; Clarke & Geffen, 1990; Fink, Ulbrich, Churan, & Wittmann, 2006; Gescheider, 1966; Hirsh & Sherrick, 1961). From this, a central-timing mechanism independent of sensory modality has been proposed as the mechanism mediating all of the aforementioned measures (Fink et al., 2006). By extension, it seems feasible that the observed reduction in both transmission and processing speed shown in the present series of experiments should also be observed in a comparable fashion across other sensory modalities. To our knowledge, there are no studies investigating alcohol's effects on temporal aspects of neural processing in other modalities using these behavioural measures. Data across modalities would provide support of an effect of alcohol on an over-arching central timing mechanism.

In summary, the data from the experiments described herein suggest that while alcohol does affect the higher integrative functions, this effect has its origins at the lowest levels. The observed slowing of responsiveness after alcohol may be a consequence of changes in processing that result from information transmitted through the system too slowly, along with an overall slowing in processing. Moreover, this reduction in transmission and processing efficiency appears to originate at stages of processing of information that exert minimal to low demands on the processing system.

References

Alais, D., & Burr, D. (2003). The 'flash-lag' effect occurs in audition and cross-modally. *Current Opinions in Biology, 13*, 59–63.

Axelrod, S., Thompson, L. W., & Cohen, L. D. (1968). Effects of senescence on the temporal resolution of somesthetic stimuli presented to one hand or both. *Journal of Gerontology, 23*, 191–195.

Bacon-Macé, N., Macé, M. J. –M., Fabre-Thorpe, M., & Thorpe, S. J. (2005). The time course of visual processing: Backward masking and natural scene categorization. *Vision Research, 45*, 1459–1469.

Berlucchi, G., Heron, W., Hyman, R., Rizzolatti, G., & Umiltà, C. (1971). Simple reaction times of ipsilateral and contralateral hand to lateralized visual stimuli. *Brain, 94*, 419–430.

Bernhard, C. G., & Skoglund, C. R. (1941). Selective suppression with ethyl alcohol of inhibition in the optic nerve and the negative component PIII of the electroretinogram. *Acta physiologica Scandinavica, 11*, 10–21.

Breitmeyer, B. G. (1984). *Visual masking: An integrative approach*. New York: Oxford University Press.

Brenner, E., & Smeets, J. B. J. (2000). Motion extrapolation is not responsible for the flash-lag effect. *Vision Research, 40*, 1645–1648.

Browning, M., Schummers, J., & Bentz, S. (1999). Alcohol, memory, and molecules. In Y. Liu & W. A. Hunt (Eds.), *The drunken synapse: Studies of alcohol related disorders* (pp. 159–168). New York: Plenum.

Clarke, C. R., & Geffen, G. M. (1990). Hemispheric equivalence for simultaneity judgements of somatosensory stimuli. *Cognitive Neuropsychology, 7*, 311–327.

Colrain, I. M., Taylor, K. J., McLean, S., Buttery, R., Wise, G., & Montgomery, I. (1993). Dose dependent effects of alcohol on visual evoked potentials. *Psychopharmacology, 112*, 383–388.

DiPerri, R., Dravid, A., Schweigerdt, A., & Himwich, H. E. (1968). Effects of alcohol on evoked potentials of various parts of the central nervous system of cat. *Quarterly Journal of Studies on Alcohol, 29*, 20–37.

Draski, L. J., & Deitrich, R. A. (1996). Initial effects of ethanol on the central nervous system. In R. A. Deitrich & V. G. Erwin (Eds.), *Pharmacological effects of ethanol on the nervous system* (pp. 227–250). Boca Raton: CRC Press.

Eagleman, D. M., & Sejnowski, T. J. (2000). Motion integration and postdiction in visual awareness. *Science, 287*, 2036–2038.

Enns, J. T., & Di Lollo, V. (2000). What's new in visual masking? *Trends in Cognitive Sciences, 4*, 345–352.

Enzer, N., Simonson, E., & Ballard, G. (1944). The effect of small doses of alcohol on the central nervous system. *American Journal of Clinical Pathology, 14*, 333–341.

Erickson, K. R., Joe Willey, T., Riley, D. M., Fuster, J. M., & Lawrence, A. F. (1982). Principal component analysis of evoked responses and the effects of alcohol on the geniculo-striate system of the monkey. *Neurological Research, 4*, 89–113.

Fink, M., Ulbrich, P., Churan, J., & Wittmann, M. (2006). Stimulus-dependent processing of temporal order. *Behavioural Processes, 71*, 344–352.

Gescheider, G. A. (1966). Resolving of successive clicks by the ears and skin. *Journal of Experimental Psychology, 71*, 378–381.

Grill-Spector, K., Kushnir, T., Hendler, T., & Malach, R. (2000). The dynamics of object-selective activation correlate with recognition performance in humans. *Nature Neuroscience, 3*, 837–843.

Gustafson, R. (1986a). Effect of moderate doses of alcohol on simple auditory reaction time in a vigilance setting. *Perception and Motor Skills, 62*, 683–690.

Gustafson, R. (1986b). Alcohol and vigilance performance: Effect of small doses of alcohol on simple visual reaction time. *Perception and Motor Skills, 62*, 951–955.

Gustafson, R. (1986c). Alcohol and vigilance performance: Effect of small doses of alcohol on simple auditory reaction time. *Perception and Motor Skills, 63*, 99–102.

Hernández, O. H., Vogel-Sprott, M., Huchín-Ramirez, T. C., & Aké-Estrada, F. (2006). Acute dose of alcohol affects cognitive components of reaction time to an omitted stimulus: Differences among sensory systems. *Psychopharmacology, 184*, 75–81.

Hetzler, B. E., Oaklay, K. E., Heilbronner, R. L., & Vestal, T. (1982). Acute effects of alcohol on photic evoked potentials of Albino rats: Visual cortex and superior colliculus. *Pharmacology, Biochemistry, & Behaviour, 17*, 1313–1316.

Hill, J. C., Powell, B., & Goodwin, D. W. (1973). Critical flicker fusion: Objective measure of alcohol tolerance. *The Journal of Nervous and Mental Disease, 157*, 46–49.

Hirsh, I. J., & Sherrick, C. E. J. (1961). Perceived order in different sense modalities. *Journal of Experimental Psychology, 62*, 423–432.

Ikeda, H. (1963). Effects of ethyl alcohol on the evoked potential of the human eye. *Vision Research, 3*, 155–169.

Jancke, D., Erlhagen, W., Schöner, G., & Dinse, H. R. (2004). Shorter latencies for motion trajectories than for flashes in population responses of cat primary visual cortex. *Journal of Physiology, 556*, 971–982.

Jellinek, E. M., & McFarland, R. A. (1940). Analysis of psychological experiments on the effects of alcohol. *Quarterly Journal of Studies on Alcohol, 1*, 272–371.

Jones, M. B., Chronister, J. L., & Kennedy, R. S. (1998). Effects of alcohol on perceptual speed. *Perceptual & Motor Skills, 87*, 1247–1255.

Kade, C.M., Steeves, J.K.E., Goodale, M.A., & Timney, B. (2005, November). Alcohol intoxication affects the ability to countermand reaching and grasping. Presented at the Society for Neuroscience Conference, Washington, USA.

Khurana, B., & Nijhawan, R. (1995). Extrapolation or attention shift? *Nature, 378*, 565–566.

Kirkpatrick, C. (2005). Examining the effect of alcohol on visuomotor processing: An investigation of automatic and corrective motor responses. Unpublished Senior Honours Thesis, University of Western Ontario.

Kovács, G., Vogels, R., & Orban, G. A. (1995). Cortical correlate of pattern backward masking. *Proceedings of the National Academy of Sciences USA, 92*, 5587–5591.

Krekelberg, B., & Lappe, M. (1999). Temporal recruitment along the trajectory of moving objects and the perception of position. *Vision Research, 39*, 2669–2679.

Krekelberg, B., & Lappe, M. (2001). Neuronal latencies and the position of moving objects. *Trends in Neuroscience, 24*, 335–339.

Krull, K. R., Smith, L. T., & Parsons, O. A. (1994). Simple reaction time event-related potentials: Effects of alcohol and diazepam. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 18*, 1247–1260.

Lemon, J., Chesher, G., Fox, A., Greeley, J., & Nabke, C. (1993). Investigation of the hangover effects of an acute dose of alcohol on psychomotor performance. *Alcoholism: Clinical & Experimental Research, 17*, 665–668.

Luce, R. D. (1986). *Response times: Their role in inferring elementary mental organization*. New York: Oxford.

MacKay, D. M. (1958). Perceptual stability of a stroboscopically lit visual field containing self-luminous objects. *Nature, 181*, 507–508.

Macknik, S. L., & Livingstone, M. S. (1998). Neuronal correlates of visibility and invisibility in the primate visual system. *Nature Neuroscience, 1*, 144–149.

Mangold, S., Laubli, T., & Krueger, H. (1996). Effects of low alcohol dose on static balance, fine motor activity, and mental performance. *Neurotoxicology & Teratology, 18*, 547–554.

Maylor, E. A., Rabbitt, P. M., James, G. H., & Kerr, S. A. (1990). Effects of and extended practice on divided-attention performance. *Perception & Psychophysics, 48*, 445–452.

Maylor, E. A., Rabbitt, P. M., James, G. H., & Kerr, S. A. (1992). Effects of alcohol, practice and task complexity on reaction time distributions. *Quantitative Journal of Experimental Psychology, 44*, 119–139.

Metzger, W. (1932). Versuch einer gemeinsamen Theorie der Phänomene Fröhlichs und Hazelhoffs und Kritik ihrer Verfahren zur Messung der Emfindungszeit. *Psychologische Forschung, 16*, 176–200.

Mitchell, M. C. (1985). Alcohol-induced impairment of central nervous system function: Behavioural skills involved in driving. *Journal of Studies on Alcohol, Supplement, 10*, 109–116.

Moskowitz, H., & Murray, J. T. (1976). Alcohol and backward masking of visual information. *Journal of Studies on Alcohol, 37*, 40–45.

Nijhawan, R. (2002). Neural delays, visual motion, and the flash-lag effect. *Trends in Cognitive Sciences, 6*, 387–393.

Nijhawan, R. (1997). Visual decomposition of colour through motion extrapolation. *Nature, 386*, 66–69.

Nijhawan, R. (1994). Motion extrapolation in catching. *Nature, 370*, 256–257.

Nijhawan, R., & Khurana, B. (2000). Conscious registration of continuous and discrete visual events. In T. Metzinger (Ed.), *Neural correlates of consciousness: Empirical and conceptual problems* (pp. 203–219). New York: MIT Press.

van Norren, D., & Padmos, P. (1977). Influences of anaesthetics, ethyl alcohol, and Freon on dark adaptation of monkey cone ERG. *Investigative Ophthalmology & Visual Science, 16*, 80–83.

Ogden, E J. D., & Moskowitz, H. (2004). Effects of alcohol and other drugs on driver performance. *Traffic Injury Prevention, 5*, 185–198.

Orban, G. A., Hoffmann, K. P., & Duysens, J. (1985). Velocity selectivity in the cat visual system. I. Responses of LGN cells to moving bar stimuli: A comparison with cortical areas 17 and 18. *Journal of Neurophysiology, 54*, 1026–1049.

Pearson, P., & Timney, B. (1998). Effects of moderate blood alcohol concentrations on spatial and temporal contrast sensitivity. *Journal of Studies on Alcohol, 59*, 163–173.

Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials. I: Evaluation of components. *Electroencephalography & Clinical Neurophysiology, 36*, 179–190.

Poffenberger, A. T. (1912). Reaction time to retinal stimulation with special references to the time lost in conduction through nervous centres. *Archives in Psychology, 23*, 1–73.

Purushothaman, G., Patel, S. S., Bedell, H. E., & Ogmen, H. (1998). Moving ahead through differential visual latency. *Nature, 396*, 424.

Rohrbaugh, J. W., Stapleton, J. M., Parasuraman, R., Zubovic, E. A., Frowein, H. W., Varner, J. L., et al. (1987). Dose-related effects of ethanol on visual sustained attention and event-related potentials. *Alcohol, 4*, 293–300.

Rolls, E. T., & Tovée, M. J. (1994). Processing speed in the cerebral cortex and the neurophysiology of visual masking. *Proceedings of the Royal Society of London: Series B, 257*, 9–15.

Rolls, E. T., Tovée, M. J., & Panzeri, S. (1999). The neurophysiology of backward visual masking: Information analysis. *Journal of Cognitive Neuroscience, 11*, 300–311.

Schulte, T., Pfefferbaum, A., & Sullivan, E. V. (2004). Parallel interhemispheric processing in aging and alcoholism: Relation to corpus callosum size. *Neuropscholigia, 42*, 257–271.

Timney, B., & Johnston, K. (2003, July). Alcohol affects reaching, but not grasping, in a simple manual prehension task. Presented at the International Conference on Perception and Action, Gold Coast, Australia.

Tovée, M. J., & Rolls, E. T. (1995). Information encoding in short firing rate epochs by single neurons in the primate temporal visual cortex. *Visual Cognition, 2*, 35–58.

Tzambazis, K., & Stough, C. (2000). Alcohol impairs speed of information processing and simple and choice reaction time and differentially impairs higher-order cognitive abilities. *Alcohol & Alcoholism, 35*, 197–201.

Virsu, V., Kykka, T., & Vahvelainen, M. (1974). Effects of alcohol on inhibition in the human visual system II. Spatial and temporal contrast sensitivity. Reports from the Institute of Psychology: The University of Helsinki.

Wallgren, H., & Barry, H. (1970). In *Actions of Alcohol. Biochemical, Physiological, & Psychological Aspects: Chronic and Clinical Aspects* (Vol. 1). New York: Elsevier Science.

Walker, J. T., & Irion, A. L. (1982). Apparent displacement of moving and stationary strobe flashes. *Human Factors, 24*, 213–224.

Whitney, D. (2002). The influence of visual motion on perceived position. *Trends in Cognitive Sciences, 6*, 211–216.

Whitney, D., & Cavanagh, P. (2000). Motion distorts visual space: Shifting the perceived position of remote stationary objects. *Nature Neuroscience, 3*, 954–959.

Whitney, D., Cavanagh, P., & Murakami, I. (2000). Temporal facilitation for moving stimuli is independent of changes in direction. *Vision Research, 40*, 3829–3839.

Whitney, D., & Murakami, I. (1998). Latency difference, not spatial extrapolation. *Nature Neuroscience, 1*, 656–659.

Whitney, D., Murakami, I., & Cavanagh, P. (2000). Illusory spatial offset of a flash relative to a moving stimulus is caused by differential latencies for moving and flashed stimuli. *Vision Research, 40*, 137–149.

Young, J. R. (1970). Blood alcohol concentration and reaction time. *Quarterly Journal* of Studies on Alcohol, *31*, 823–831.