



Contents lists available at SciVerse ScienceDirect

# Vision Research

journal homepage: [www.elsevier.com/locate/visres](http://www.elsevier.com/locate/visres)

## How alcohol intake affects visual temporal processing

Marina Kunchulia<sup>a,\*</sup>, Karin S. Pilz<sup>b,c</sup>, Michael H. Herzog<sup>c</sup>

<sup>a</sup>Laboratory of Vision Physiology, Beritashvili Center of Experimental Biomedicine, Tbilisi, Gotua 14, 0160 Tbilisi, Georgia

<sup>b</sup>School of Psychology, University of Aberdeen, Scotland, UK

<sup>c</sup>Laboratory of Psychophysics, Brain Mind Institute, School of Life Sciences, Ecole Polytechnique Federale de Lausanne (EPFL), Switzerland

### ARTICLE INFO

#### Article history:

Received 26 January 2012

Received in revised form 13 June 2012

Available online 23 June 2012

#### Keywords:

Backward masking  
Spatial processing  
Temporal processing  
Alcohol

### ABSTRACT

Alcohol affects vision. However, the influence of alcohol on visual processing is largely unknown. Here, we investigated the effects of alcohol on visual spatiotemporal processing. We employed a visual paradigm, the shine through backward masking paradigm, in which a vernier is either presented alone or followed by a variety of mask. We investigated performance for women at blood alcohol levels of 0 mg/kg, 400 mg/kg and 600 mg/kg and for men at 0 mg/kg, 400 mg/kg and 800 mg/kg. When the vernier was presented alone, vernier offset discrimination was not affected by alcohol. When the vernier was followed by a mask, stimulus onset asynchronies (SOAs) between target and mask were significantly longer after alcohol intake. However, as a second experiment showed, spatial and temporal processing *per se* were not impaired by alcohol. In addition, spatial processing was not affected by moderate alcohol consumption. Hence, moderate consumption of alcohol does not affect visual processing *per se*. We propose that the longer SOAs after alcohol intake are related to changes in mechanisms of target stabilization rather than changes in spatial and temporal sensitivity as has been previously suggested.

© 2012 Elsevier Ltd. All rights reserved.

### 1. Introduction

Alcohol intake affects most perceptual and motor functions: It increases reaction times, decreases motor and cognitive functions, and strongly affects attention (Abroms & Fillmore, 2004; Abroms, Gottlob, & Fillmore, 2006; Field et al., 2010; Ivanec, Svagelj, & Rebic, 2009; Koelega, 1995; Patel et al., 2010; Sauls et al., 2007). Visual functions are affected by alcohol intake such as depth perception (Watten & Lie, 1996; Wegner & Fahle, 1999), contrast sensitivity (Nicholson et al., 1995; Pearson & Timney, 1998; Watten & Lie, 1996), visual short term memory (Wegner & Fahle, 1999) and visual temporal processing. For example, the critical flicker fusion frequency, the highest rate at which a light can be flashed on and off before it is perceived as being continuous, is reduced (Carpenter, 1962; Hill, Powell, & Goodwin, 1973; Pearson & Timney, 1998). In addition, Khan and Timney (2007) found prolonged interhemispheric transmission, larger flash lags, and prolonged backward masking after alcohol intake. These results are usually taken as evidence that alcohol slows neural processing.

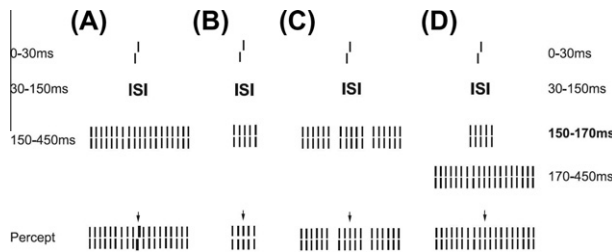
Most studies on alcohol induced visual deficits focus on male subjects (Nicholson et al., 1992a, 1992b, 1995; Watten & Lie, 1996) or do not specifically investigate sex differences (Khan & Timney, 2007; Pearson & Timney, 1998; Wegner & Fahle, 1999). However, it is well known that alcohol affects men and women

differently and that the effects of alcohol are more pronounced in women than in men (Avant, 1990; Dougherty, Bjork, & Bennett, 1998; Hoppenbrouwers, Hofman, & Schutter, 2010; Miller, Weafer, & Fillmore, 2009), which might be related to differences in body fat (Addolorato et al., 1999), the availability of the enzyme alcohol dehydrogenase that is important for alcohol break-down (Baraona et al., 2001), or a higher tolerance for alcohol due to different drinking habits.

In the current paper, we aim at investigating the effects of alcohol on spatiotemporal processing for men and women. We used the shine-through effect, a well-established backward masking technique (Herzog, Fahle, & Koch, 2001; Herzog & Koch, 2001; Herzog, Koch, & Fahle, 2001). The methods used in the current study are based upon a protocol for special populations such as schizophrenic patients (Herzog, Kopmann, & Brand, 2004). In the shine-through paradigm, a vernier stimulus, which consists of two abutting bars, precedes a grating that comprises 25 elements. The vernier is perceived as being wider and brighter than it really is and seems to be superimposed on the grating (Fig. 1). However, if the grating has five elements, shine-through does not occur and the vernier is almost invisible even though the 5-element grating is contained within the 25-element grating. Shine-through is affected by both temporal and spatial alterations of the grating. For example, spatial inhomogeneities, such as gaps inserted close to the center of the 25-element grating render the vernier invisible (Herzog, Fahle, & Koch, 2001; Herzog & Koch, 2001; Herzog, Kopmann, & Brand, 2004). Likewise, the brief presentation of a five

\* Corresponding author. Fax: +995 32 237 1157.

E-mail address: [marina.kunchulia@gmail.com](mailto:marina.kunchulia@gmail.com) (M. Kunchulia).



**Fig. 1.** Shine-through. (A) vernier is presented for a short time and followed by a grating comprising more than seven elements. The foregoing vernier appears to be superimposed on the grating, looking wider, brighter, and for some observers, even longer. (B) For a grating with five elements, the visibility of the preceding vernier is strongly diminished. (C) Visibility is strongly diminished if an extended grating contains gaps (gap grating). (D) Performance also deteriorates for temporal inhomogeneities in the mask such as presenting a five-element grating for 20 ms before a grating with 25 elements appears (see scale on the right for timing in this condition). Only in condition (A), the vernier is clearly visible as a shine-through element, i.e., weaker masking. In all other conditions, shine-through is strongly diminished or even abolished. The interstimulus interval (ISI) denotes the time difference between vernier offset and grating onset. The stimulus onset asynchrony (SOA) is defined as the difference between grating and vernier onset and is the sum of vernier duration and ISI ( $SOA = \text{vernier duration} + \text{ISI}$ ). The bottom row shows the percept corresponding to the physical stimulus shown on top of the arrows. The vernier offset shown is strongly exaggerated in this figure. The spacing between grating elements is  $200''$ , whereas the vernier offsets are often much smaller.

element grating immediately before the presentation of the 25-element grating dramatically deteriorates vernier offset discrimination, which indicates that the five element grating is perceived despite its short presentation time (Herzog, Koch, & Fahle, 2001; Herzog, Kopmann, & Brand, 2004). It has been argued that shine-through is related to perceptual grouping of the mask: Only if the grating is perceived as a homogeneous and extended object, shine-through occurs. However, if spatial or temporal inhomogeneities segment the extended grating into smaller parts, the shine-through effect is decreased or even extinguished (Fig. 1).

## 2. Materials and methods

### 2.1. Subjects

Fifty seven healthy volunteers participated in this study (20–41 years, 27 males). All gave written informed consent. The research was approved by the Georgian National Bioethics Committee. All participants had normal or corrected to normal vision, with visual acuity of at least 0.8 in at least one eye as tested with the Freiburg Visual Acuity Test (Bach, 1996). None of the participants had a history of alcohol abuse. Also, none of the subjects

reported neurological disorders, pregnancy, allergy to alcohol, or prescription to any medication for any mental or physical illness.

### 2.2. Alcohol administration

The experimental design was between groups and each participant took part in one session only. The procedure of the experiment was single-blind and the experimenter knew which group the participant belonged to. However, data analysis was carried out independently and without knowledge of the participants' identity. Participants were asked to have a light breakfast on the day of testing, not less than 2 h before the start of the experiment. Alcohol was administered at 400 mg/kg ( $n = 11$ , mean = 32.6 years) and 600 mg/kg ( $n = 9$ , mean = 26 years) for female participants and 400 mg/kg ( $n = 7$ , mean = 30 years) and 800 mg/kg ( $n = 10$ , mean = 24.9 years) for male subjects. An increased alcohol dosage for male participants (600 mg/kg for women and 800 mg/kg for men) was chosen in accordance with pilot experiments in our lab, which showed that alcohol administration at 400 and 600 mg/kg had no or very little effect on performance in male subjects (also see Avant, 1990). The administered beverage consisted of grapefruit juice mixed with vodka. In the no-alcohol condition, 10 male (mean = 24.2 years) and 8 female (mean = 30.1 years) received the grapefruit juice with 0.25 ml of vodka that was floated on the drink surface and around the rim of the glass to mask olfactory cues (Terry et al., 2009). For all no-alcohol participants, blood alcohol level remained zero after administration of the drink (Fig. 2). The mean total volume of the drink was 230 ml, and consumed within 6 min. Participants were informed that their drink might or might not contain alcohol. However, observers were oblivious about the exact contents of the administered beverage.

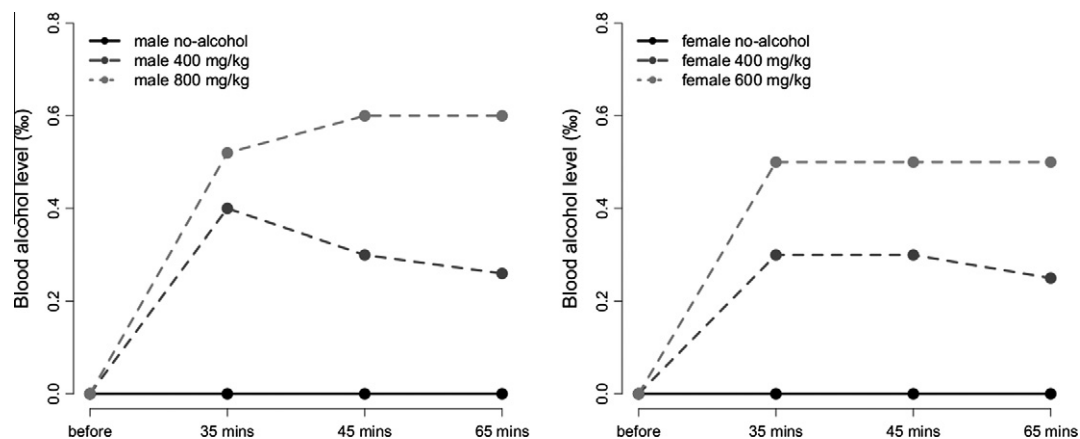
The administered doses of alcohol were relatively low and none of the subjects reported any signs of alcohol intake such as loss of coordination or blurred vision. All subjects had clear speech and were able to follow task instructions.

### 2.3. Alcohol concentrations

Alcohol concentrations were determined from breath samples measured by an alcohol tester (COSMOS CA-2001). Alcohol concentrations were measured before alcohol administration, at 35, 45 and 65 min after drinking.

### 2.4. Stimuli and apparatus

Stimuli and apparatus were similar to Herzog, Kopmann, and Brand (2004). The stimuli consisted of white vertical verniers and gratings comprising either 25 or five elements presented on a black



**Fig. 2.** Alcohol concentration for male (left) and female subjects (right) before and after the intake of alcohol.

background. The vernier was composed of two bars that were slightly displaced in the horizontal direction either to the left or to the right. The length of a segment of the vernier, i.e. one bar, was  $10'$ . Segments were separated by a small gap of  $1'$ . The aligned verniers of the gratings had the same lengths as the verniers. The horizontal distance between grating elements was about  $3.33'$ . The vernier and the central element of the grating always appeared in the middle of the screen. The stimuli appeared on a Samsung SyncMaster 957DF CRT screen with a 100 Hz refresh rate. Maximal screen luminance was about  $100 \text{ cd/m}^2$  as measured with a GretagMacbeth Eye-One Display 2 colorimeter. The background luminance on the screen was below  $1 \text{ cd/m}^2$ . Observers were seated in a dimly lit room at 5 m from the monitor.

### 2.5. Procedure

The procedure was similar to Herzog, Kopmann, and Brand (2004). Verniers were presented in the middle of the screen without a masking grating. Observers had to determine the offset direction (left or right) of the lower bar. In the first step, we determined the shortest vernier duration (VD) for which vernier offset discrimination thresholds (VO) was below  $40'$  for each observer. We used the adaptive PEST procedure (Creelman & Taylor, 1969). In the first block, verniers were presented for 150 ms. In the following blocks, vernier duration was reduced when the threshold for offset discrimination was below the predefined value of  $40'$  and increased when the threshold for offset discrimination was above  $40'$ . The procedure was stopped when a threshold of about  $40'$  was reached. Two participants did not reach a VD of below 30 ms and were excluded at this stage of the experiment. Each block consisted of 80 trials.

After the individual VD was determined, participants were administered the beverage. 35 min after administration of the beverage, individual VO was re-tested.

In the second step, stimulus-onset asynchronies (SOAs) for backward masking were determined using the adaptive PEST procedure (Creelman & Taylor, 1969). The vernier was followed by an ISI and then a grating. The grating was comprised of either 5 or 25 aligned verniers, i.e., verniers without offset. We adaptively assessed the stimulus-onset-asynchrony (SOA) between target and mask. For each observer, we used the individual vernier duration as determined in step one. The vernier offset size was set to  $71'$ . The SOA was varied from trial to trial. We determined the critical SOA for which a performance level of 75% correct responses was obtained with Probit and Maximum Likelihood analysis. The starting value of the SOA was set to 200 ms. For each grating two thresholds were determined. The mean of the two thresholds was taken as the critical SOA. If observers were unable to reach a threshold value of 400 ms or below, a value of 450 ms was recorded (for details see Herzog, Fahle, & Koch, 2001).

In the third step, we tested performance for two inhomogeneous masks. We used the individual SOA and vernier duration as determined in step one. Baseline performance was comparable across all observers. We determined vernier offset discrimination using the adaptive PEST strategy (Creelman & Taylor, 1969), which means that we varied vernier offset size adaptively. In the first condition, we presented the standard 25-element grating for 300 ms as described above. In the second condition, we presented a 25-grating for 300 ms, in which the middle two lines were removed (gap grating) (Fig. 1). The gap width was  $250'$  and separated the central five elements from the peripheral  $2 \times 10$  elements. In the third condition, we presented a 25-grating for 280 ms, which was preceded by a 5-element grating for 20 ms (5–25 grating). Hence, duration of the combined gratings was 300 ms as was the presentation time for conditions 1 and 2.

## 3. Results

### 3.1. Alcohol concentration

Fig. 2 shows alcohol concentration as measured with an alcohol tester from breath samples. The alcohol level for males and females is very similar at a level of 400 mg/kg. At the level of 800 mg/kg, alcohol concentration for men is roughly comparable to the alcohol concentration for women at a level of 600 mg/kg.

### 3.2. Vernier duration and offset

Apart from two male and three female subjects, all subjects had a vernier duration of 20 ms. The mean vernier offsets (VOs) for male and female subjects are shown in Fig. 3. A 2 (time of testing – before and after alcohol intake)  $\times$  3 (alcohol groups) repeated measure ANOVA showed no main effects for male and female subjects. Only the time of testing showed a marginally significant improvement for male subjects ( $F(1,22) = 3.9, p = 0.06$ ).

### 3.3. Backward masking

Fig. 4 shows SOAs for male (left) and female subjects (right) for 5- and 25-element gratings for all groups of participants. An ANOVA on the factor of alcohol group revealed significant main effects for male subjects for SOA5 ( $F(2,24) = 5.8, p < 0.01$ ) and SOA25 ( $F(2,24) = 6.6, p < 0.01$ ), which were mainly due to the fact that the SOA was elevated for men at 800 mg/kg alcohol. There were no significant differences between the groups who had 400 mg/kg and the no-alcohol group for both SOA5 and SOA25.

For female subjects, the SOAs were elevated but there was no significant difference between alcohol groups for both SOA5 and SOA25. However, when comparing both alcohol groups with the

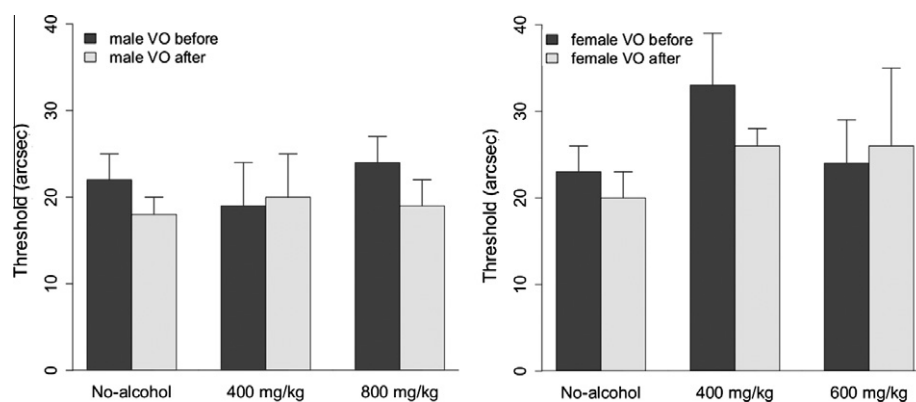


Fig. 3. Vernier duration for male (left) and female subjects (right) at all administered alcohol levels before and after the intake of alcohol.

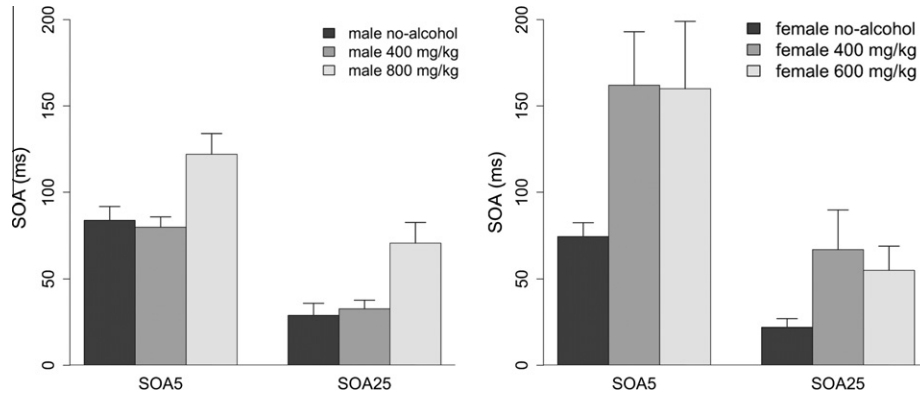


Fig. 4. SOAs for male (left) and female subjects (right) for 5- and 25-element gratings at all alcohol levels.

no-alcohol group, there was a significant difference between the groups with alcohol and the group with no alcohol for SOA5 ( $t(22) = 3.2, p < 0.002$ ) but not SOA25 ( $t(22) = 1.4, p = 0.8$ ).

3.4. Inhomogeneous masks

Fig. 5 (top) shows thresholds (arcsec) for male (left) and female subjects (right) for the 25 grating, gap grating and 5–25 grating at all alcohol levels. There was no significant difference between alco-

hol groups for any of the inhomogeneous mask for both male and female subjects (Fig. 5). To be able to investigate the effects of alcohol on temporal processing and figure ground segregation independent of backward masking, we normalized the performance for the inhomogeneous gap grating and 5–25 grating to the performance for the 25 grating by subtracting the individual results for the grating 25 from the individual results for the gap grating and the 5–25 grating (Fig. 5 bottom). *T*-tests on the normalized data were significantly different from zero for both the gap grating

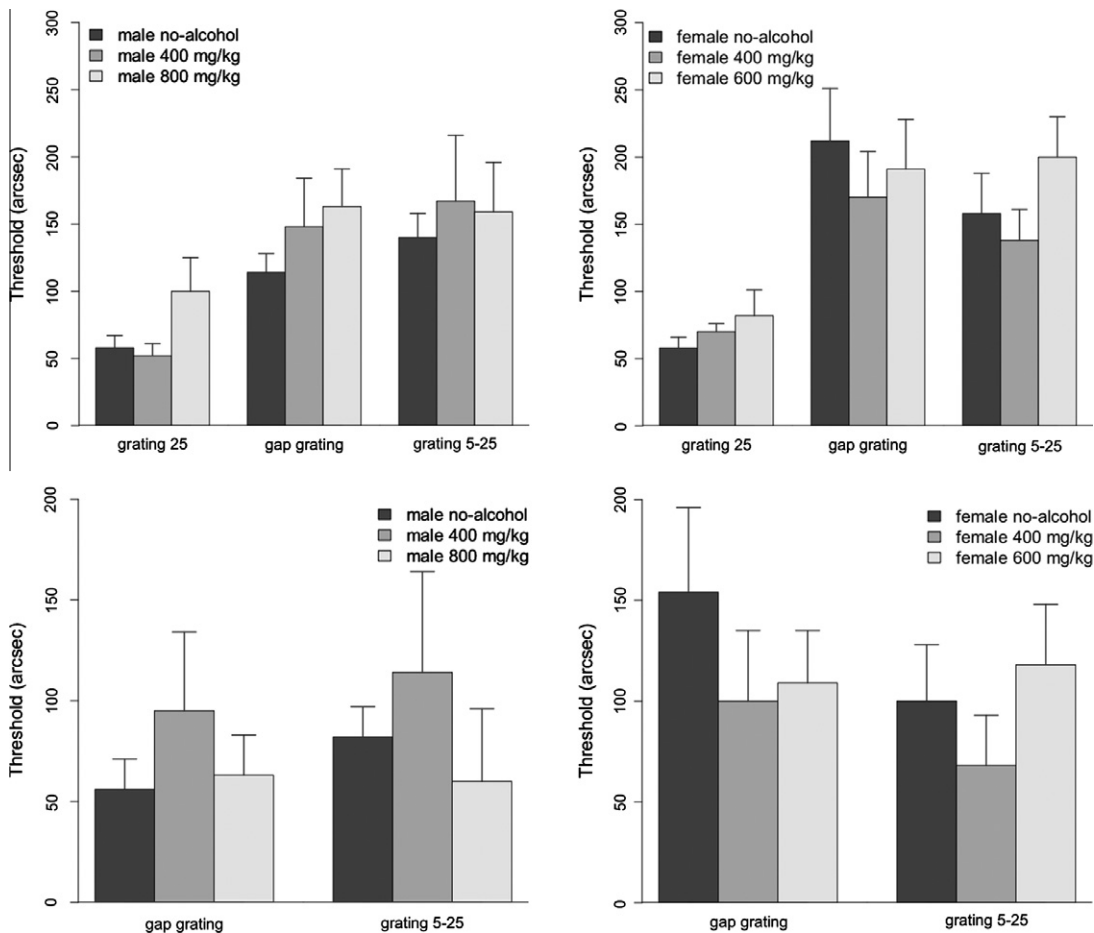


Fig. 5. Top: Thresholds (arcsec) for male (left) and female subjects (right) for the 25 grating, gap grating and 5–25 grating at all alcohol levels. Bottom: Normalized thresholds for the 25 grating (arcsec) for male (left) and female subjects (right) for the gap grating and the 5–25 grating at all alcohol levels. Individual thresholds for the 25 grating were subtracted from the individual thresholds for the gap and the 5–25 grating.

(males:  $t(26) = 5.1, p < 0.001$ ; females:  $t(27) = 5.96, p < 0.001$ ) and the grating 5–25 (males:  $t(26) = 4.3, p < 0.001$ ; females:  $t(27) = 5.8, p < 0.001$ ).

#### 4. Discussion

We investigated the effects of alcohol intake on spatiotemporal processing for men and women (Herzog, Fahle, & Koch, 2001; Herzog & Koch, 2001; Herzog, Koch, & Fahle, 2001; Herzog, Kopmann, & Brand, 2004). In the first part of the study we found that vernier offset discrimination was very little affected by alcohol. These results are in accordance with a previous study by Hogan and Gilmartin (1985). However, Wegner and Fahle (1999), however, found elevated thresholds after alcohol consumption. They presented two verniers in succession and asked observers which offset was larger. Hence, their task required both the accurate perception of the vernier offset and a visual short-term memory component. Thus, it seems that vernier offset discrimination is rather intact under the influence of alcohol as long as visual memory is not necessary to perform the task.

In the second part of the study, we investigated the effects of alcohol on backward masking. Here, a vernier was masked by a 5-element or a 25-element grating. For all groups of subjects, SOAs for the 25-element grating were lower than for the 5-element grating as has been found in many previous studies (Herzog, Kopmann, & Brand, 2004; Roinishvili et al., 2011). For both gratings, alcohol increased thresholds for women at 400 mg/kg, whereas for male subjects thresholds were only elevated after the intake of 800 mg/kg alcohol. Our results are in good accordance with the few previous studies on visual masking and alcohol consumption that showed elevated SOAs after alcohol intake (Khan & Timney, 2007; Moskowitz & Murray, 1976).

Next, we tested the effects of alcohol on inhomogeneous masks. We found that performance in all groups was very similar with increased thresholds in both the gap grating and the 5–25 grating condition compared to the 25 grating condition. The elevated thresholds indicate that even with high alcohol blood concentrations the masks are carefully processed. Alcohol has been found to blur vision (Kerr et al., 1990). Our results suggest that blurred vision or lowpass filtering of the stimulus cannot account for the observed effects. In case of blurring or lowpass filtering of the visual input, the gaps within the gap grating should have become less visible and thresholds for the gap grating should have become more similar to the homogenous 25 element grating, i.e., performance should have improved. However, our results show that the gaps efficiently increased masking. A similar argument applies to the 5–25 element grating. If the five element grating, which was only presented for 20 ms, had been merged with or wiped out by the 25 element grating, which was presented for 300 ms, we would have expected an improved performance. However, also here our results show that the five element grating was processed efficiently and strongly increased masking. Therefore, in both cases mask processing seems to be fast and accurate, indicating that visual processing *per se* is not affected under the influence of alcohol. It needs to be noted, however, that the alcohol doses that were tested in the course of the current experiments are very low. Administering higher doses of alcohol may have stronger effects on mask processing.

In summary, we found that alcohol intake neither affected vernier offset discrimination without the presence of a mask, nor the processing of the masks. In the basic masking conditions, however, SOAs were strongly increased due to alcohol intake. There are several approaches to explain these results: First, alcohol might decrease visual processing and account for the larger SOAs based on alcohol intake. However, vernier offset discrimination is largely

unaffected by alcohol intake and therefore, this hypothesis is rather unlikely. In addition, the masks themselves seem to be processed in the same way with and without alcohol consumption, which underlines the hypothesis that visual processing itself is not impaired. We rather propose that alcohol intake deteriorates mechanisms of target processing. The target vernier is presented for just a brief period of time and therefore, needs to be stabilized or “protected” from the 300 ms grating. If the vernier had not been the target, there would have been no need for target protection and the vernier would have possibly gone unnoticed.

Very little is known about the mechanisms related to target stabilization. A potential mechanism might be related to attention. It has been shown that attention suffers strongly from alcohol consumption (do Canto-Pereira et al., 2007; Post et al., 1996; Roehrs et al., 1994; Rohrbaugh et al., 1987; Schulte et al., 2001). Previous neurophysiological studies showed that attention can boost neural signals related to target processing, whereas it inhibits responses unrelated to the attended target (Martinez-Trujillo & Treue, 2004; Maunsell & Treue, 2006; Treue & Martinez Trujillo, 1999). Alcohol might diminish the enhancing effect of attention on the neural signals related to the vernier so that the mask simply overrides these signals compared to when no alcohol is consumed. Thus, increased SOAs under the influence of alcohol might be explained by a negative effect of alcohol on target stabilization.

Another explanation for the increased SOAs under the influence of alcohol might be related to the neuromodulatory effects of alcohol. Previous studies have shown that alcohol reduces global cortical excitability and increases cortical inhibition in the visual system, which seems to be mediated by an increase in GABA-related inhibition and a decrease in NMDA-related glutamatergic excitability (Eckardt et al., 1998; Hoffman et al., 1989; Wang et al., 2000; Weiner & Valenzuela, 2006). Therefore, increased SOAs under the influence of alcohol might be explained by an imbalance between excitatory and inhibitory cortical mechanisms.

The results of the current study are in accordance with recent studies on schizophrenic patients (Chkonia et al., 2010; Herzog, Kopmann, & Brand, 2004). With the same paradigm as described here, schizophrenic patients required only slightly longer vernier durations and clearly longer SOAs compared to healthy observers. However, when both VD and SOA were adjusted individually, fast and intact processing of the gap and the 5–25 element grating was found. In the current study, vernier duration did not change due to alcohol intake and fast and intact processing was found with the 5–25 element grating. These results indicate that target processing depends on neuromodulation or attention, whereas visual processing *per se* does not seem to be affected by alcohol intake or schizophrenia. Masking performance (SOA) in the shine-through effect has also been shown to be strongly modulated by the intake of the benzodiazepine lorazepam, and lorazepam induced deficits are comparable to performance in schizophrenic patients (Giersch & Herzog, 2004). We also looked at different effects of alcohol on men and women. The qualitative effects alcohol had on the results were similar for men and women. Quantitatively, women showed effects of alcohol intake as indicated by longer SOAs for backward masking at a dosage as low as 400 mg/kg, whereas men only showed effects after the intake of 800 mg/kg. These results are in accordance with previous studies showing that alcohol affects men at higher doses than women (Avant, 1990) which might be related to differences in body fat (Addolorato et al., 1999), the availability of the enzyme alcohol dehydrogenase that is important for alcohol break-down (Baraona et al., 2001), or a higher tolerance for alcohol due to different drinking habits.

In conclusion, our results reveal that moderate consumption of alcohol does not affect the perception of visual stimuli *per se*. Longer SOAs for backward masking might be related to changes in mechanisms related to target stabilization rather than changes in

spatiotemporal sensitivity of the visual system. In addition, spatial processing does not seem to be affected by low doses of alcohol up to 800 mg/kg in males and 600 mg/kg in females.

## Acknowledgment

This work was supported by the Volkswagen Foundation project “Between Europe and the Orient – A Focus on Research and Higher Education in/on Central Asia and the Caucasus”.

## References

- Abroms, B. D., & Fillmore, M. T. (2004). Alcohol-induced impairment of inhibitory mechanisms involved in visual search. *Experimental and Clinical Psychopharmacology*, *12*(4), 243–250.
- Abroms, B. D., Gottlob, L. R., & Fillmore, M. T. (2006). Alcohol effects on inhibitory control of attention: Distinguishing between intentional and automatic mechanisms. *Psychopharmacology (Berl)*, *188*(3), 324–334.
- Addolorato, G., Capristo, E., Caputo, F., Greco, A. V., Ceccanti, M., Stefanini, G. F., et al. (1999). Nutritional status and body fluid distribution in chronic alcoholics compared with controls. *Alcoholism, Clinical and Experimental Research*, *23*(7), 1232–1237.
- Avant, L. L. (1990). Alcohol impairs visual presence/absence detection more for females than for males. *Perceptual Psychophysiology*, *48*(3), 285–290.
- Bach, M. (1996). The freiburg visual acuity test – Automatic measurement of visual acuity. *Optometry and Vision Science*, *73*(1), 49–53.
- Baraona, E., Abittan, C. S., Dohmen, K., Moretti, M., Pozzato, G., Chayes, Z. W., et al. (2001). Gender differences in pharmacokinetics of alcohol. *Alcoholism, Clinical and Experimental Research*, *25*(4), 502–507.
- Carpenter, J. A. (1962). Effects of alcohol on some psychological processes. A critical review with special reference to automobile driving skill. *Quarterly Journal of Studies on Alcohol*, *23*, 274–314.
- Chkonia, E., Roinishvili, M., Makhatadze, N., Tserava, L., Stroux, A., Neumann, K., et al. (2010). The shine-through masking paradigm is a potential endophenotype of schizophrenia. *PLoS One*, *5*(12), e14268.
- Creelman, C. D., & Taylor, M. M. (1969). Some pitfalls in adaptive testing: Comments on “temporal integration and periodicity pitch”. *Journal of the Acoustical Society of America*, *46*(6), 1581–1582.
- do Canto-Pereira, L. H. M., de PA David, I., Machado-Pinheiro, W., & Ranvaud, R. D. (2007). Effects of acute alcohol intoxication on visuospatial attention. *Human and Experimental Toxicology*, *26*(4), 311–319.
- Dougherty, D. M., Bjork, J. M., & Bennett, R. H. (1998). Effects of alcohol on rotary pursuit performance: A gender comparison. *Psychological Record*, *48*(3), 393–405.
- Eckardt, M. J., File, S. E., Gessa, G. L., Grant, K. A., Guerri, C., Hoffman, P. L., et al. (1998). Effects of moderate alcohol consumption on the central nervous system. *Alcoholism, Clinical and Experimental Research*, *22*(5), 998–1040.
- Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking. *Alcoholism, Clinical and Experimental Research*, *34*(8), 1346–1352.
- Giersch, A., & Herzog, M. H. (2004). Lorazepam strongly prolongs visual information processing. *Neuropsychopharmacology*, *29*(7), 1386–1394.
- Herzog, M. H., Fahle, M., & Koch, C. (2001). Spatial aspects of object formation revealed by a new illusion, shine-through. *Vision Research*, *41*(18), 2325–2335.
- Herzog, M. H., & Koch, C. (2001). Seeing properties of an invisible object: Feature inheritance and shine-through. *Proceedings of the National Academy of Sciences of the United States of America*, *98*(7), 4271–4275.
- Herzog, M. H., Koch, C., & Fahle, M. (2001). Shine-through: Temporal aspects. *Vision Research*, *41*(18), 2337–2346.
- Herzog, M. H., Kopmann, S., & Brand, A. (2004). Intact figure-ground segmentation in schizophrenia. *Psychiatry Research*, *129*(1), 55–63.
- Hill, S. Y., Powell, B., & Goodwin, D. W. (1973). Critical flicker fusion: Objective measure of alcohol tolerance? *Journal of Nervous and Mental Disease*, *157*(1), 46–49.
- Hoffman, P. L., Rabe, C. S., Moses, F., & Tabakoff, B. (1989). N-methyl-d-aspartate receptors and ethanol: Inhibition of calcium flux and cyclic gmp production. *Journal of Neurochemistry*, *52*(6), 1937–1940.
- Hogan, R. E., & Gilmartin, B. (1985). The relationship between tonic vergence and oculomotor stress induced by ethanol. *Ophthalmic and Physiological Optics*, *5*(1), 43–51.
- Hoppenbrouwers, S. S., Hofman, D., & Schutter, D. J. L. G. (2010). Alcohol breaks down interhemispheric inhibition in females but not in males: Alcohol and frontal connectivity. *Psychopharmacology (Berl)*, *208*(3), 469–474.
- Ivanec, D., Svagelj, A., & Rebic, V. (2009). The impact of different levels of blood alcohol concentration on psychomotor tasks. *Suvremena Psihologija*, *12*(1), 81–89.
- Kerr, D., Macdonald, I. A., Heller, S. R., & Tattersall, R. B. (1990). Alcohol causes hypoglycaemic unawareness in healthy volunteers and patients with Type 1 (insulin-dependent) diabetes. *Diabetologia*, *33*, 216–221.
- Khan, S. A., & Timney, B. (2007). Alcohol slows interhemispheric transmission, increases the flash-lag effect, and prolongs masking: Evidence for a slowing of neural processing and transmission. *Vision Research*, *47*(13), 1821–1832.
- Koelega, H. S. (1995). Alcohol and vigilance performance: A review. *Psychopharmacology (Berl)*, *118*(3), 233–249.
- Martinez-Trujillo, J. C., & Treue, S. (2004). Feature-based attention in-creases the selectivity of population responses in primate visual cortex. *Current Biology*, *14*(9), 744–751.
- Maunsell, J. H. R., & Treue, S. (2006). Feature-based attention in visual cortex. *Trends in Neurosciences*, *29*(6), 317–322.
- Miller, M. A., Weafer, J., & Fillmore, M. T. (2009). Gender differences in alcohol impairment of simulated driving performance and driving-related skills. *Alcohol and Alcoholism*, *44*(6), 586–593.
- Moskowitz, H., & Murray, J. T. (1976). Alcohol and backward masking of visual information. *Journal of Studies on Alcohol*, *37*(1), 40–45.
- Nicholson, M. E., Andre, J. T., Tyrrell, R. A., Wang, M., & Leibowitz, H. W. (1995). Effects of moderate dose alcohol on visual contrast sensitivity for stationary and moving targets. *Journal of Studies on Alcohol*, *56*(3), 261–266.
- Nicholson, M. E., Wang, M., Airhihenbuwa, C. O., Mahoney, B. S., Christina, R., & Maney, D. W. (1992a). Variability in behavioral impairment involved in the rising and falling BAC curve. *Journal of Studies on Alcohol*, *53*(4), 349–356.
- Nicholson, M. E., Wang, M. Q., Airhihenbuwa, C. O., Mahoney, B. S., & Maney, D. W. (1992b). Predicting alcohol impairment: Perceived intoxication versus BAC. *Alcoholism, Clinical and Experimental Research*, *16*(4), 747–750.
- Patel, M., Modig, F., Magnusson, M., & Fransson, P. A. (2010). Alcohol intoxication at 0.06 and 0.10% blood alcohol concentration changes segmental body movement coordination. *Experimental Brain Research*, *202*(2), 431–443.
- Pearson, P., & Timney, B. (1998). Effects of moderate blood alcohol concentrations on spatial and temporal contrast sensitivity. *Journal of Studies on Alcohol*, *59*(2), 163–173.
- Post, R. B., Lott, L. A., Maddock, R. J., & Beede, J. I. (1996). An effect of alcohol on the distribution of spatial attention. *Journal of Studies on Alcohol*, *57*(3), 260–266.
- Roehrs, T., Beare, D., Zorick, F., & Roth, T. (1994). Sleepiness and ethanol effects on simulated driving. *Alcoholism, Clinical and Experimental Research*, *18*(1), 154–158.
- Rohrbaugh, J., Stapleton, J. M., Parasuraman, R., Frowein, H., Eckardt, M. J., & Linnoila, M. (1987). Alcohol intoxication in humans: Effects on vigilance performance. *Alcohol and Alcoholism. Supplement*, *1*, 97–102.
- Roinishvili, M., Chkonia, E., Stroux, A., & Herzog, M. H. (2011). Combining vernier acuity and visual backward masking as a sensitive test for visual temporal deficits in aging research. *Vision Research*, *51*(2011), 417–423.
- Saults, J. S., Cowan, N., Sher, K. J., & Moreno, M. V. (2007). Differential effects of alcohol on working memory: Distinguishing multiple processes. *Experimental and Clinical Psychopharmacology*, *15*(6), 576–587.
- Schulte, T., Miller-Oehring, E. M., Strasburger, H., Warzel, H., & Sabel, B. A. (2001). Acute effects of alcohol on divided and covert attention in men. *Psychopharmacology (Berl)*, *154*(1), 61–69.
- Terry, P., Doumas, M., Desai, R. I., & Wing, A. M. (2009). Dissociations between motor timing, motor coordination, and time perception after the administration of alcohol or caffeine. *Psychopharmacology (Berl)*, *202*(4), 719–729.
- Treue, S., & Martinez Trujillo, J. C. (1999). Feature-based attention influences motion processing gain in macaque visual cortex. *Nature*, *399*(6736), 575–579.
- Wang, G. J., Volkow, N. D., Franceschi, D., Fowler, J. S., Thanos, P. K., Scherbaum, N., et al. (2000). Regional brain metabolism during alcohol intoxication. *Alcoholism, Clinical and Experimental Research*, *24*(6), 822–829.
- Watten, R. G., & Lie, I. (1996). Visual functions and acute ingestion of alcohol. *Ophthalmic and Physiological Optics*, *16*(6), 460–466.
- Wegner, A. J., & Fahle, M. (1999). Alcohol and visual performance. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *23*(3), 465–482.
- Weiner, J. L., & Valenzuela, C. F. (2006). Ethanol modulation of gabaergic transmission: The view from the slice. *Pharmacology & Therapeutics*, *111*(3), 533–554.