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Effects of acute alcohol intoxication on visuospatial attention

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The aim of the present study was to examine the effects of acute alcohol intoxication on the spatial distribution of visual attention measured with simple reaction times (RTs) to targets presented over an extended region of the visual field. Control ($n = 10$) and alcohol groups ($n = 14$) were tested with the same protocol. Participants were tested in two different conditions; in Experiment I, participants were instructed to direct their visual attention to the centre, while in Experiment II they were asked to orient their attention covertly to both right and left, but not to the centre. Throughout participants were required to fixate a small cross in the centre of the computer screen. In the alcohol group, participants received an alcohol dose of 0.4 g/kg so as to produce a blood alcohol concentration (BAC) in the range of 0.08% during the experiments. The spatial distribution of RTs was analysed graphically with geostatistical methods and statistically through analysis of variance of particular regions of the visual field. Results showed that controls were able to direct their attention tightly towards the centre (Expt I) and also to divide attention (Expt II) to the right and left. Participants in the alcohol group fixed their attention more diffusely in the centre (Expt I) and were unable to disengage attention from the centre in Experiment II. We conclude that acute alcohol intoxication impairs the ability to dissociate attention from gaze. *Human & Experimental Toxicology* (2007) **26,** *311–319*

Key words: acute alcohol intoxication; divided attention; geostatistics; simple reaction times; spatial distribution; visual attention

Introduction

The effects of alcohol on task performance have been extensively investigated. However, we do not known precisely all the mechanisms by which this drug exerts its effects. Although alcohol use, in general, is socially accepted, countless deleterious effects have been recognized. Acute alcohol intoxication is closely associated with car accidents, as well as antisocial acts and risk attitudes.[1–4] The role of alcohol in such behaviours has been related with the negative effects of this drug on cognitive processes, especially on attention.[5–8]

In the past 20 years visual attention has been described in terms of varied metaphors, including a spotlight,[9] a zoom lens[10] and a gradient field.[11,12] An essential aspect of all these metaphors is the distribution of attentional resources in the visual field, which is not necessarily determined by the fixation point, and may depend on both external events and internal mental processes.

A much-debated question is whether the flexibility of attentional distribution in space includes the possibility of attending to two separate areas, to the exclusion of the space between them. Awh and Pashler, for example, present recent evidence in favour of the possibility of split attentional foci,[13] whereas several prior experiments seemed to question this possibility.[14–17] Other evidence in favour of the two spatially separate attentional foci comes from Müller *et al*., who recorded evoked potentials in the visual primary cortex of human subjects, showing elegant contour maps of cortical activity as the subjects were exposed to different stimuli.[18] Further recent research has proposed a more flexible view of this subject. For instance, Gobell *et al*. studied a task where subjects should spread their attention to multiple disjoint locations.[19] In

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order to reinforce the division of attention, subjects were forced to ignore distracters positioned in the unattended locations; with this procedure subjects were able to promote a more efficient suppression of the intervening regions, obtaining a clearer distribution of attention to separate locations. Additionally, they found that this capacity of splitting attention to multiple locations could be influenced by many other factors such as spatial frequency and target eccentricity. Kraft *et al*. found further evidence in favour of this.[20] According to these authors, spatial distribution of attention is also influenced by task difficulty and by where attentional targets are located over hemifields, so many factors determine how visual attention is distributed in space.

One aspect that is frequently emphasized is how alcohol could affect allocation of visual attention (see Koelega for review).[21] Although it is well known that alcohol impairs the performance in tasks that require attention, the evidence indicates that alcohol does not uniformly impair all aspects involved in selective attention. For instance, Fillmore *et al*. dissociated the effects of alcohol on controlled and automatic allocation of attention.[22] They found that alcohol reduces the influence of controlled processes whereas it increases the influence of automatic, non-voluntary processes. This view is supported by others, and there is general agreement that alcohol consumption impairs voluntary or cognitive control of attention.[23–26]

Alcohol seems to impair the ability to voluntarily allocate and use attention in a variety of tasks; however, such effects are more pronounced when dealing with competing demands, ie, when subjects are asked to detect a stimulus while executing another task. For example, Moskowitz and Sharma assessed the effects of alcohol on peripheral vision.[27] They measured the time required to detect a stimulus located in the periphery while, at the same time, a central task was being executed, which consisted in counting the blinks of a central light. They showed a performance decrement in intoxicated subjects only when the detection task was simultaneous with the counting task. No impairment was observed when the detection task was performed alone, without the central blink counting. Post *et al*. also theorized that alcohol might constrict the spatial distribution of attention, impairing intoxicated subjects to notice stimuli occurring in their peripheral field of vision.[28] They measured the manual reaction times (RTs) to the offset of stimuli positioned in distinct eccentricities. They found that the higher the eccentricity, the slower was the response, both in controls and in the intoxicated group. Moreover, intoxicated subjects showed greater increases in RTs to the most peripheral stimuli. Thus, acute consumption of alcohol affects allocation of spatial attention, and particularly impairs responses to peripheral stimuli.

The assumptions above are in agreement with the alcohol myopia model.[5,29] According to this model, attentional capacity is limited during acute alcohol intoxication, making it impossible to properly encode all relevant stimuli in the environment. Thus, the limited attentional resources are preferentially allocated to solve the primary task or to process the stimuli that are more immediate. Consequently, fewer resources will be available for secondary task or stimuli, which are not related with the central task, resulting in performance impairment. Consistent with this hypothesis, much research has demonstrated that tasks requiring subjects to divide their attention across distinct spatial locations or to more than one task are severely impaired by acute alcohol intoxication.[7,21,30] Moreover, intoxicated subjects seem to give priority to processing of primary or central tasks, even in a situation where the secondary task has emotional significance.[31,32] All these findings contribute evidence in favour of the hypothesis that alcohol produces a narrowing of cognitive processing, leading to an increment in the sensitivity to direct cues and a decrement in the sensitivity to information outside the main focus.

In order to apply these theoretical ideas to real situations, more research is needed. In particular, an important aspect that requires clarification is the way that alcohol generally affects the spatial distribution of attention. Particularly interesting is the critical situation of attentional division, where two targets must be simultaneously attended in distinct and separated positions. Although the effect of alcohol on behaviour has been attributed to the restriction of the attentional focus, no adequate method has been used to properly measure this effect.

In this study we use a novel graphical approach to the question of attentional distribution, based on geostatistical analysis.[33–40] This method has been widely used in geographic phenomena (eg, petroleum geology, hydrogeology, oceanography and agriculture) and is useful when data present spatial dependence, ie, when data values that are close spatially show less variability than data values that are far away from each other.

Our aim here is to investigate how the spatial distribution of visual attention is affected by acute alcohol intoxication, measuring simple RTs to stimuli presented over a large portion of the visual field.

Material and methods

Human subjects, equipment and procedure

Graduate and undergraduate students ($n = 24$) from the University of São Paulo participated in this study. They were divided into two groups: control ($n = 10$) and alcohol ($n = 14$). All subjects in the alcohol group were previously screened on the basis of their drinking habits and medical history. Inclusion criteria: 1) 20/25 Snellen best-corrected visual acuity or better, 2) absence of known ophthalmological pathologies, 3) absence of any medical condition that might contraindicate alcohol use, 4) right handed and 5) right eye dominance. Exclusion criteria: any history of alcohol problems or other drug dependency. The pattern of alcohol consumption was assessed through individual interviews and classified according to Cahalan and Cisin.[41] Thus, only moderate social drinkers were tested. Also, each subject had their body mass index [$BMI = Wt/Ht^2$] calculated in order to exclude overweight or extremely thin individuals.[42] All the procedures were approved by the local ethics committee and, before the experiment, subjects signed the informed consent.

RTs were measured with the software E-Prime v 1.1 (Psychology Software Tools, Inc., Pittsburg, USA)[43] using a 19' Samsung 997 DF monitor powered by a PC Athlon XP 2400/512 driven by a 10-bit Matrox P650 graphics board with a refresh rate of 100 Hz and a resolution of 800 × 600. Participants' responses (button presses) were registered with millisecond accuracy[44] through a joystick connected to the game port of the PC. Participants were positioned in front of the monitor and maintained in this position during the experiment with their heads stabilized by a chin rest. An infrared camera was used to monitor eye movements during the experiments, and trials were discarded if the eye deviated more than 1° from the fixation point.

All experimental sessions were performed in a darkened, sound-attenuated room. Participants viewed stimuli with their dominant right eye, and were requested to respond as quickly as possible to the onset of the target by pressing a button on the joystick with the index finger of the dominant right hand. Prior to data collection, subjects went through a training session allowing them to become acquainted with the procedure. Subjects were required to keep their gaze on a small cross in the centre of the computer screen. The target to which subjects had to respond as quickly as possible was a white dot, subtending 0.2° of visual angle, with a luminance of 80 cd/m^2, against a black background presented at different positions on the computer screen. Stimulus duration was brief (100 ms) to avoid eye movements and concomitant attentional shifts.[18] RTs below 150 ms and above 500 ms were discarded.

In Experiment I participants were instructed to direct their visual attention towards a grey square frame subtending 4° of visual angle, containing 80 target positions, and centred at the fixation cross; the rest of the screen had 78 target positions (Figure 1A).

In Experiment II participants were instructed to keep their gaze in the fixation cross in the centre of the screen but to direct their attention simultaneously towards two grey square frames subtending 4° of visual angle, each one containing 41 target positions, centred 10° to the right and left of the

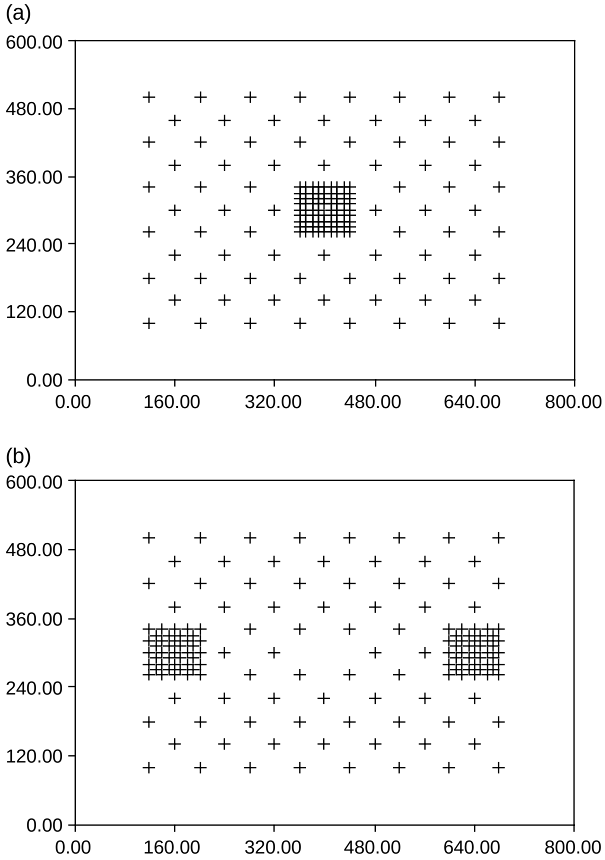

Figure 1 (A) Grid of positions where stimuli were presented in the Experiment I. The central area of the computer screen, where participants must direct their attention, were densely distributed when compared to the sparsely distributed points in the rest of the screen; x and y in pixels units. (B) Experiment II positions grid where stimuli were presented. Here there are two regions densely distributed, where participants must direct their attention, both right and left and the same sparsely distributed points on the rest of the screen as in Experiment I; x and y in pixels units.

fixation cross; the rest of the screen had 72 target positions (Figure 1B).

Experiments were carried out in a counterbalanced way, ie, half of the participants in both groups were tested first in Experiment I and then in Experiment II, while the other half of participants were tested in the reverse order.

Figure 1 shows the positions where the stimuli were presented in the two experiments. The grid of stimuli subtended 24° by 16° of visual angle, and each point in the grid was presented only once in each experiment, thus Experiment I consisted of 158 trials and Experiment II consisted of 154 trials. The interstimulus interval was randomly assigned between 750 and 1500 ms.

All procedures, except alcohol administration, were the same for the control and for the alcohol group. In the alcohol group, participants were required not to drink ethanol for 24 hours and abstained from food for 2 hours prior to the testing. They were informed about the amount of alcohol to be administrated and the expected symptoms. Before beginning the experimental session, each participant drank a mixture of vodka (Stolichnaya®) containing 40% alcohol by volume and orange juice in a 1:1 ratio. The amount of alcohol to be consumed by each subject to reach a peak blood alcohol concentration (BAC) of approximately 0.08% during the test phase was calculated using a formula based upon the subject's weight (0.4 g of ethanol per kg of body weight). Participants were instructed to drink the beverage within a period of 5 min and had to wait, after that, 25 min before data collection.

BAC was measured only in the alcohol group and the BAC values were obtained indirectly by means of a breath alcohol analyser (CA 2000®). The first measurement was taken 10 min after the subjects had finished drinking. Following that first measurement a total of 11 further readings were taken at 5-min intervals. Thus, this procedure resulted in 12 alcohol concentration values (numbered from 1 to 12 covering the period from 10 to 65 min after terminating alcohol ingestion). Thus, some measurements were acquired before (pre-test phase), during (test phase) and after (post-test phase) the end of the acquisition of the experimental data. The critical measurements of the 'test phase' were those obtained immediately before the beginning of data collection. Once the tests were finished, participants left the laboratory only after their BAC was below 0.03%.

Data analysis

Data were analysed with standard commercial statistical software (SigmaStat 1.0 and Statistica 6.0).

A two-way repeated measures analysis of variance (ANOVA) was used to verify global differences between groups and experiments.

In order to verify if the qualitative difference in the geostatistical maps correspond to significant differences in RTs, we decided to run another ANOVA. The aim of this second ANOVA was to verify whether RTs of the central and lateral regions of interest (ROIs, ie, the $4^\circ \times 4^\circ$ squares defined by grey frames, see Figure 1) were different, and how they were influenced by the experimental conditions (Expts 1 and 2) for both experimental groups (control and alcohol). In these analyses, we calculated the mean RTs obtained for each subject in the central ROI and those obtained in the lateral ROIs. It is important to mention that RTs of both lateral (left and right) ROIs were pooled together in this analysis. Therefore, in this ANOVA, group (control and alcohol) was used as a between-subject factor, and instructions (pay attention to the centre – Expt. 1, and pay attention toward the laterals – Expt. 2) and ROIs (central and laterals) were used as within-subject factors. Moreover, planned comparisons were made, within each group, to check the effects of experimental condition on RTs for both central and laterals ROIs.

RTs were also analysed through a geostatistical method using GeoVisual software version 2.2.[35] Geostatistics is useful whenever data show spatial dependence, ie, whenever data values that are close spatially show less variability than data values that are farther away from each other. The exact nature of this pattern varies from experiment to experiment. The variability of the data as a function of distance between points is a function called semivariogram, $\gamma(h)$, given by:

$$\gamma(h) = \frac{1}{2n} \sum_{i=1}^{n} [Z(x+h) - Z(x)]^2$$

where n is the number of pairs of points separated by a distance h; $Z(x)$ is the value of the regionalized variable (data value) at point x; and $Z(x+h)$ is the value of the regionalized variable at point $(x+h)$. A detailed description of the geostatistical analysis is given in Goovaerts.[34]

A unifactorial (12 levels) ANOVA was used to analyse variations on BAC, only in the alcohol group as a function of time following alcohol administration. Thus, BAC measurements (moments from 1 to 12) were used as within-subjects factor. When necessary, we also performed a post hoc analysis using the Newman–Keuls method. The significance level adopted was $P < 0.05$.

Results

Error rate
Trials that were exclude (RT <150 ms or >500 ms) or trials where eye movements occurred constituted less than 4% of the total number of trials in both experimental conditions and for both alcohol and control groups.

Blood alcohol concentration
Analysis of variance of BAC values measured at 12 moments following alcohol administration showed a statistically significant difference, as would be expected during the hour following alcohol administration [$F_{11,143} = 12.41$; $P < 0.01$]. However, the post hoc analysis showed that the mean BACs obtained during the time participants performed Experiments I and II (moments 4–7) did not differ from each other ($P > 0.05$ for all). Differences were only obtained when BACs of the test phase were compared to BACs obtained for the pre-test and/or post-test phase. Thus, the BAC values during the attentional tests did not vary significantly, presenting a mean value of 0.08 ± 0.01%.

RTs
Two-way repeated measures ANOVA showed that globally controls did not differ in their overall RTs from the alcohol group [$F_{1,22} = 0.016$; $P = 0.897$], nor was any difference present between Experiments I and II [$F_{1,1} = 0.048$; $P = 0.827$]. These global results are shown in Figure 2.

The second ANOVA, however, showed that ROIs [$F_{1,22} = 18.73$; $P < 0.001$] was a significant source of variance, and also the interactions between instructions and ROIs [$F_{1,22} = 10.29$; $P = 0.004$], and the triple interaction among group, instructions and ROIs [$F_{1,22} = 5.03$; $P = 0.03$]. In the factor ROIs, RTs for central stimuli were shorter (278 ± 31 ms) than those obtained for lateral ones (295 ± 26 ms). The triple interaction means that the effects of the instructions and ROIs between the two groups were different. Figure 3 shows the main results of this ANOVA. The planned comparisons confirm and extended the ANOVA results showing that: 1) Control group – RTs obtained for the lateral stimuli were significantly shorter ($P = 0.009$) when subjects were instructed to pay attention toward the lateral ROIs (283 ± 24 ms), in relation to the instruction to pay attention to the centre (310 ± 35 ms). For central stimuli, the difference between the instruction to pay attention to the centre (271 ± 20 ms) and that to pay attention toward the sides (302 ± 46 ms) also was marginally significant ($P = 0.053$). Therefore, for the control groups, it is clear that the instructions of Expts I and II caused differences in RTs in both ROIs (Figure 3A). 2) Alcohol group – RTs obtained for the central stimuli were not affected by the instructions of Expts I and II: RTs obtained when the instruction was to pay attention to the centre (273 ± 26 ms), did not differ from those when the instruction was to pay attention toward the sides (268 ± 31 ms, $P = 0.692$). This means that, for this group, independently of the instructions, attention was always strong in the centre. For lateral stimuli there was also marginal significance ($P = 0.061$) for shorter RTs when subjects were instructed to pay attention toward the lateral ROIs (286 ± 18 ms), in relation to the instruction to pay attention to the centre (302 ± 45 ms) (Figure 3B). Taken together, the data obtained for the alcohol group suggest that, despite the instructions they received, they

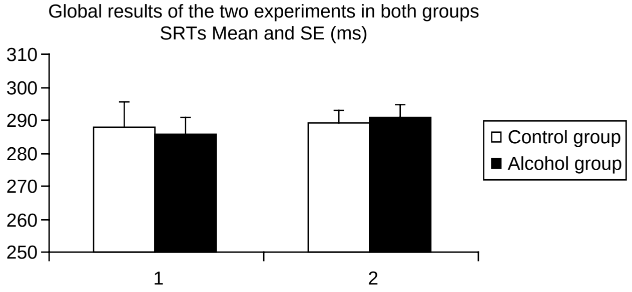

Figure 2 Data (mean RTs in ms) from Experiments I and II for controls and alcohol group

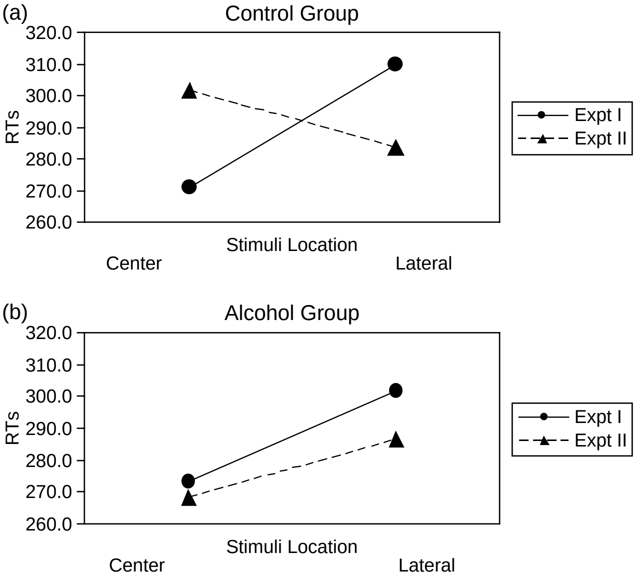

Figure 3 (A) Mean RTs for ROIs in Experiments I and II for the control group. (B) Mean RTs for ROIs in Experiments I and II for the alcohol group.

always maintained their attention around the point of gaze.

The data were also submitted to graphical analysis using geostatistical methods and then plotted as pixel maps. Maps of Experiment I were not very different for the alcohol and the control groups, except that the attentional focus of the alcohol group appeared to be more diffuse than that of controls. Maps of Experiment II, however, showed a remarkable difference between the two groups. Whereas controls were able to split their attentional focus to both lateral targets (leaving the centre relatively unattended), characterizing a divided attention situation, the alcohol group was not able to do so. Their main attentional focus was coincident with the fixation point at all times. Figure 4A–D show the pixel maps obtained through ordinary kriging of Experiments I and II in both groups. Greyscale coding indicates shorter RTs as lighter areas and longer RTs as darker areas, indicating higher or lower attentional focus respectively.

Discussion

Acute alcohol intoxication disrupts the distribution of spatial attention, as shown in the pixel map of Experiment II (Figure 4D). Participants in the alcohol group maintained their attention tightly focused near the point of gaze, and thus presented impairment in endogenously preferentially orienting attention to peripheral regions, as shown not only by the pixel maps but also by the second ANOVA.

This tendency of intoxicated subjects to allocate their attention always around their point of gaze can be related to other findings. For example, Wegner and Fahle have demonstrated that alcohol-intoxicated subjects take more advantage of a gap condition than sober volunteers in saccadic responses, ie, they show an increased gap effect.[45] The gap effect is the reduction observed in saccadic RTs when 200 ms before the onset of a peripheral target, the fixation point disappears (gap condition), relative to the condition with no fixation point offset

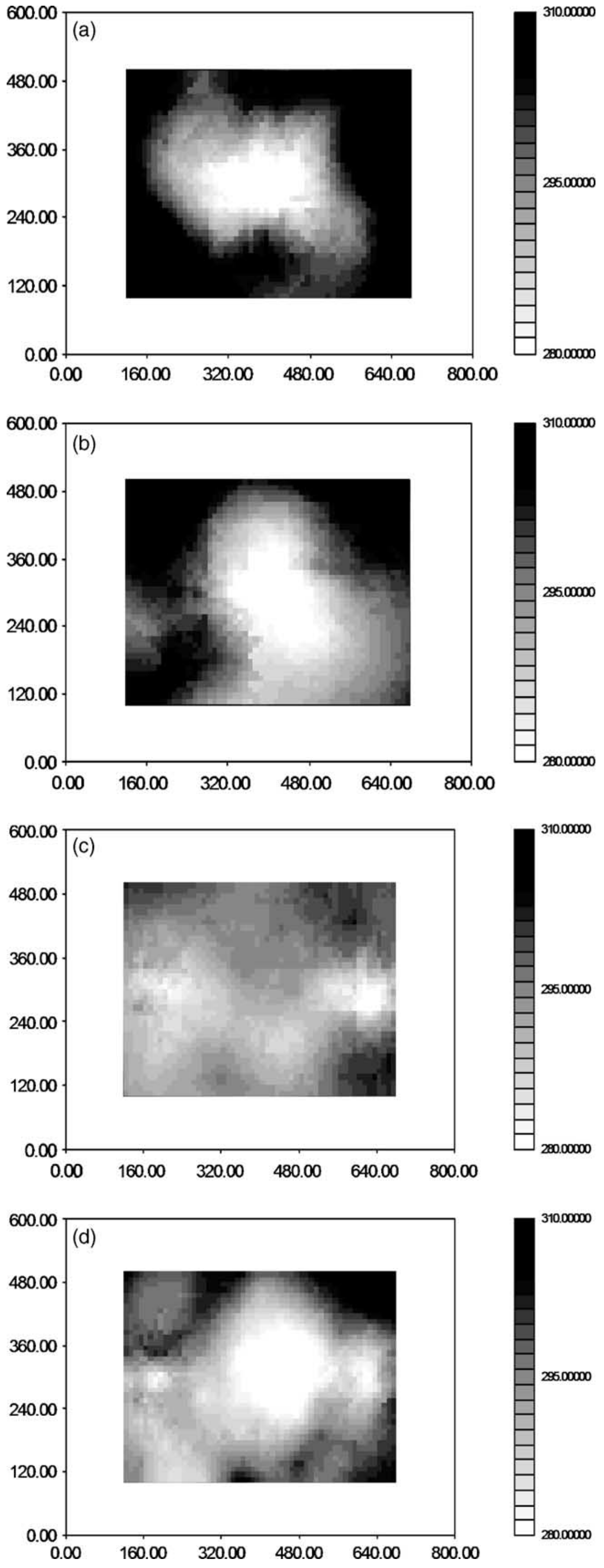

Figure 4 (A) Pixel map of Experiment I for controls shows clearly shorter RTs in the centre coincident with the attentional focus. (B) Experiment I for alcohol group shows the same pattern but with a slightly bigger focus. (C) In experiment II controls were able to orient their attention to the two lateral targets leaving the central region unattended. (D) Experiment II's pixel map clearly shows a remarkable difference when compared to controls; it was not possible for participants to disengage their attentional focus from the centre where the fixation point was located, ie, where their gaze were located.

(overlap condition).[46–48] Some authors interpret this latency reduction as a consequence of the disengagement of attention induced by the fixation point offset.[47,49,50]

According to this view, while attention is disengaged, the detection of any other stimuli will be facilitated and thus the saccadic response time would decrease. Wegner and Fahle showed that the amount of reduction in saccadic response time obtained in the gap condition for controls was 8% of the latency in the classic condition.[45] On the other hand, the gain obtained under the influence of alcohol reached 18%. This finding is consistent with the supposition that the sensitivity of intoxicated subjects to foveal stimuli is increased and that they tend to allocate attentional resources near the fovea.

An alternative explanation is based on the alcohol myopia model,[5] as mentioned in the introduction. According to this model, attentional capacity is reduced in intoxicated subjects, making it difficult to properly process all the relevant stimuli in the environment. This assumption gives rise to two possible explanations for the effects of alcohol on attentional tasks. In complex situations, especially when the primary task presents a high level of difficulty, inebriated subjects will allocate their limited attentional resources in the most important stimulus or the primary task. As result, the processing of secondary tasks or the effect of distracters will be greatly impaired. On the other hand, when the primary task is simple and low-demanding, more resources will be available for processing distracters and its disruptive effect on the primary task will become more evident. In our experiments, subjects did not execute a high demanding task. Thus, their primary task in Experiment II was to covertly orient their attention to the sides, in order to facilitate the visual perception of the stimulus that would probably appear in the attended location. Although sober participants did not have any problems with this task, intoxicated participants had difficulty in properly orienting their attention to the indicated location shifting away from the fixation point. The effects of alcohol on the spatial spread of visual attention were clearly observed in the pixel map of Experiment II and in the second ANOVA. According to our interpretation, owing to the low-demanding characteristic of the primary task in our experiments, intoxicated participants were not able to ignore the ‘distractor stimulus’ – the fixation point, presented in the fovea. Thus, alcohol would impair performance on attention by compromising the ability to direct attention to relevant positions (the targets) and away from the irrelevant one (the fixation point).

According to Kraft *et al*., targets positioned in the same hemifield impair splitting of attention.[20] Thus, it is easier to split attention across targets in opposite hemifields than in the same hemifield. In agreement with this supposition, in our paradigm the stimuli were positioned in different hemifields and in the control situation we found evidence in favour of divided attention.[51] In addition, it is also postulated by Kraft *et al*. that divided attention is more likely to occur in a task with high-demanding characteristic.[20]

In summary, our study showed that pixel maps obtained through geostatistical analysis are useful to show, with good resolution and precise location of the focus(i) of visual attention, that alcohol intoxication affects the capacity to voluntarily allocate visual attention in space. These effects were evident in Experiment II where participants were required to divide their attention between two spatial locations before target appearance. In this condition, controls were able to split their attentional focus according to the instructions and responded faster to stimuli appearing in both locations. Intoxicated participants, on the other hand, presented impairment in this situation, focusing their attention primarily on the fixation point, ie, they did not seem able to disengage their attention form their gaze. Although alcohol-related performance decrements are not restricted to only this situation, these results are compatible with those of others researchers who showed that alcohol is related to impairment in dividing attention.[7,21,42]

Geostatistical methods constitute a useful tool to analyse the effects of acute alcohol intoxication on the spatial distribution of visual attention. Through this method it was possible to visualize alcohol’s effects on attention with a new perspective through pixel maps. It was possible to demonstrate that alcohol impaired disengagement of the focus of visual attention from gaze. Thus it appears that alcohol promotes an ‘attentional anchorage’ to the location of the eye gaze.

In conclusion, in the present study we were able to demonstrate an attentional effect of acute alcohol intoxication. Geostatistics thus constitutes a promising resource to investigate attentional effects of other neurotoxicants and psychotropic drugs.

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References

1 Híjar M, Flores M, López MV, Rosovsky H. Alcohol intake and severity of injuries on highways in Mexico: a comparative analysis. *Addiction* 1998; **93**: 1543–51.
2 Giancola PR, Zeichner A, Yarnell JE, Dickson KE. Relation between executive cognitive functioning and the adverse consequences of alcohol use in social drinkers. *Alcohol Clin Exp Res* 1996; **20**: 1094–98.
3 Cohen DA, Mason K, Scribner R. The population consumption model, alcohol control practices and alcohol-related traffic fatalities. *Prev Med* 2001; **34**: 187–97.
4 Farquhar K, Lambert K, Drummond GB, Tiplady B, Wright P. Effect of ethanol on psychomotor performance and on risk taking behaviour. *J Psychopharmacol* 2002; **16**: 379–84.
5 Steele CM, Josephs RA. Alcohol myopia: its prized and dangerous effects. *Am Psychol* 1990; **45**: 921–33.
6 Herzog TA. Effects of alcohol intoxication on social inferences. *Exp Clin Psychopharmacol* 1999; **7**: 448–53.
7 Rosselló J, Munar E, Justo S, Arias R. Effects of alcohol on divided attention and on accuracy of attentional shift. *Psychol Spain* 1999; **3**: 69–74.
8 Bartholow BD, Pearson M, Sher KJ, Wieman LC, Fabiani M, Gratton G. Effects of alcohol consumption and alcohol susceptibility on cognition: a psychophysiological examination. *Biol Psychol* 2003; **64**: 167–90.
9 Posner MI. Orienting of attention. *Q J Exp Psychol* 1980; **32**: 3–25.
10 Eriksen CW, St. James JD. Visual attention within and around the field of focal attention: a zoom lens model. *Percept Psychophys* 1986; **40**: 225–40.
11 Downing CJ, Pinker S. The spatial structure of visual attention. In Posner MI, Marin OSM eds. *Attention and performance XI: Mechanisms of attention*. Erlbaum, 1985.
12 LaBerge D. *Attentional processing: the brain's art of mindfulness. Perspectives in cognitive neuroscience*. Harvard University Press, 1995.
13 Awh E, Pashler H. Evidence for split attentional foci. *J Exp Psychol Hum Percept Perform* 2000; **26**: 834–46.
14 Eriksen CW, Yeh YY. Allocation of attention in the visual field. *J Exp Psychol Hum Percept Perform* 1985; **11**: 583–98.
15 Pan K, Eriksen C. Attentional distribution in the visual field during same-different judgments as assessed by response competition. *Percept Psychophys* 1993; **53**: 134–44.
16 Heinze H, Luck SJ, Muente TF, Goes A, Mangun GR, Hillyard SA. Attention to adjacent and separate positions in space: An electrophysiological analysis. *Percept Psychophys* 1994; **56**: 42–52.
17 Eimer M. Attending to quadrants and ring–shaped regions: EPR effects of visual attention in different spatial selection tasks. *Psychophysiology* 1999; **36**: 491–503.
18 Müller MM, Malinowski P, Gruber T, Hillyard SA. Sustained division of the attentional spotlight. *Nature* 2003; **424**: 309–12.
19 Gobell JL, Chia-huei T, Sperling G. The spatial distribution of visual attention. *Vision Res* 2004; **44**: 1273–96.
20 Kraft A, Müller NG, Hagendorf H, Schira MM, Dick S, Fendrich RM, Brandt SA. Interactions between task difficulty and hemispheric distribution of attended locations: implications for the splitting attention debate. *Cognit Brain Res* 2005; **24**: 19–32.
21 Koelega HS. Alcohol and vigilance performance: a review. *Psychopharmacology* 1995; **118**: 233–49.
22 Fillmore MT, Vogel-Sprott M, Gavrilescu D. Alcohol effects on intentional behavior: dissociating controlled and automatic influences. *Exp Clin Psychopharmacol* 1999; **7**: 372–78.
23 Peterson JB, Rothfleish J, Zelazo PD, Pihl RO. Acute alcohol intoxication and cognitive functioning. *J Stud Alcohol* 1990; **51**: 114–22.
24 Giancola PR. Executive functioning: a conceptual framework for alcohol related aggression. *Exp Clin Psychopharmacol* 2000; **8**: 576–97.
25 Casbon TS, John JC, Lang AR, Patrick CJ. Deleterious effects of alcohol intoxication: diminished cognitive control and its behavioral consequences. *J Abnorm Psychol* 2003; **112**: 476–87.
26 Curtin JJ, Fairchild BA. Alcohol and cognitive control: implications for regulation of behavior during response conflict. *J Abnorm Psychol* 2003; **112**: 424–36.
27 Moskowitz H, Sharma S. Effects of alcohol on peripheral vision as a function of attention. *Hum Factors* 1974; **16**: 174–80.
28 Post RB, Lott LA, Maddock RJ, Beede JI. An effect of alcohol on the distribution of spatial attention. *J Stud Alcohol* 1996; **57**: 260–66.
29 Josephs RA, Steele CM. The two faces of alcohol myopia: attentional mediation of psychological stress. *J Abnorm Psychol* 1990; **99**: 115–26.
30 Schulte T, Müller-Oehring EM, Strasburger H, Warzel H, Sabel BA. Acute effects of alcohol on divided and covert attention in men. *Psychopharmacology* 2001; **154**: 61–69.
31 Curtin JJ, Lang AR, Patrick CJ, Strizke WGK. Alcohol and fear-potentiated startle: the role of competing cognitive demands in the stress-reducing effects of intoxication. *J Abnorm Psychol* 1998; **107**: 547–57.
32 Curtin JJ, Patrick CJ, Lang AR, Cacioppo JT, Birbaumer N. Alcohol affects emotion through cognition. *Psychol Sci* 2001; **12**: 527–31.
33 Journel AG. Geostatistics: models and tools for the earth sciences. *Math Geol* 1986; **18**: 119–40.
34 Goovaerts P. *Geostatistics for natural resources evaluation*. Oxford University Press, 1997.

35 Yamamoto JK. An alternative measure of the reliability of ordinary kriging estimates. *Math Geol* 2000; **32**: 489–509.

36 Fuchs K, Deutz A. Use of variograms to detect critical spatial distances for the Knox's test. *Prev Vet Med* 2002; **54**: 37–45.

37 Ludovisia A, Minozzo M, Pandolfia P, Taticchia MI. Modelling the horizontal spatial structure of planktonic community in Lake Trasimeno (Umbria, Italy) using multivariate geostatistical methods. *Ecol Modelling* 2005; **181**: 247–62.

38 Miranda ML, Dolinoy DC. Using GIS-based approaches to support research on neurotoxicants and other children's environmental health threats. *Neurotoxicology* 2005; **26**: 223–28.

39 Mather FJ, White LE, Langlois EC, Shorter CF, Swalm CM, Shaffer JG, Hartley WR. Statistical methods for linking health, exposure, and hazards. *Environ Health Perspect* 2004; **112**: 1440–45.

40 Canto-Pereira LHM, Dias H, Ranvaud R. A geostatistical approach to assess the spatial distribution of visual attention. *Invest Ophthalmol Vis Sci* 2005; **46**: 5653.

41 Cahalan D, Cisin IH. American drinking practices: summary of findings from a national probability sample: I. Extent of drinking by population subgroup. *Q J Stud Alcohol* 1968; **29**: 130–51.

42 Kuczmarski RJ, Flegal KM. Criteria for definition of overweight in transition: background and recommendations for the United States. *Am J Clin Nutr* 2000; **72**: 1074–81.

43 Schneider W, Eschman A, Zuccolotto A. *E-Prime user's guide*. Psychology Software Tools Inc, 2002.

44 Segalowitz SJ, Graves R. Suitability of the IBM XT, AT and PS/2 keyboard, mouse, and game port as response devices in reaction time paradigms. *Behav Res Methods Instrum Comput* 1990; **22**: 283–89.

45 Wegner AJ, Fahle M. Alcohol and visually guided saccades: gap effect and predictability of target location. *Psychopharmacology* 1999; **146**: 24–32.

46 Saslow MG. Effects of components of displacement-step stimuli upon latency of saccadic eye movements. *J Opt Soc Am* 1967; **57**: 1024–29.

47 Fischer B, Weber H. Express saccades and visual attention. *Behav Brain Sci* 1993; **16**: 553–610.

48 Faria AJP Jr, Machado-Pinheiro W. Looking for the gap effect in manual responses and the role of contextual influences in reaction time experiments. *Braz J Med Biol Res* 2004; **37**: 1175–84.

49 Mayfrank L, Mobashery M, Kimming H, Fischer B. The role of fixation and visual attention in the occurrence of express saccades in man. *Eur Arch Psychiatry Neurol Sci* 1986; **235**: 269–75.

50 Mackeben M, Nakayama K. Express attentional shifts. *Vis Res* 1993; **33**: 85–90.

51 Canto-Pereira LHM, Ranvaud R. Splitting the beam? Evidence of divided visual attention assessed through a geostatistical method. *Perception* 2005; **34**: 178–79.