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Moderate alcohol consumption in humans impairs feature binding in visual perception but not across perception and action

Lorenza S. Colzato*, Vicki Erasmus, Bernhard Hommel

Department of Psychology, Cognitive Psychology Unit, Leiden University, Postbus 9555, 2300 RB Leiden, The Netherlands

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Abstract

Animal studies suggest a relationship between activation of the cholinergic system and neural synchronization, which again has been suggested to mediate feature binding. We investigated whether suppressing cholinergic activity through moderate alcohol consumption in healthy humans affects behavioral measures of feature binding in visual perception and across perception and action. Indeed, evidence of the binding of shape and color, and of shape and location, of visual objects disappeared after alcohol consumption, whereas bindings between object features and the manual response were unaffected.

Keywords: Alcohol; Binding problem; Visual perception; Neuronal synchronization; Sensory-motor integration; Cholinergic system

Imagine that you are watching a red cat and a black dog. Given that colors, shapes, locations, and semantic features are processed in different cortical areas, how does our brain correctly integrate the features belonging to the same event but does not, say, make you perceive a red dog and a black cat? To solve this feature-binding problem the brain needs to employ some mechanism that interlinks and integrates the neural patterns coding the features of a given perceptual event [19]. One such mechanism might be the neural synchronization of cell populations [1,2]. That is, the firing rates of cells coding features of the same perceptual event may synchronize, which would provide a neural marker of 'eventhood' and support the individual codes in their competition with other codes in their respective feature domains. Indeed, transient increases of synchronization in the gamma frequency range have been observed in perceptual tasks like figure-ground distinctions and feature binding [2], switching between bistable visual figures [8], or the retention of visual patterns in short term memory [18]. Even motor tasks have revealed reliable links between neural synchronization and integrative cognitive processes such as the planning of multi-featured actions [3].

If the idea that neural synchronization at least mediates the binding of perceptual and, perhaps, action features is correct, one would expect that factors that are known to impact synchronization in a particular fashion affect behavioral measures of feature integration in the same way. One such candidate factor is alcohol, which is suspected to cause a hypoactivity of the cholinergic system. Apart from chronic alcohol consumption [10], acute ethanol intake has been found to inhibit muscarinic receptors of the cholinergic system [12,17], which again is involved in driving at least visually induced synchronization [14,15]. The aim of the present study was thus to test whether the intake of alcohol hampers the binding of features and whether this impact is specific to visual features.

We adopted the task from Hommel [4], which involves the repetition of task-related and unrelated visual features and of the response (see Fig. 1). The standard findings are interactions between (a) the task-related stimulus features (e.g. shape, if and only if it signals the response [4], and location, if and only if the responses are spatially defined [5]); (b) the non-spatial stimulus features (e.g. shape and color); and (c) the relevant stimulus features and the response (for an overview, see [7]). The patterns of these interactions all look alike: Performance is impaired in partial-repetition trials, that is, if one stimulus feature (or the response) is repeated while the other is not. This suggests that the mere co-occurrence of a feature-feature or feature-response conjunction is sufficient to create a temporary binding of the respective feature codes – an 'event file' [4,6,7]. Reactivating one member of this binding (as in the case of feature repetitions) reactivates other member(s) as well,

* Corresponding author.
E-mail address: colzato@fsw.leidenuniv.nl (L.S. Colzato).

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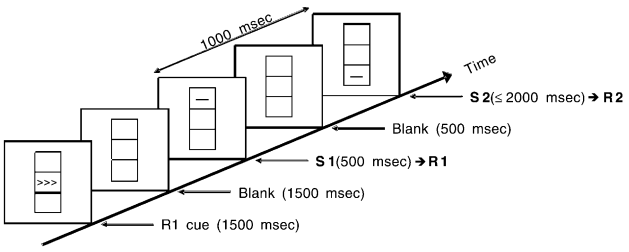

Fig. 1. Sequence of events in the present experiment (cf. [4]). A response cue signaled a left or right key press (R1) that was to be delayed until presentation of S1, a red or green, vertical or horizontal line in a top or bottom box. S2 appeared 1 s later – another red or green, vertical or horizontal line in the top or bottom box. S2 shape signaled R2, also a quick left or right key press. R2 speed and accuracy were analyzed as function of the repetition vs. alternation of stimulus shape, color, and location, and of the response.

which leads to confusion and requires a time-consuming re-binding process in partial-repetition trials. Importantly for present purposes, this partial-repetition cost can be taken to indicate feature–feature and feature–response binding, which is why we chose it as behavioral marker. According to the reasoning outlined above we thus expected moderate alcohol consumption to decrease partial-repetition costs.

Seventeen right-handed volunteers served in two experimental sessions. Informed consent was obtained from all participants after the nature and possible consequences of the study were explained to them, and the protocol was approved by the local ethics committee. Subjects were social drinkers (2–3 units per day on average) in the age range of 20–30 years, they were healthy non-smokers, not on medication or drugs, and without neurological or psychiatric history according to self-report. To minimize circadian-cycle influences, experimental sessions always started at 15:00 h, after subjects had had their regular lunch, but abstained from eating and from drinking caffeine-containing liquids for 2.5 h and abstained from alcohol consumption for 24 h. A double-blind, placebo-controlled, randomized cross-over design with counterbalancing of the order of conditions was used to avoid alcohol-expectancy effects. Sessions were separated by 3–7 days. Placebo and dose alcohol quantities corresponded to 0.00 and 0.45 g/kg, respectively. Body-weight dependent measures of vodka (containing 37.5% ethanol) were dissolved in orange juice such that total liquid volume amounted to 500 cc. Adding peppermint oil [13] and serving the beverage in a sealed milkshake beaker effectively prevented that subjects tasted or smelled the presence of alcohol. Blood-alcohol concentration, recorded before the beginning (after 30 min of drinking) and after the end of the experiment, averaged 0.34‰ (S.D. 0.04) in dose sessions and 0.0‰ (S.D. 0.0) in placebo sessions.

Subjects completed a version of the task adapted from Hommel [4] (see Fig. 1). They faced three gray, vertically arranged boxes in the middle of a monitor and carried out two responses per trial. R1 was a delayed simple reaction with the left or right key, as indicated by a 100% valid response cue (three left- or right-pointing arrows in the middle box) that preceded the trigger stimulus S1 by 3000 ms. S1 varied randomly in shape (a thin vertical or horizontal line), color (red or green), and location (top or bottom box). R1 was to be carried out as soon as S1 appeared, independent of its shape, color, or location; i.e. subjects were encouraged to respond to the mere onset of S1. R2 was a binary-choice reaction to the shape of S2 (vertical or horizontal orientation), which also appeared in red or green, and in the top or bottom box, 1000 ms after S1 onset. Responses to S1 and to S2 were made by pressing the left or right shift-key of the computer keyboard with the corresponding index finger. Each session was composed of a factorial combination of the two possible shapes, colors, and locations of S2, the repetition vs. alternation of shape, color, location, and the response, and three replications per condition.

After excluding trials with missing or anticipatory responses (1.3%), mean reaction times (RTs) and proportions of errors for R2 (i.e. the response to S2) were analyzed as a function of treatment (placebo vs. alcohol) and the repetition vs. alternation of response (R1 → R2), stimulus shape, color, and location (S1 → S2). Replicating earlier findings [4,6], RTs revealed significant interactions between shape and color, $F(1, 16) = 6.01$, $P < 0.05$, and shape and location, $F(1, 16) = 8.52$, $P < 0.01$ – repeating one but not the other feature slows down responding. However, apart from an interaction with shape, $F(1, 16) = 5.69$, $P < 0.05$, treatment was involved in a three-way interaction with shape and location, $F(1, 16) = 7.47$, $P < 0.05$, and in a four-way interaction that also comprised color, $F(1, 16) = 4.93$, $P < 0.05$. To disentangle these effects, separate analyses of variance were run on the data from placebo and alcohol conditions. As obvious from Fig. 2 (top panels), the shape-by-color and shape-by-location interactions were reliable in the placebo condition, $F(1, 16) = 6.53$, $P < 0.05$, and $F(1, 16) = 21.83$, $P < 0.001$, but not in the alcohol condition, $Ps > 0.25$. This can be taken to indicate that the intake of alcohol prevents the binding of visual features to a degree that overt performance is no longer affected. Interestingly, alcohol did not impact effects reflecting stimulus-response bindings: Although we replicated the standard interactions between response and shape, $F(1, 16) = 11.16$, $P < 0.005$, and response and location, $F(1, 16) = 16.12$, $P < 0.001$, these effects were not involved in any interaction with treatment (see Fig. 2, bottom panels). The errors followed the same pattern: Significant interactions were obtained between shape and response, $F(1, 16) = 11.47$, $P < 0.005$, and location and response, $F(1, 16) = 4.65$, $P < 0.05$, due to fewer errors in conditions where the stimulus feature and the response were both repeated or both alternated, as compared to conditions where the stimulus feature but not the response was repeated, or vice versa. Again, these effects were not modified by treatment.

Our results suggest two conclusions: moderate alcohol consumption impairs feature binding and it selectively affects the binding of visual features while sparing cross-domain bindings between visual features and manual

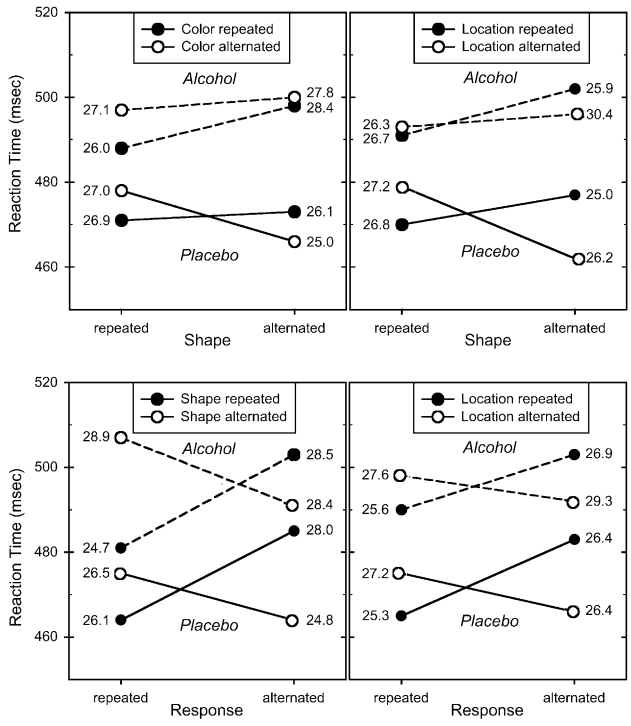

Fig. 2. Mean reaction times (symbols) and their standard errors (numbers) as a function of treatment (placebo vs. alcohol) and the repetition vs. alternation of stimulus shape and stimulus color (top left panel), of stimulus shape and stimulus location (top right panel), of response and stimulus shape (bottom left panel), and of response and stimulus location (bottom right panel). Note that apart from an interaction with shape, treatment affected only the interactions of shape and color and of shape and location.

responses. The finding that alcohol affects feature binding at all supports the hypothesized links between the cholinergic system and neural synchronization [14,15] on the one hand and between synchronization and feature integration [1,2,19] on the other. We can imagine at least two reasons for why the impact of alcohol might be restricted to local, in our case visual-visual binding. First, the cholinergic system may selectively drive neural synchronization in visual or, more generally, in perception-related areas of the cortex but not in areas involving motoric activity. Although we know of no studies that would definitely rule out this possibility, the central role of acetylcholine in voluntary motor control and its rather direct impact on neural activity in the motor cortex [11] renders this possibility somewhat unlikely. A second reason considers the anatomical distance of the to-be-synchronized neural networks. Local integration processes in both visual and motor cortex are commonly associated with synchronization frequencies in the gamma band [2]. In contrast, synchronization between more distant networks, such as in visuomotor integration, has been found to use the lower, beta frequency band [16]. If we assume that alcohol intake impairs neural synchrony by increasing the variability of firing rates, and if we consider that this should affect higher frequencies more than lower frequencies [9], local, short-range bindings should indeed be more vulnerable to alcohol-induced effects than long-range bindings.

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