

Acute alcohol intoxication impairs motor inhibitory control via a disruption of the fronto-parietal attention allocation system

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Impairment in inhibitory control, the ability to suppress irrelevant cognitive and motor processes, is a key symptom of acute alcohol intoxication and is thought to account for the impulsive behaviors typically associated with drunkenness. Yet, the precise effects of alcohol on the functional activity of the executive control brain networks and how these modulations eventually impair inhibitory control remains largely unresolved.

To address this question, we conducted a double-blind, placebo-controlled, randomized trial, assessing the effect of acute alcohol intoxication (0.6g/kg) on the behavioral performance and electrical neuroimaging analyses of event-related potentials recorded in young healthy individuals during a motor inhibition Go/NoGo task. The inhibitory control task consisted in responding as fast as possible by a button press to a set of stimuli while withholding responses to another set of stimuli (No-Go stimuli).

The present thesis focuses on a preliminary analysis of a subgroup of 36 participants and of one of the tasks of a larger research project. Behaviorally we did not find effects of alcohol intake on the average response time during the task (our primary measure of central tendency), but observed a larger distribution of the response time in the alcohol than placebo group. Electrophysiologically, there were two distinct phases distinguishing the functional processing of the inhibitory control stimuli: a significant global field power modulation (at latencies 240-280 and 410-480 ms post-stimulus onset), indicating an effect of alcohol on the synchronized pyramidal neurons' firing rates, as well as two phases of significant topographic modulation (210-240 and 340-450 ms), indicating a modification in the configuration of the underlying neural generators. Source estimation located these effects in the right parietal and the right inferior frontal cortices.

We interpret our results in terms of a reduced attention-allocation to the No-Go stimuli in the alcohol group, via an impairment of response conflict detection and the allocation of top-down mechanisms needed to initiate response inhibition. Alcohol might have impaired conflict evaluation, leading to a lack of adaptation of behavior during the conflicting situation. The localization of our effect in the fronto-parietal executive control network further support the hypothesis of reduced attention allocation and reduced activity in the posterior and, indirectly, in the anterior attention system in the alcohol condition. Considering the importance of the evaluative and regulative component (of the anterior attention system) for an adapted behavior, these findings might explain the disinhibited behavior of drunk people.

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