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Acute alcohol intoxication modulates the temporal dynamics of resting electroencephalography networks

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Abstract

This study aimed to provide a currently missing link between general intoxication-induced changes in overall brain activity and the multiple cognitive control deficits typically observed during acute alcohol intoxication. For that purpose, we analyzed the effects of acute alcohol intoxication (1.1‰) on the four archetypal electroencephalography (EEG) resting networks (i.e., microstates A–D) and their temporal dynamics (e.g., coverage and transitions from one microstate to another), as well as on self-reported resting-state cognition in $n = 22$ healthy young males using a counterbalanced within-subject design. Our microstate analyses indicated that alcohol increased the coverage of the visual processing-related microstate B at the expense of the autonomic processing-related microstate C. Add-on exploratory analyses revealed that alcohol increased transitions from microstate C to microstate B and decreased bidirectional transitions between microstate C and the attention-related microstate D. In line with the observed alcohol-induced decrease of the autonomic processing-related microstate C, participants reported decreases of their somatic awareness during intoxication, which were positively associated with more transitions from microstate C to microstate B. In sum, the observed effects provide mechanistic insights into how alcohol might hamper cognitive processing by generally prioritizing the bottom-up processing of visual stimuli over top-down internal information processing. The fact that this was found during the resting state further proves that alcohol-induced changes in brain activity are continuously present and do not only emerge during demanding situations or tasks.

KEYWORDS
alcohol intoxication, microstates, resting-state EEG

1 | INTRODUCTION

Consuming alcohol is part of many human's social lives.[1] Aside from desirable effects, it is associated with various health risks and socioeconomic costs, and a considerable number of individuals develop an alcohol use disorder (AUD) with continued use.[2,3] AUD, but also social drinking, can result in heavy drinking episodes ("binge drinking"[4]) that may cause significant harm to a person's well-being and functioning in the short term as well as the long term.[5] Hence, we need to better understand how acute high-dose alcohol intoxication,

as occurring during binge drinking, affects our behavior and cognition[6] by modulating neural processing in the central nervous system.[7]

Acute alcohol intoxication has repeatedly been shown to alter and/or impair different cognitive processes like attention and executive functions (including inhibition),[8–11] but experimental studies on high-dose intoxications are still comparatively rare.[12] Although the neuropharmacological effects of acute intoxication are rather well understood,[13–15] general changes in overall brain activity and connectivity (that are not associated with specific tasks or cognitive functions) have remained rather unclear. So far, it has never been investigated whether and how acute alcohol intoxication alters the temporal dynamics of resting electroencephalography (EEG) microstates, which reflect information processing in circumscribed large-scale brain networks in the absence of specific cognitive or behavioral tasks.[16–21] Learning more about alcohol-induced changes in resting network activity might help to explain why and how alcohol intoxication gives rise to various executive and attentional effects. The reason for this is that microstates are considered to represent "fundamental building blocks of cognition"[22] and that resting-state activity seems to strongly determine how response-relevant information will be processed, with each of the four archetypical microstates facilitating different associated neuropsychological processes.[23,24] Hence, reduced resting-state activity in neural networks that are functionally associated with top-down processing (like cognitive control or attention) could provide a plausible explanation as to why intoxication makes it harder to induce and maintain behavioral control in various task contexts. This could provide a valuable and currently missing link between general (task-independent) changes in overall brain activity and the multiple cognitive deficits observed during acute intoxication. Therefore, we set out to investigate the effects of high-dose alcohol intoxication on neural network activity identified by means of a spatiotemporal analysis of multichannel EEG recorded at rest. As a key benefit, this approach allows for studying rapid shifts of intrinsically generated, context-independent activity among distributed neural networks with milliseconds resolution.[16,19,20] Consequently, it paves the way for a comprehensive understanding of alcohol effects on neural processing by tracking how alcohol intoxication modulates the temporal dynamics of activity in large-scale brain circuitry, thereby accounting for the widespread and complex effects of this drug throughout the brain.[21,25]

Different metabolic neuroimaging methods have already revealed widespread modulatory effects of acute alcohol intoxication on neural processing at rest in the spatial domain.[7] Functional magnetic resonance imaging (fMRI) studies suggest that low to moderate doses of alcohol (0.25–0.75 g/kg) increase the functional connectivity within and between sensory processing-related networks (e.g., auditory and visual cortices[26,27]). Studies using positron emission topography (PET) show that low to moderate doses of alcohol decrease global glucose metabolism, with relatively higher decreases in cortical regions of the brain (e.g., visual cortex) and relatively higher increases in several subcortical regions (e.g., amygdala, insula, and striatum[28,29]). Finally, studies using PET or arterial spin labeling (ASL) and low to high (>0.75 g/kg) doses of alcohol report relative increases in regional cerebral blood flow in prefrontal and temporal regions and relative decreases in the cerebellum as well as the occipital cortex.[30–32] These somewhat contradictory findings on alcohol effects (e.g., for the visual cortex; fMRI: increased intrinsic connectivity; PET: decreased glucose metabolism; ASL and PET: decreased blood flow) may be linked to the distinct metabolic neuroimaging methods, as the vasoactive effects of alcohol likely affect the coupling between the hemodynamic response and the underlying local neural activity.[33] Yet, all of these approaches are largely restricted to the detection of overall changes in brain activity/connectivity and thus do not allow to investigate short-lived dynamic changes that provide information on the distinction between bottom-up sensory processing and top-down higher order processing.

Therefore, it might be more promising and reliable to study acute alcohol intoxication effects on intrinsically generated neural activity in the temporal domain by capitalizing on methods providing a more direct measure of neural activity with higher temporal resolution. Electrophysiological studies of alcohol effects on neural processing at rest have a long tradition,[34,35] but most of them have analyzed the effects of alcohol intoxication on brain oscillations by means of frequency analyses, demonstrating that moderate to high doses of alcohol increase both power (alpha,[36] delta,[37] theta[37–39]) and functional connectivity[40] of slow-wave oscillations and decrease an oscillation-related measure of neural noise during the resting state.[41] Adding to this, the spatiotemporal EEG analysis approach applied in this study allows to investigate how high-dose alcohol intoxication modulates amplitude-independent and broadband temporal dynamics of neural resting networks occurring on a milliseconds scale.

More specifically, the applied analysis approach clusters the resting EEG signal into a limited number of scalp electrical potential topographies that remain stable for certain time periods (60–120 ms) and then dynamically change into a different topography that remains stable again.[18] These time periods of stable topographies are referred to as "microstates," and transitions between microstates are thought to represent sequential coordinated activity of various distributed neural networks.[16,42] Of note, just four archetypal topographies (termed microstates A–D[43]), which occur with high reproducibility across multiple independent studies, explain large portions of the global variance in the EEG data (up to 80%). The temporal dynamics of these four microstates (e.g., coverage = presence and transitions = communication between networks) are known to vary across various behaviors, personality types, and neuropsychiatric disorders[17] and are modulated by psychoactive drugs.[20,44–46] To interpret these findings, researchers have sought to link these four microstates to specific underlying neural networks, with microstates A and B being associated with bottom-up sensory processing (microstate A: audition; microstate B: vision) and microstates C and D with top-down processing (microstate C: autonomic processing/saliency and possibly cognitive control; microstate D: attention).[16,17,22] Analyzing how alcohol modulates the temporal dynamics of these four networks could thus help to better understand the neurophysiological mechanisms underlying the behavioral and cognitive changes brought about by this widely used and abused drug.

The current study examined the effects of an acute high-dose alcohol intoxication on neurophysiological processing at rest in a

counterbalanced within-subject design in men, who appear to be more vulnerable toward developing AUD than females (based on commonly reported prevalence rates).[2,33] In two appointments separated by 2–7 days, we assessed 22 young healthy male participants (resting EEG, self-reported mood, and resting-state cognition) while they were either sober or intoxicated. By means of spatiotemporal EEG analysis, we then tested for alcohol-induced differences in the presence (i.e., the percentage of time a given microstate is present) and number of transitions (i.e., the number of observed transitions from one microstate to another; for details, please see Section 2.5) between the four microstates. Given previous reports of intensified processing in sensory processing-related networks after alcohol intoxication,[26,27] we hypothesized that alcohol increases the presence of bottom-up sensory processing-related microstates A and B as well as transitions toward and between these networks. Furthermore, recent evidence suggests that alcohol impairs top-down control-related processing, such as interoceptive-autonomic processing,[47] task-relevant processing of proprioceptive information,[48] and performance monitoring.[49,50] Therefore, we hypothesized that acute alcohol intoxication decreases the presence of microstates C and D, which are commonly assumed to be functionally related to control, as well as transitions toward and between these networks. We further expected that alcohol decreases self-reported somatic awareness (for details, see Section 2). Finally, we tested whether alcohol-induced differences observed in microstate characteristics remained significant when controlling for potential alcohol-induced mood changes.

2 | MATERIALS AND METHODS

2.1 | Participants

We used G*power software[51,52] to determine the required sample size. We conducted an a priori required sample size estimation for repeated-measures ANOVA within-factor effects. Using eight repeated measures (two alcohol states × four microstates), an alpha error probability of 5%, a power of 95%, and the suggested standard medium effect size $f = 0.25$ yielded a required sample size of $n = 23$, which we then increased to $n = 24$ in order to be able to properly balance appointment order. Hence, we recruited $n = 24$ young healthy males at the local university (TU Dresden) using flyers and online advertisements. All participants underwent a telephone screening for the following: Inclusion criteria were male sex, age 18–30 years, and moderate/non-risky drinking habits, as defined by 1–12 binge-drinking incidents (more than eight standard units of alcohol in one evening) per year and 1–15 points in the Alcohol Use Disorder Identification Test (AUDIT).[53] Exclusion criteria were female sex (females had not been approved by the ethics committee), any history of addiction and/or drinking habits that did not match the inclusion criteria, any current somatic, neurologic, or psychiatric disorder, current intake of medication, and recollections of previous aggressive episodes under the influence of alcohol. Participants signed informed consent before the start of each study appointment and were reimbursed with 10€ per hour. The study was approved by the local ethics committee (EK293082014) and conducted in accordance with the Declaration of Helsinki. Of note, the majority of participants included in this study also contributed to the samples of other publications from authors of this paper.[41,54,55]

2.2 | Procedure

We used a counterbalanced within-subject study design, where each participant was assessed once sober and once intoxicated (for details on alcohol administration, please refer to previous studies[41] and/or the supplementary material). The minimum time span in between the two appointments was 2 days, and the maximum time span between the two appointments was 7 days.

Resting-state EEG was recorded for 5 min at each appointment. There were five alternating periods of eyes closed (40 s) and eyes open (20 s), which were instructed by the auditory commands "Augen auf" and "Augen zu," which translate to "close eyes" and "open eyes." The rationale for using this protocol[20,56–58] is that these alternations minimize fluctuations in participants' vigilance state, as participants may already become drowsy after 3 min of recording resting-state brain activity.[59]

Immediately after the resting-state recording of each appointment, participants were asked to fill in the Amsterdam Resting-State Questionnaire (ARSQ[60]) and Positive and Negative Affect Schedule (PANAS[61,62]). After that, breath alcohol (BrAC) levels were assessed.

2.3 | EEG recording

EEG was recorded from 60 Ag–AgCl electrodes at standard equidistant scalp positions, with Fpz as reference electrode. Electrode impedances were kept below 5 kΩ. The data were recorded with a QuickAmp amplifier (Brain Products, Inc.) using a sampling rate of 500 Hz.

2.4 | EEG preprocessing

EEG preprocessing was performed using the BrainVision Analyzer (Version 2.0.1.327; Brain Products GmbH, Munich). In line with previous research,[20,56–58] we analyzed eyes-closed periods during which the influence of external visual stimulus processing and eye blinks is minimized.[63,64] First, we band-pass filtered the data between 2 and 20 Hz and re-derived data to average reference. We then replaced heavily corrupted channels by using a linear interpolation of adjacent electrodes, before correcting for eye movements using a semiautomatic independent component analysis (ICA)-based correction process. After removing these components, we screened channels a second time and interpolated additional channels, if necessary. We then performed automatic artifact rejection (maximum amplitude: +/−100 μV) and manually eliminated residual artifacts based on the ratings of two independent raters. Finally, we segmented all artifact-free data into 2-s epochs for further analyses (sober: mean 181.36 s;

standard deviation [SD] 15.19 s; range from 138 to 194 s; intoxicated: mean 178.18 s; SD 17.05 s; range from 112 to 193 s).

2.5 | EEG microstate analysis

Microstate analysis was conducted using the microstate plugin[65] for the Matlab toolbox EEGLAB.[66] Following standard procedures,[42,67,68] we submitted maps at the momentary peaks of the Global Field Power (GFP) of each individual (i.e., maximum voltage values at all electrodes at that time point that represent time points of optimal signal-to-noise ratio) to a modified spatial cluster analysis using the atomize–agglomerate hierarchical clustering (AAHC[69,70]) methods. Doing so, we could identify the four most dominant cluster maps that represent the four archetypal EEG resting networks in every single participant. To allow for comparison of the grand mean topographies of the four archetypal microstate maps between conditions, we performed the AAHC method for the intoxicated condition, for the sober condition, and across both conditions. We then submitted individual cluster maps to a second cluster analysis, yielding separate grand mean maps for participants under intoxicated, sober, and both conditions. Next, these grand mean maps were sorted according to the standard labeling (microstate A: left–right orientation; microstate B: right–left orientation; microstate C: anterior–posterior orientation; microstate D: fronto-central maximum; see ref.[16]). To obtain each individual's microstates characteristics for our statistical analyses, we sorted the individual maps that we had previously identified across conditions. This was done separately for both conditions (i.e., intoxicated and sober) on the basis of spatial correlations using the templates identified in the literature.[43] We then assigned the GFP peaks of individual EEG data to the best fitting individually identified cluster maps. We linearly interpolated this assignment to the time periods between the GFP peaks in order to obtain a continuous temporal stream of microstates occurring in each individual and condition. From this last step, we extracted four microstate characteristics in each condition: (1) the total percentage of time during which a given microstate is dominant, representing the total presence of the underlying network (*coverage*); (2) the mean microstate duration, representing an index of the temporal stability of the underlying resting network (*duration*); (3) the mean frequency of occurrence for each microstate independent of its individual duration, representing an index of relative usage of the underlying resting network (*occurrence*); and (4) the number of observed transitions normalized for the overall count of transitions (i.e., more transitions than expected from the number of occurrence of each microstate, given in Δ%) from one microstate to any other microstate, which reveal sequential activation of underlying neural networks (*transitions*).

2.6 | Statistical analysis

To compare topographies of microstate maps between conditions, we used a randomization test (topographic analysis of variance [TANOVA][71,72]). This test uses nonparametric randomization statistics to compare the topographical maps across conditions. For this, the global dissimilarity (i.e., configuration differences) between electrical field maps is computed. As this variable represents a single measure of difference, nonparametric randomization tests are used to assess statistical significance. For further details on this procedure, please refer to Murray et al.[73] First, we compared microstate coverages across conditions with a repeated-measures ANOVA using IBM SPSS Statistics software (IBM Corp., NY, USA) and the within-subject factors "condition" (sober vs. intoxicated) and "microstate class" (A vs. B vs. C vs. D). To further clarify the origin of significant differences in microstate coverage, we ran analogous analyses for the occurrence and duration of the four microstates (as coverage = duration × occurrence). Finally, we compared microstate transitions (transitions between microstate classes, e.g., for Class C: C → A, C → B, C → D, A → C, B → C, D → C) across conditions using a comparable ANOVA. In the case of significant interactions, we conducted separate post hoc ANOVAs comparing single microstate characteristics across conditions. Given that the order of both appointments was balanced across the sample and we had no hypothesis as to why resting-state activity (other than some cognitive tasks) should systematically differ between those orders, we did not include "order" as a between-subject factor in any of the analyses (in that context, please note that including "order" in exploratory add-on analyses not reported here did not change the reported ANOVA findings with respect to the reported significances). Finally, we analyzed changes in self-report measures across conditions. First, we analyzed changes in positive and negative mood (as measured by the PANAS) in separate repeated-measures ANOVAs using "condition" as a within-subject factor. We also repeated all ANOVAs with microstate characteristics as dependent variables, adding changes in positive and negative mood as covariates. Second, we analyzed changes in resting-state cognition (as measured by the ARSQ) using "condition" and "scale" (10 ARSQ scales) as within-subject factors in the ANOVA and conducted an exploratory post hoc ANOVA regarding changes in the ARSQ scale "somatic awareness." Third, we calculated Pearson correlations to investigate associations between intoxication-induced changes in "somatic awareness" and alcohol-associated changes in microstate characteristics.

In all analyses, Greenhouse–Geisser correction was applied whenever necessary. Post hoc tests did not undergo Bonferroni correction. All results are reported as mean value, SD, and range. In the main manuscript, we only report significant effects ($P < .050$, two-sided) involving the factor "condition," as our main question was how alcohol modulates microstate parameters (for significant effects involving the factor "microstate class"/"microstate class transition," please see supplementary material).

3 | RESULTS

3.1 | Sample characteristics

Before data analyses, we excluded $n = 2$ participants from the sample, as it was unclear which questionnaire had been filled out in which

condition for one participant, and another participant reported current psychological distress (we had set the BSI-18 sum score cutoff value to 13 [mean + 1 SD based on Spitzer et al.[74]] and he had a sum score of 20). Yet, please note that including these participants into the microstate analyses would not have changed the reported ANOVA findings with respect to the reported significances.

The remaining n = 22 participants were on average 23.9 years old (SD 3.0; range from 19 to 29 years), were 181.5 cm tall (SD 7.6; range from 164 to 194 cm), and weighed 74.5 kg (SD 13.9; range from 52.5 to 119 kg). Based on this, they received on average 276.5 mL vodka (40 vol %) (SD 30.6; range from 220 to 363 mL), which resulted in an average BrAC of 1.1‰ (SD 0.2; range from 0.5 to 1.5‰) at the time that the resting-state EEG was recorded and the questionnaires were filled out. n = 10 were sober on their first appointment and intoxicated on their second appointment, whereas the other n = 12 were intoxicated on their first appointment and sober on their second appointment. Lastly, the average AUDIT score in the sample was 7.5 (SD 3.0; range from 3 to 14).

3.2 | The four archetypal microstates

Applying the AAHC clustering algorithm, we could identify the four archetypal microstate topographies A–D (see Figure 1). In a first step, we used a TANOVA to test whether the topographies of these maps differed between the intoxicated and sober condition. We found no differences (microstate A: P = 0.095; microstate B: P = 0.055; microstate C: P = 0.944; microstate D: P = 0.349), which provides the rationale for comparing microstate characteristics between conditions based on the maps identified across both treatments. On average, these four maps explained 71.86% of the data's variance in the intoxicated condition (SD 4.22%; range from 61% to 78%) and 73.93% of the data's variance in the sober condition (SD 3.58%; range from 67% to 79%). Of note, the explained variance did not differ across conditions, as evidenced by a repeated-measures ANOVA ($F_{1,21}$ = 3.71; P = 0.068; η_p^2 = 0.150).

3.3 | Temporal dynamics of resting networks in the intoxicated versus sober condition

The repeated-measures ANOVA of the coverage measure revealed a significant interaction of condition and microstate class ($F_{3,63}$ = 5.06; P = 0.003; η_p^2 = 0.194). Separate post hoc ANOVAs for each microstate showed that compared with the sober condition, microstate B increased during intoxication ($F_{1,21}$ = 5.50; P = 0.029; η_p^2 = 0.207; sober: 22.78% ± 5.22, intoxicated: 25.01% ± 4.04) and microstate C decreased during intoxication ($F_{1,21}$ = 17.06; P < 0.001; η_p^2 = 0.448; sober: 29.78% ± 6.08, intoxicated: 26.00% ± 5.40; see Figure 2A). Microstates A and D showed no such condition differences (all $F \leq 1.60$, $P \geq 0.220$). In order to further clarify the origin of these differences in microstates B and C, we ran analogous analyses for the occurrence and duration of the four microstates, which determine a given microstate's coverage (coverage = duration × occurrence).

The repeated-measures ANOVA of the duration measure revealed a significant interaction of condition and microstate class ($F_{3,63}$ = 4.20; P = 0.009; η_p^2 = 0.167). Separate post hoc ANOVAs for each microstate showed that compared with the sober condition, the duration of microstate C decreased during intoxication ($F_{1,21}$ = 5.49; P = 0.029; η_p^2 = .207; sober: 46.2 ± 13.5 ms, intoxicated: 41.9 ± 11.9 ms; see Figure 2B). Microstates A, B, and D showed no such condition differences (all $Fs \leq 0.57$, $P \geq 0.459$). Given that not all of the included variables were normally distributed, as indicated by a Kolmogorov–Smirnov tests, we ran an additional nonparametric (Wilcoxon signed-rank) test, which confirmed the significantly decreased duration of microstate C during intoxication (Z = –2.22, P = 0.026).

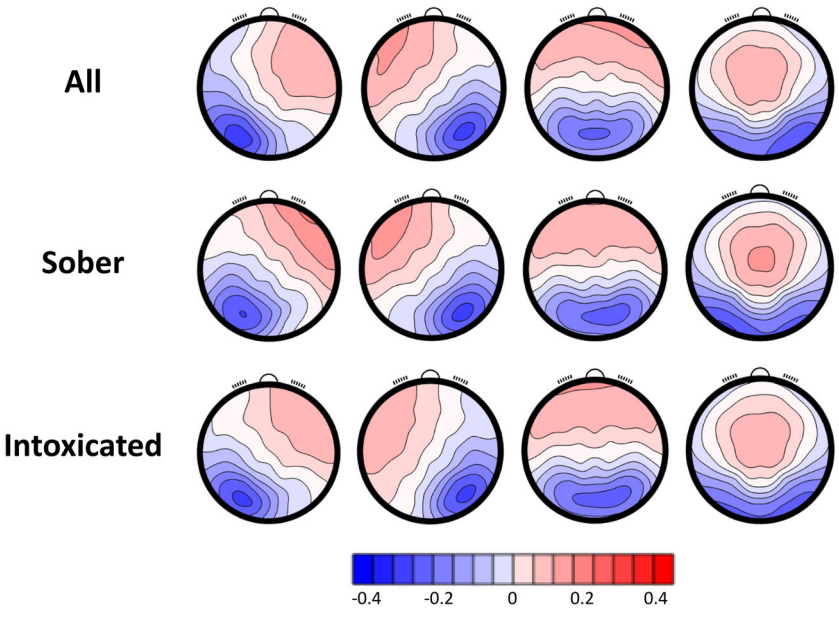

FIGURE 1 Microstate maps. Topographies of the four archetypal microstates A–D identified in the intoxicated condition (bottom row), in the sober condition (second row), and across both conditions (top row). Head seen from above, nose up, left ear left, red and blue are color labels chosen arbitrarily for areas of opposite polarity. As there were no topographical differences between the topographies of the intoxicated and sober condition (all $Ps \geq 0.055$), we used the topographies identified across both conditions for the fitting and identification of each individual's microstate characteristics

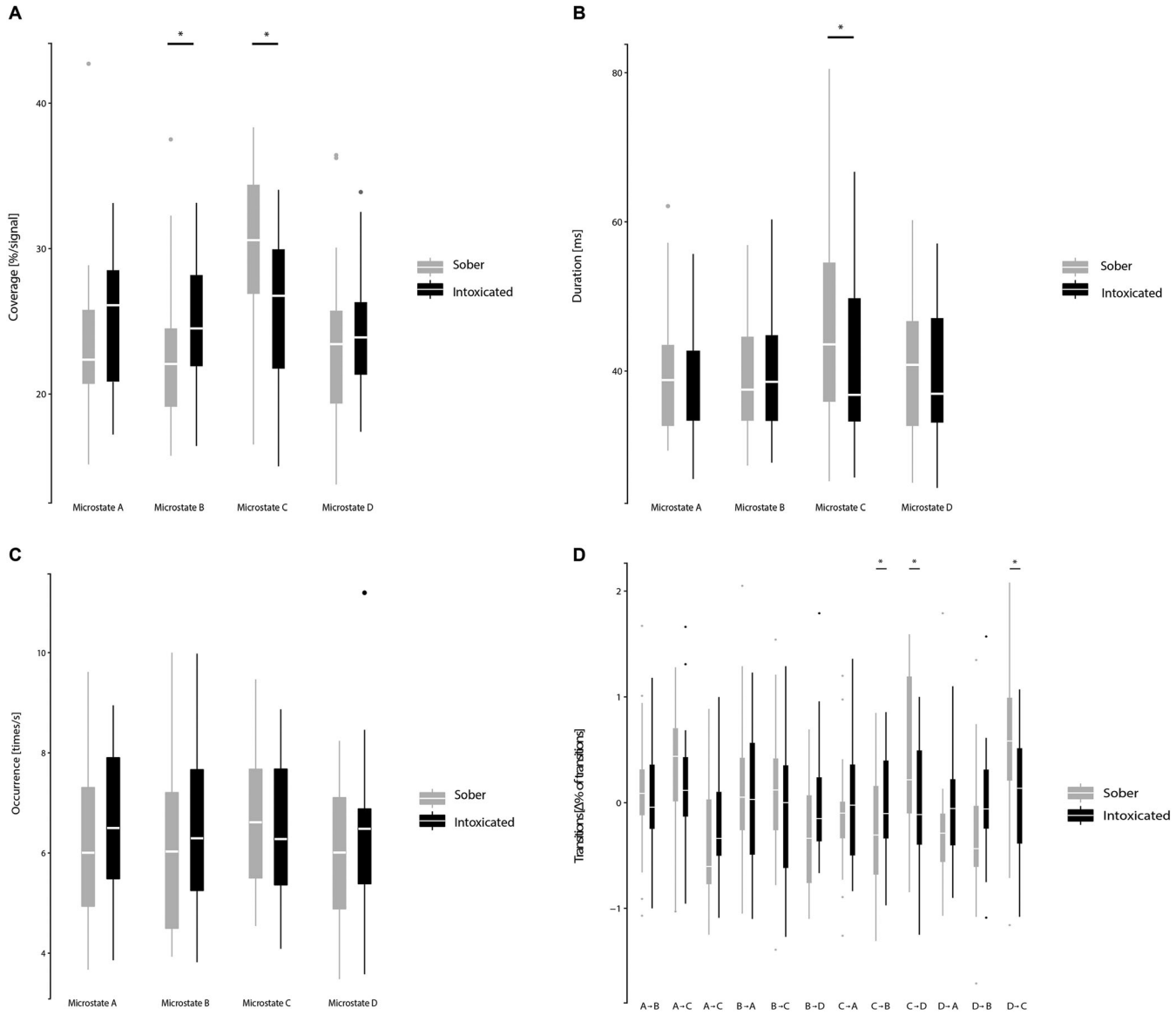

FIGURE 2 Boxplots (lower end of the whisker: 2.5% percentile; lower horizontal end of the box = 25% percentile; middle horizontal line = median; upper horizontal end of the box = 75% percentile; upper end of the whisker: 97.5% percentile) depicting differences in the temporal dynamics of microstates, that is, resting networks between conditions (sober = gray color; intoxicated = black color). Asterisks indicate significant differences between conditions ($P < 0.050$). (A) Differences in coverage in % of the signal. Microstate B covered significantly more time in the intoxicated condition ($P = 0.029$), whereas microstate C covered significantly less time in the intoxicated condition ($P < 0.001$) (interaction effect "microstate class X condition," $P = 0.007$). (B) Differences in mean duration in milliseconds (ms). The mean duration of microstate C was significantly shorter in the intoxicated condition ($P = 0.029$; interaction effect "microstate class X condition," $P = 0.014$). (C) Differences in mean occurrence in times per second. There were no significant differences between the conditions (all $P > 0.094$). (D) Differences in transitions in Δ% (observed − expected; see Section 2). There were significantly more transitions from microstate C to microstate B and significantly less bilateral transitions between microstates C and D in the intoxicated condition (microstate C → B: $P = 0.0430$, microstate C → D: $P = .0490$, microstate D → C: $P = 0.003$) (interaction effect of "microstate class transition X condition," $P = 0.004$)

The repeated-measures ANOVA of the occurrence measure revealed a significant interaction of condition and microstate class ($F_{3,63} = 3.57$; $P = 0.019$; $\eta_p^2 = 0.145$). However, none of the intoxication effects survived post hoc testing, as separate post hoc ANOVAs for each microstate showed no significant differences between the sober and intoxicated condition for any of the four microstates (all $Fs \leq 3.08$, $P \geq 0.094$; see Figure 2C). In sum, the reduced coverage of microstate C observed during intoxication seemed to be largely driven by a shorter duration, and not by a less frequent occurrence of this microstate. But even though the decrease in microstate C coverage co-occurred with an increased coverage of microstate B, no differences in duration or occurrence were observed between conditions with regard to this microstate.

Finally, we tested whether the above-reported intoxication-induced changes in the coverages of microstates B and C also led to changes in transitions from or toward these microstates. The

repeated-measures ANOVA of microstate transitions involving those from and toward microstates B and C (10 transitions in total) revealed a significant interaction of condition and microstate transition class ($F_{4.60,96.62}$ = 3.55; P = 0.007; η_p^2 = 0.145). Separate post hoc ANOVAs comparing the sober and intoxicated condition for each microstate transition showed that alcohol intoxication increased transitions from microstate C to microstate B ($F_{1,21}$ = 4.64; P = 0.043; η_p^2 = 0.181; sober: −0.32% ± 0.66%, intoxicated: −0.02% ± 0.48%) and decreased transitions from microstate C to microstate D ($F_{1,21}$ = 4.39; P = 0.049; η_p^2 = 0.173; sober: 0.41 ± 0.77%, intoxicated: 0.00% ± 0.61%) and from microstate D to microstate C ($F_{1,21}$ = 11.41; P = 0.003; η_p^2 = 0.352; sober: 0.58% ± 0.78%, intoxicated: 0.09% ± 0.60%; see Figure 2D). All other transitions showed no significant condition effect (all $Fs \leq 4.15$, $P \geq 0.054$). In sum, alcohol intoxication mainly changed transitions from and toward microstate C, decreasing bidirectional transitions with microstate D and increasing transitions from microstate C to microstate B.

3.4 | Self-report measures across the intoxicated and sober treatments

In a final set of analyses, we investigated potential associations between the intoxication-induced neurophysiological changes and psychological processes by testing for treatment differences in self-report measures on mood and resting-state cognition.

First, we analyzed changes in participants' mood (as measured by the PANAS). Repeated-measures ANOVAs showed that alcohol intoxication did neither change positive (sober: 3.55 ± 0.67; intoxicated: 3.40 ± 0.63; $F_{1,21}$ = 1.54; P = 0.229) nor negative affect (sober: 1.60 ± 0.40; intoxicated: 1.77 ± 0.60; $F_{1,21}$ = 3.35; P = 0.082). Furthermore, we added difference scores in both positive and negative affect (pre–post) as covariates in the above-reported analyses. We found that alcohol effects regarding the coverages of microstates B and C, transitions between microstates C and D, and transitions from microstate C to microstate B remained significant (all $Fs > 6.12$, all $P < 0.023$), whereas the effect regarding the duration of microstate C was no longer significant ($F_{1,19}$ = 2.75; P = 0.114). In other words, apart from the intoxication-induced decrease in microstate C's duration, the observed differences in neurophysiological processing across conditions remained significant when controlling for changes in participants' mood.

Finally, we analyzed changes in participants' resting-state cognition (as measured by the ARSQ). A repeated-measures ANOVA involving the within-subject factors "ASRQ scale" and "condition" revealed no significant interaction of scale and condition ($F_{5.18,108.93}$ = 1.77; P = 0.122; η_p^2 0 = 0.078). Given the known association of microstate C's coverage and experienced somatic awareness,[75] we nevertheless conducted an exploratory post hoc ANOVA for the ARSQ scale "somatic awareness." This ANOVA revealed a significant difference across conditions (no other scale showed a significant condition effect, all $P > 0.059$) with alcohol intoxication decreasing somatic awareness (2.72 ± 0.68) compared with the sober condition (3.24 ± 0.98; $F_{1,21}$ = 7.96, P = 0.010, η_p^2 = 0.275). Participants with larger decreases in somatic awareness under intoxication showed larger increases of transitions from microstate C to microstate B (r_{20} = −0.458, P = 0.032; associations of all other intoxication-induced neurophysiological and somatic awareness changes: $P > 0.064$). Thus, there was some evidence that intoxication-induced changes in microstate transitions were associated with changes in somatic awareness during the resting-state measurement.

4 | DISCUSSION

The present study investigates the effects of acute alcohol intoxication on the temporal dynamics of EEG resting networks to advance our understanding of which general changes in neuronal activity might underlie the widespread effects of alcohol onto cognitive processing and neuropsychological functioning. In short, we found that alcohol intoxication decreased the presence of the autonomic processing- and control-related microstate C in favor of the visual processing-related microstate B. Moreover, it decreased bidirectional transitions/communication between microstate C and the attention-related microstate D in favor of transitions/communication from microstate C to B. These neurophysiological effects remained significant when controlling for (nonsignificant) changes in self-reported participants' mood, suggesting that the alcohol-induced neurophysiological changes are most likely not due to changes in mood. Furthermore, participants reported decreased somatic awareness when intoxicated, which was associated with some of the observed neurophysiological changes. Together, these findings suggest that acute alcohol intoxication might hamper cognitive processing by promoting a bottom-up sensory processing-related network at the expense of networks associated with top-down control-related processing (i.e., by increasing the former and dampening the latter).

Our analyses revealed several alcohol-induced changes in the temporal characteristics of EEG resting networks. With respect to the presence of these networks, we had initially hypothesized that the bottom-up sensory processing-related microstates A and B should be increased, whereas the top-down control-related microstates C and D might be decreased. Indeed, and in line with previous reports of intensified processing in sensory processing-related networks during alcohol intoxication,[26,27] we found an increased presence of visual processing-related microstate B. We could, however, not find evidence of an increased presence of auditory processing-related microstate A. This dissociation is interesting against the background that visual processing of alcohol-related images can impair concurrent auditory processing in individuals with AUD, potentially because alcohol intoxication increases the dominance of the visual modality.[76] Importantly, the alcohol-induced promotion of vision-related sensory processing seems to have happened largely at the expense of autonomic information processing during microstate C, the coverage of which was significantly reduced during intoxication. Further analyses revealed that alcohol reduced the coverage of microstate C mostly by decreasing its duration, but much less so its occurrence, suggesting

that autonomic processing did not become rarer, but shorter during alcohol intoxication. This means that a neuronal network, which has repeatedly been associated with higher order processing of internal information and possibly with cognitive control,[22] is significantly less activated during alcohol intoxication. Given that resting-state activity seems to strongly determine how response-relevant information is processed,[23,24] the reduced duration of microstate C might help to explain why alcohol intoxication seems to make it harder to stably maintain cognitive control when needed (even though this phenomenon needs to be further investigated in order to underpin our interpretation). Moreover, we observed that alcohol increased transitions from microstate C to microstate B, but decreased bidirectional transitions between microstate C and another, attention-related microstate (microstate D). This change of microstate transitions is in line with our hypotheses stating that alcohol promotes bottom-up sensory processing at the expense of top-down control-related processing in microstates C and D. Although the reduction in microstate D failed to reach significance, the significantly reduced transitions from C to D suggest that there might potentially also be some, albeit smaller, reduction in the attention-related microstate D. Interestingly, this finding might also offer a different perspective on the so-called alcohol myopia,[77–79] which describes neuropsychological changes during intoxication that have commonly been interpreted as a narrowed attentional focus.[79,80] Yet, we found more prominent changes in the visual processing-related microstate B than in the attention-related microstate D (as one would expect in case of compromised attention). This suggests that phenomena like alcohol myopia might be largely driven by an increase in bottom-up sensory processing, rather than by changes in attention networks, and that those two networks are not necessarily strongly interdependent. In other words, behavioral phenomena commonly interpreted as indicators of altered attention might actually be caused by an imbalance in network activation, which favors bottom-up over top-down information processing.

Together, the reported alcohol-induced changes on the temporal dynamics of microstates B and C suggest that in the resting state, acute alcohol intoxication prioritizes the activation of networks associated with bottom-up sensory processing over the activation of networks associated with the processing of internal stimuli and cognitive control. To obtain further evidence corroborating this conjecture, we analyzed alcohol effects on self-reported resting-state cognition as measured by the ARSQ.[60] As microstate C has previously been shown to be associated with experienced somatic awareness,[75] we further explored alcohol effects on this scale despite the nonsignificant effects in the overall analyses of resting-state cognition. In line with our hypothesis that alcohol decreases self-reported somatic awareness, we found that somatic awareness was decreased during intoxication and that this decrease was positively correlated with alcohol-induced increases of transitions from microstate C to microstate B. Thus, intoxication-induced changes in microstate transitions might potentially be associated with changes in participants' somatic awareness during the resting-state measurement.

Despite the use of a counterbalanced within-subject design including acute high-dose alcohol intoxication in combination a spatiotemporal EEG analysis approach, the present study also has limitations. While the within-subject design increases statistical power and lowers the risk that confounding variables might drive treatment differences, the counterbalancing of appointment order and associated effects might have introduced noise into the data (which should however be negligible, as we analyzed resting-state data, not task performance data). Also, it was somewhat difficult to generate a priori hypotheses on all of the analyzed networks' temporal characteristics (e.g., transitions) due to the lack of comparable studies in the field. Future studies will be required to reproduce our study's findings and test whether the reported neurophysiological changes apply to female participants as well. Finally, though the analysis of resting EEG microstates holds the potential to reveals unique insights into the fundamental building blocks of human cognition, more research is needed to underpin the assumed link of these states to specific underlying networks and neuropsychological processes.[16]

In summary, the present study investigated general (i.e., task-independent) alcohol intoxication effects on the temporal dynamics of large-scale brain circuits using a spatiotemporal EEG analysis approach. This revealed that alcohol intoxication alters fundamental neuropsychological processing even when there is no task at hand. Overall, we found that alcohol intoxication reduced the presence of an autonomic processing- and potentially control-related network (= microstate C) as well as that network's bilateral transitions toward and from an attention-related one (= microstate D), ultimately benefiting both the presence of and the transitions toward a visual processing-related network (= microstate B). These findings suggest that alcohol prioritizes networks associated with the bottom-up processing of sensory (visual) stimuli over those associated with top-down processing of internal stimuli during the resting state, thereby potentially contributing to the cognitive (control and attention) deficits commonly observed during alcohol intoxication. To further corroborate this assumption, future follow-up research could analyze (and relate) the effects of acute alcohol intoxication on both task-independent and task-dependent neural processing.[58,81] Doing so would further deepen our understanding of the neuropsychological mechanisms underlying the cognitive and behavioral effects of alcohol.

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AUTHOR CONTRIBUTIONS

CB and AS designed the study and wrote the protocol. AS collected the data. AS and BS undertook the data analysis and wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

DATA AVAILABILITY STATEMENT

All study data are available upon request.

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REFERENCES

1. European Social Survey. ESS round 8. Data file edition 2.1. NSD - Norwegian Centre for Research Data, Norway – data archive and distributor of ESS data for ESS ERIC. 2016. https://doi.org/10.21338/NSD-ESS8-2016
2. World Health Organization. Global status report on alcohol and health 2018. 2018.
3. Heinz A, Kiefer F, Smolka MN, et al. Addiction research consortium: losing and regaining control over drug intake (ReCoDe)-from trajectories to mechanisms and interventions. *Addict Biol*. 2020;25(2):e12866. https://doi.org/10.1111/adb.12866
4. Courtney KE, Polich J. Binge drinking in young adults: data, definitions, and determinants. *Psychol Bull*. 2009;135(1):142-156. https://doi.org/10.1037/a0014414
5. Kuntsche E, Kuntsche S, Thrul J, Gmel G. Binge drinking: health impact, prevalence, correlates and interventions. *Psychol Health*. 2017;32(8):976-1017. https://doi.org/10.1080/08870446.2017.1325889
6. Zoethout RWM, Delgado WL, Ippel AE, Dahan A, van Gerven JMA. Functional biomarkers for the acute effects of alcohol on the central nervous system in healthy volunteers. *Br J Clin Pharmacol*. 2011;71(3):331-350. https://doi.org/10.1111/j.1365-2125.2010.03846.x
7. Bjork JM, Gilman JM. The effects of acute alcohol administration on the human brain: insights from neuroimaging. *Neuropharmacology*. 2014;84:101-110. https://doi.org/10.1016/j.neuropharm.2013.07.039
8. Field M, Wiers RW, Christiansen P, Fillmore MT, Verster JC. Acute alcohol effects on inhibitory control and implicit cognition: implications for loss of control over drinking. *Alcohol Clin Exp Res*. 2010;34(8):1346-1352. https://doi.org/10.1111/j.1530-0277.2010.01218.x
9. Field M, Schoenmakers T, Wiers RW. Cognitive processes in alcohol binges: a review and research agenda. *Curr Drug Abuse Rev*. 2008;1(3):263-279.
10. Stock A-K. Barking up the wrong tree: why and how we may need to revise alcohol addiction therapy. *Front Psychol*. 2017;8:884. https://doi.org/10.3389/fpsyg.2017.00884
11. Trantham-Davidson H, Chandler LJ. Alcohol-induced alterations in dopamine modulation of prefrontal activity. *Alcohol*. 2015;49(8):773-779. https://doi.org/10.1016/j.alcohol.2015.09.001
12. Crabbe JC, Harris RA, Koob GF. Preclinical studies of alcohol binge drinking. *Ann N Y Acad Sci*. 2011;1216(1):24-40. https://doi.org/10.1111/j.1749-6632.2010.05895.x
13. Most D, Ferguson L, Harris RA. Molecular basis of alcoholism. *Handb Clin Neurol*. 2014;125:89-111. https://doi.org/10.1016/B978-0-444-62619-6.00006-9
14. Siggins GR, Roberto M, Nie Z. The tipsy terminal: presynaptic effects of ethanol. *Pharmacol Ther*. 2005;107(1):80-98. https://doi.org/10.1016/j.pharmthera.2005.01.006
15. Chastain G. Alcohol, neurotransmitter systems, and behavior. *J Gen Psychol*. 2006;133(4):329-335. https://doi.org/10.3200/GENP.133.4.329-335
16. Michel CM, Koenig T. EEG microstates as a tool for studying the temporal dynamics of whole-brain neuronal networks: a review. *Neuroimage*. 2018;180(Part B):577-593. https://doi.org/10.1016/j.neuroimage.2017.11.062
17. Khanna A, Pascual-Leone A, Michel CM, Farzan F. Microstates in resting-state EEG: current status and future directions. *Neurosci Biobehav Rev*. 2015;49:105-113. https://doi.org/10.1016/j.neubiorev.2014.12.010
18. Michel CM, Koenig T, Brandeis D, Gianotti LR, Wackermann J (Eds). *Electrical Neuroimaging*. Cambridge, UK: Cambridge University Press; 2009.
19. Schiller B, Kleinert T, Teige-Mocigemba S, Klauer KC, Heinrichs M. Temporal dynamics of resting EEG networks are associated with prosociality. *Sci Rep*. 2020;10(1):13066. https://doi.org/10.1038/s41598-020-69999-5
20. Schiller B, Koenig T, Heinrichs M. Oxytocin modulates the temporal dynamics of resting EEG networks. *Sci Rep*. 2019;9(1):13418. https://doi.org/10.1038/s41598-019-49636-6
21. Abreu R, Jorge J, Leal A, Koenig T, Figueiredo P. EEG microstates predict concurrent fMRI dynamic functional connectivity states. *Brain Topogr*. 2021;34(1):41-55. https://doi.org/10.1007/s10548-020-00805-1
22. Seitzman BA, Abell M, Bartley SC, Erickson MA, Bolbecker AR, Hetrick WP. Cognitive manipulation of brain electric microstates. *Neuroimage*. 2017;146:533-543. https://doi.org/10.1016/j.neuroimage.2016.10.002
23. Britz J, Michel CM. State-dependent visual processing. *Front Psychol*. 2011;2:370. https://doi.org/10.3389/fpsyg.2011.00370
24. Muthukrishnan S-P, Ahuja N, Mehta N, Sharma R. Functional brain microstate predicts the outcome in a visuospatial working memory task. *Behav Brain Res*. 2016;314:134-142. https://doi.org/10.1016/j.bbr.2016.08.020
25. Abrahao KP, Salinas AG, Lovinger DM. Alcohol and the brain: neuronal molecular targets, synapses, and circuits. *Neuron*. 2017;96(6):1223-1238. https://doi.org/10.1016/j.neuron.2017.10.032
26. Esposito F, Pignataro G, Di Renzo G, et al. Alcohol increases spontaneous BOLD signal fluctuations in the visual network. *Neuroimage*. 2010;53(2):534-543. https://doi.org/10.1016/j.neuroimage.2010.06.061
27. Khalili-Mahani N, Zoethout RMW, Beckmann CF, et al. Effects of morphine and alcohol on functional brain connectivity during "resting state": A placebo-controlled crossover study in healthy young men. *Hum Brain Mapp*. 2012;33(5):1003-1018. https://doi.org/10.1002/hbm.21265
28. Volkow ND, Ma Y, Zhu W, et al. Moderate doses of alcohol disrupt the functional organization of the human brain. *Psychiatr Res Neuroimag*. 2008;162(3):205-213. https://doi.org/10.1016/j.pscychresns.2007.04.010
29. Schreckenberger M, Amberg R, Scheurich A, et al. Acute alcohol effects on neuronal and attentional processing: striatal reward system and inhibitory sensory interactions under acute ethanol challenge. *Neuropsychopharmacology*. 2004;29(8):1527-1537. https://doi.org/10.1038/sj.npp.1300453
30. Volkow ND, Mullani N, Gould L, et al. Effects of acute alcohol intoxication on cerebral blood flow measured with PET. *Psychiatry Res*. 1988;24(2):201-209. https://doi.org/10.1016/0165-1781(88)90063-7
31. Ingvar M, Ghatan PH, Wirsén-Meurling A, et al. Alcohol activates the cerebral reward system in man. *J Stud Alcohol*. 1998;59(3):258-269. https://doi.org/10.15288/jsa.1998.59.258
32. Tolentino NJ, Wierenga CE, Hall S, et al. Alcohol effects on cerebral blood flow in subjects with low and high responses to alcohol. *Alcohol Clin Exp Res*. 2011;35(6):1034-1040. https://doi.org/10.1111/j.1530-0277.2011.01435.x
33. Kawano Y. Physio-pathological effects of alcohol on the cardiovascular system: its role in hypertension and cardiovascular disease. *Hypertens Res*. 2010;33(3):181-191. https://doi.org/10.1038/hr.2009.226
34. Davis PA, Gibbs FA, Davis H, Jetter WW, Trowbridge LS. The effects of alcohol upon the electroencephalogram (brain waves). *Q J Stud Alcohol*. 1941;1(4):626-637.

35. Engel GL, Rosenbaum M. Delirium: iii. Electroencephalographic changes associated with acute alcoholic intoxication. *Arch Neurol Psychiatry*. 1945;53(1):44-50. https://doi.org/10.1001/archneurpsyc.1945.02300010054004
36. Rosen BQ, O'Hara R, Kovacevic S, Schulman A, Padovan N, Marinkovic K. Oscillatory spatial profile of alcohol's effects on the resting state: anatomically-constrained MEG. *Alcohol*. 2014;48(2):89-97. https://doi.org/10.1016/j.alcohol.2013.12.004
37. Gevins A, Smith ME, McEvoy LK. Tracking the cognitive pharmacodynamics of psychoactive substances with combinations of behavioral and neurophysiological measures. *Neuropsychopharmacology*. 2002;26(1):27-39. https://doi.org/10.1016/S0893-133X(01)00300-1
38. Stenberg G, Sano M, Rosén I, Ingvar DH. EEG topography of acute ethanol effects in resting and activated normals. *J Stud Alcohol*. 1994;55(6):645-656. https://doi.org/10.15288/jsa.1994.55.645
39. Lei X, Wang YL, Yuan H, Mantini D. Neuronal oscillations and functional interactions between resting state networks: effects of alcohol intoxication. *Hum Brain Mapp*. 2014;35(7):3517-3528. https://doi.org/10.1002/Hbm.22418
40. Lithari C, Klados MA, Pappas C, et al. Alcohol affects the brain's resting-state network in social drinkers. *PLoS One*. 2012;7(10):e48641. https://doi.org/10.1371/journal.pone.0048641
41. Stock A-K, Pertermann M, Mückschel M, Beste C. High-dose ethanol intoxication decreases 1/f neural noise or scale-free neural activity in the resting state. *Addict Biol*. 2019;25(6):e12818. https://doi.org/10.1111/adb.12818
42. Lehmann D. Principles of spatial analysis. In: Gevins AS, Remond A, eds. *Handbook of Electroencephalography and Clinical Neurophysiology. Methods of Analysis of Brain Electrical and Magnetic Signals*. Amsterdam: Elsevier; 1987:309-354.
43. Koenig T, Prichep L, Lehmann D, et al. Millisecond by millisecond, year by year: normative EEG microstates and developmental stages. *Neuroimage*. 2002;16(1):41-48. https://doi.org/10.1006/nimg.2002.1070
44. Kinoshita T, Strik WK, Michel CM, Yagyu T, Saito M, Lehmann D. Microstate segmentation of spontaneous multichannel EEG map series under diazepam and sulpiride. *Pharmacopsychiatry*. 2007;28(2):51-55. https://doi.org/10.1055/s-2007-979588
45. Serrano JI, Del Castillo MD, Cortés V, et al. EEG microstates change in response to increase in dopaminergic stimulation in typical Parkinson's disease patients. *Front Neurosci*. 2018;12:714-714. https://doi.org/10.3389/fnins.2018.00714
46. Yoshimura M, Koenig T, Irisawa S, et al. A pharmaco-EEG study on antipsychotic drugs in healthy volunteers. *Psychopharmacology (Berl)*. 2007;191(4):995-1004. https://doi.org/10.1007/s00213-007-0737-8
47. Abrams K, Cieslowski K, Johnson S, et al. The effects of alcohol on heartbeat perception: implications for anxiety. *Addict Behav*. 2018;79:151-158. https://doi.org/10.1016/j.addbeh.2017.12.023
48. Stock A-K, Mückschel M, Beste C. Reversal of alcohol-induced effects on response control due to changes in proprioceptive information processing. *Addict Biol*. 2017;22(1):246-256. https://doi.org/10.1111/adb.12296
49. Ribordy Lambert F, Wicht CA, Mouthon M, Spierer L. Acute alcohol intoxication and expectations reshape the spatiotemporal functional architecture of executive control. *Neuroimage*. 2020;215:116811. https://doi.org/10.1016/j.neuroimage.2020.116811
50. Bailey K, Bartholow BD, Saults JS, Lust SA. Give me just a little more time: effects of alcohol on the failure and recovery of cognitive control. *J Abnorm Psychol*. 2014;123(1):152-167. https://doi.org/10.1037/a0035662
51. Faul F, Erdfelder E, Lang A-G, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*. 2007;39(2):175-191. https://doi.org/10.3758/BF03193146
52. Faul F, Erdfelder E, Buchner A, Lang A-G. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behav Res Methods*. 2009;41(4):1149-1160. https://doi.org/10.3758/BRM.41.4.1149
53. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. The alcohol use disorders identification test guidelines for use in primary care. Second Edition. 2001. Accessed May 1, 2013. http://whqlibdoc.who.int/hq/2001/who_msd_msb_01.6a.pdf
54. Chmielewski WX, Zink N, Chmielewski KY, Beste C, Stock A-K. How high-dose alcohol intoxication affects the interplay of automatic and controlled processes. *Addict Biol*. 2018;25(1):e12700. https://doi.org/10.1111/adb.12700
55. Zink N, Zhang R, Chmielewski WX, Beste C, Stock A-K. Detrimental effects of a high-dose alcohol intoxication on sequential cognitive flexibility are attenuated by practice. *Prog Neuropsychopharmacol Biol Psychiatry*. 2019;89:97-108. https://doi.org/10.1016/j.pnpbp.2018.08.034
56. Schiller B, Gianotti LRR, Baumgartner T, Knoch D. Theta resting EEG in the right TPJ is associated with individual differences in implicit intergroup bias. *Soc Cogn Affect Neurosci*. 2019;14(3):281-289.
57. Baumgartner T, Langenbach BP, Gianotti LRR, Müri RM, Knoch D. Frequency of everyday pro-environmental behaviour is explained by baseline activation in lateral prefrontal cortex. *Sci Rep*. 2019;9(1):7. https://doi.org/10.1038/s41598-018-36956-2
58. Schiller B, Gianotti LR, Nash K, Knoch D. Individual differences in inhibitory control--relationship between baseline activation in lateral PFC and an electrophysiological index of response inhibition. *Cereb Cortex*. 2014;24(9):2430-2435. https://doi.org/10.1093/cercor/bht095
59. Tagliazucchi E, Laufs H. Decoding wakefulness levels from typical fMRI resting-state data reveals reliable drifts between wakefulness and sleep. *Neuron*. 2014;82(3):695-708. https://doi.org/10.1016/j.neuron.2014.03.020
60. Diaz B, Van Der Sluis S, Benjamins J, et al. The ARSQ 2.0 reveals age and personality effects on mind-wandering experiences. *Front Psychol*. 2014;5:271. https://doi.org/10.3389/fpsyg.2014.00271
61. Breyer B, Bluemke M. Deutsche version der positive and negative affect schedule PANAS (GESIS panel). *Zusammenstelleung Sozialwissenschaftlicher Items Udn Skalen*. doi: https://doi.org/10.6102/zis242
62. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect - the PANAS scales. *J Pers Soc Psychol*. 1988;54(6):1063-1070.
63. Barry RJ, Clarke AR, Johnstone SJ, Magee CA, Rushby JA. EEG differences between eyes-closed and eyes-open resting conditions. *Clin Neurophysiol*. 2007;118(12):2765-2773. https://doi.org/10.1016/j.clinph.2007.07.028
64. van Diessen E, Numan T, van Dellen E, et al. Opportunities and methodological challenges in EEG and MEG resting state functional brain network research. *Clin Neurophysiol*. 2015;126(8):1468-1481. https://doi.org/10.1016/j.clinph.2014.11.018
65. Koenig T. EEGLAB plugin for microstates. 2017. http://www.thomaskoenig.ch/index.php/software/microstates-in-eeglab
66. Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods*. 2004;134(1):9-21. https://doi.org/10.1016/j.jneumeth.2003.10.009
67. Strik WK, Lehmann D. Data-determined window size and space-oriented segmentation of spontaneous EEG map series. *Electroencephalogr Clin Neurophysiol*. 1993;87(4):169-174. https://doi.org/10.1016/0013-4694(93)90016-O
68. Wackermann J, Lehmann D, Michel CM, Strik WK. Adaptive segmentation of spontaneous EEG map series into spatially defined microstates. *Int J Psychophysiol*. 1993;14(3):269-283. https://doi.org/10.1016/0167-8760(93)90041-M

69. Tibshirani R, Walther G. Cluster validation by prediction strength. *J Comput Graph Stat*. 2005;14(3):511-528. https://doi.org/10.1198/106186005X59243
70. Britz J, Landis T, Michel CM. Right parietal brain activity precedes perceptual alternation of bistable stimuli. *Cereb Cortex*. 2009;19(1):55-65. https://doi.org/10.1093/cercor/bhn056
71. Koenig T, Kottlow M, Stein M, Melie-Garcia L. Ragu: a free tool for the analysis of EEG and MEG event-related scalp field data using global randomization statistics. *Comput Intell Neurosci*. 2011;2011:938925. https://doi.org/10.1155/2011/938925
72. Koenig T, Stein M, Grieder M, Kottlow M. A tutorial on data-driven methods for statistically assessing ERP topographies. *Brain Topogr*. 2014;27(1):72-83. https://doi.org/10.1007/s10548-013-0310-1
73. Murray MM, Brunet D, Michel CM. Topographic ERP analyses: a step-by-step tutorial review. *Brain Topogr*. 2008;20(4):249-264. https://doi.org/10.1007/s10548-008-0054-5
74. Spitzer C, Hammer S, Löwe B, et al. Die Kurzform des Brief Symptom Inventory (BSI -18): erste Befunde zu den psychometrischen Kennwerten der deutschen Version. *Fortschr Neurol Psychiatr*. 2011;79(9):517-523. https://doi.org/10.1055/s-0031-1281602
75. Pipinis E, Melynyte S, Koenig T, et al. Association between resting-state microstates and ratings on the Amsterdam resting-state questionnaire. *Brain Topogr*. 2017;30(2):245-248. https://doi.org/10.1007/s10548-016-0522-2
76. Monem RG, Fillmore MT. Alcohol-related visual cues impede the ability to process auditory information: Seeing but not hearing. *Psychol Addict Behav*. 2016;30(1):12-17. https://doi.org/10.1037/adb0000140
77. Steele CM, Josephs RA. Alcohol myopia. Its prized and dangerous effects. *Am Psychol*. 1990;45(8):921-933.
78. Lac A, Berger DE. Development and validation of the alcohol myopia scale. *Psychol Assess*. 2013;25(3):738-747. https://doi.org/10.1037/a0032535
79. Harvey AJ, Bayless SJ, Hyams G. Alcohol increases inattentional blindness when cognitive resources are not consumed by ongoing task demands. *Psychopharmacology (Berl)*. 2018;235(1):309-315. https://doi.org/10.1007/s00213-017-4772-9
80. Bayless SJ, Harvey AJ. Testing alcohol myopia theory: examining the effects of alcohol intoxication on simultaneous central and peripheral attention. *Perception*. 2017;46(1):90-99. https://doi.org/10.1177/0301006616672221
81. Morishima Y, Schunk D, Bruhin A, Ruff CC, Fehr E. Linking brain structure and activation in temporoparietal junction to explain the neurobiology of human altruism. *Neuron*. 2012;75(1):73-79. https://doi.org/10.1016/j.neuron.2012.05.021

SUPPORTING INFORMATION

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