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The extrastriate symmetry response is robust to alcohol intoxication

Elena Karakashevska[1] | **Yiovanna Derpsch**[2] | **Andrew Jones**[3] | **Alexis D. J. Makin**[1]

[1]Department of Psychology, Population Health Institute, University of Liverpool, Liverpool, UK

[2]Faculty of Social Sciences, School of Psychology, University of East Anglia, Norwich, UK

[3]Faculty of Health, School of Psychology, Liverpool John Moores, Liverpool, UK

Correspondence
Elena Karakashevska, Population Health Institute, Department of Psychology, University of Liverpool, Liverpool, UK.
Email: e.karakashevska@liverpool.ac.uk

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Abstract

Visual symmetry activates a network of regions in the extrastriate cortex and generates an event-related potential (ERP) called the sustained posterior negativity (SPN). Previous work has found that the SPN is robust to experimental manipulations of task, spatial attention, and memory load. In the current study, we investigated whether the SPN is also robust to alcohol-induced changes in mental state. A pilot experiment ($N=13$) found that alcohol unexpectedly increased SPN amplitude. We followed this unexpected result with two new experiments on separate groups, using an alcohol challenge paradigm. One group completed an Oddball discrimination task ($N=26$). Another group completed a Regularity discrimination task ($N=26$). In both groups, participants consumed a medium dose of alcohol (0.65 g/kg body weight) and a placebo drink, in separate sessions. Alcohol reduced SPN amplitude in the Oddball task (contrary to the pilot results) but had no effect on SPN amplitude in the Regularity task. In contrast, the N1 wave was consistently dampened by alcohol in all experiments. Exploratory analysis indicated that the inconsistent effect of alcohol on SPN amplitude may be partly explained by individual differences in alcohol use. Alcohol reduced the SPN in light drinkers and increased it in heavier drinkers. Despite remaining questions, the results highlight the automaticity of symmetry processing. Symmetry still produces a large SPN response, even when participants are intoxicated, and even when symmetry is not task relevant.

KEYWORDS

alcohol/alcoholism, EEG, SPN, symmetry, visual processes

1 | INTRODUCTION

Visual symmetry contributes to perceptual organization and object formation (Bertamini et al., 2018; Makin et al., 2023). Psychophysical research demonstrates that the detection of symmetry is fast and noise tolerant. Symmetry can be discriminated from random within 25 ms (Locher & Wagemans, 1993) and when it is presented in the visual periphery (Barlow & Reeves, 1979; Rampone et al., 2016). The neural basis of symmetry perception has been researched in the last two decades. Converging evidence from fMRI and TMS has found that

symmetry is coded in extrastriate visual areas, with the strongest response in the shape-sensitive Lateral Occipital Complex. V1 and V2 have smaller receptive fields and do not respond to symmetry (Audurier et al., 2022; Bona et al., 2015; Keefe et al., 2018; Kohler et al., 2016; Sasaki et al., 2005; Tyler et al., 2005; Van Meel et al., 2019).

The extrastriate symmetry response can also be measured with electroencephalography (EEG). Both symmetrical and random patterns produce Event-Related Potential (ERP) at posterior electrodes. After the P1 and N1 components of the visual evoked potential, amplitude is lower for symmetrical patterns (Höfel & Jacobsen, 2007; Jacobsen & Höfel, 2003; Norcia et al., 2002). This symmetry-random difference wave is called the Sustained Posterior Negativity (SPN, Makin et al., 2012). SPN amplitude scales with the salience of different visual regularities (Figure 1, Makin et al., 2016, 2020a; Palumbo et al., 2015). The SPN is generated whatever the participant's task, but amplitude is enhanced when symmetry is task relevant (Figure 2).

Majority of the neuroimaging research conducted to measure the brain response to symmetrical patterns has been done under laboratory and isolated conditions. However, more recently researchers have begun to explore the SPN under generic conditions by using real-life objects and patterns disrupted by perspective (Makin, Rampone, Karakashevska, & Bertamini, 2020b). Derpsch et al. (2019) found that while the brain response to symmetry can be enhanced when symmetry is presented in attended regions of the screen, it is still robust when symmetry is presented in unattended regions. In another study, Derpsch et al. (2021) found that the SPN is not diminished by a concurrent visual working memory task. In the current work, we extended this research program by investigating whether the SPN is sensitive to pharmacologically induced changes in mental state. Alcohol inhibits the central nervous system by slowing neural processing and interhemispheric transmission and thus impairing cognition (Khan & Timney, 2007). Regarding visual cognition the effect of alcohol is varied, for example, alcohol can alter eye movement (Marinkovic et al., 2013), impact retinal image quality and night vision performance (Castro et al., 2014), as well as

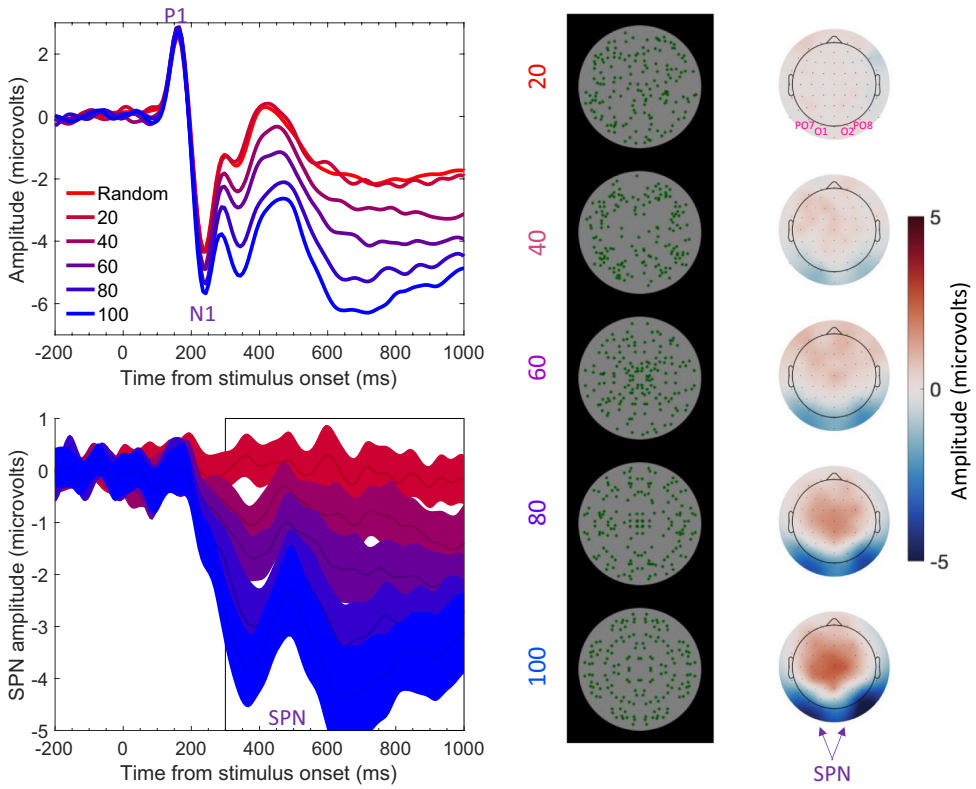

FIGURE 1 Results of Makin, Rampone, Morris, and Bertamini (2020a). The grand average ERPs are shown in the upper left panel, and difference waves (symmetry-random) are shown in the lower left panel. A large SPN is a difference wave that falls a long way below zero. Topographic difference maps are shown on the right, aligned with the representative stimuli. The difference maps depict a head from above, and the SPN appears as blue at the back. Purple labels indicate electrodes used for ERP waves [PO7, O1, O2, and PO8]. SPN amplitude increases (that is, becomes more negative) with the proportion of symmetry in the image. In this example, the SPN increased from approximately 0 to −3.5 microvolts as symmetry increased from 20% to 100%. Figure from Makin et al. (2022).

deteriorate binocular vision (Hogan & Linfield, 1983). Alcohol alters several known ERP components, including the MNN, P3b, ERN, N180, N2b, N2c, and N450 (for meta-analysis, see Fairbairn et al., 2021). However, the effect of alcohol on SPN amplitude had not been studied before. If alcohol affects SPN amplitude, it signals that alcohol disrupts perceptual organization.

1.1 | Pilot study

A pilot study ($N=13$ social drinkers) found surprising results (Figure 3). In this pilot study, participants discriminated normal trials (e.g., with black elements) from differently colored oddballs (e.g., with green elements). Participants completed the task twice, in separate sessions on separate days (a standard procedure in alcohol research, see Halsall et al. (2022) for meta-analysis). In one session, they drank 0.65 g/kg alcohol before EEG recording; in the other they drank an equivalent placebo drink before EEG recording. Despite our pre-registered predictions (https://aspredicted.org/xp4dd.pdf), alcohol enhanced the SPN, particularly during later 400–1000 ms time window ($t(12)=-4.188$, $p<.001$, $d_z=1.161$). This suggests alcohol may disinhibit the visual cortex, making it more sensitive to task-irrelevant symmetry. More specifically, alcohol may make the task-irrelevant symmetry response more persistent, as if the system carries on coding irrelevant information for longer than necessary. However, this unexpected result required replication. The pilot study also found that alcohol had no effect on the P1 peak ($t(12)=-0.112$, $p=.913$, $d_z=0.031$) and reduced N1 dip ($t(12)=4.460$, $p<.001$, $d_z=1.237$).

1.2 | Current study

The unexpected results of the pilot study raised interesting possibilities. It could be that sensory cortices are disinhibited by alcohol, and this could explain some aesthetic effects. One possibility is that altered sensitivity to visual symmetry contributes to the "beer goggles effect," whereby drinking makes other people appear more attractive (Halsey et al., 2010; Harvey et al., 2023). It is also well known that symmetrical faces and bodies are attractive (Grammer et al., 2003; Little et al., 2011; Rhodes et al., 1998) and alcohol might increase attraction partly by increasing the salience of phenotypic symmetry.

Our first aim was to replicate the pilot study in a new experiment. We refer to this new experiment as

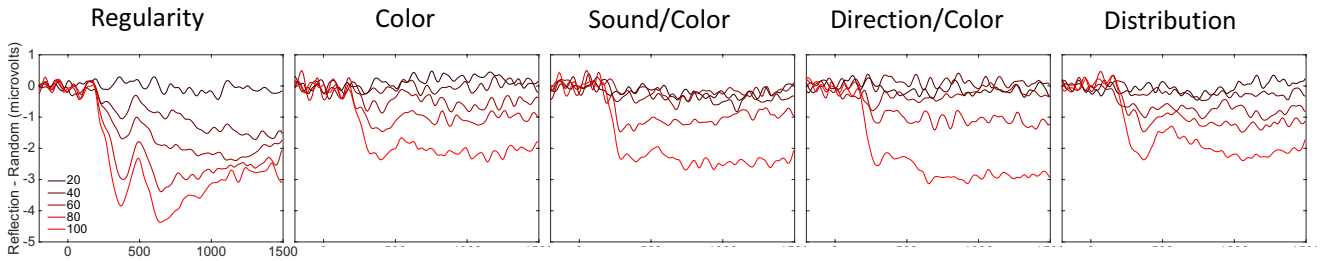

FIGURE 2 In Makin et al., (2020a), the parametric SPN response was evident in five tasks (from 5 different groups of 26 participants) but selectively enhanced in the Regularity task (left). Figure from Makin et al. (2023).

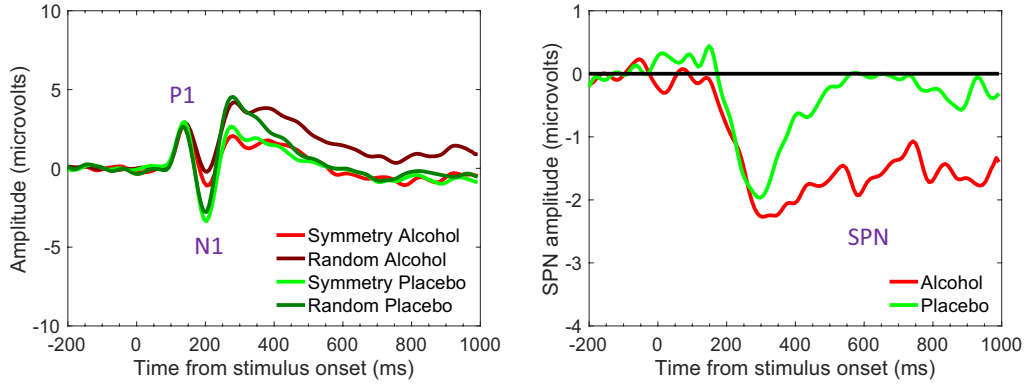

FIGURE 3 Pilot experiment ERP waves (left) and SPN waves (right). Alcohol had little effect on P1, reduced N1, and surprisingly, enhanced the SPN.

the Oddball task. We conservatively assumed that the true effect size of the late alcohol-induced SPN difference is half that found in the pilot ($d_z = 0.58$) and collected a larger sample accordingly ($N = 26$, power = 0.8, alpha = 0.05, two-tailed). Given the pilot study results, we predicted that alcohol would enhance the SPN in the later 400–1000 ms window (https://aspredicted.org/hj9eh.pdf). However, the late SPN was slightly reduced, rather than enhanced, in the alcohol session (the opposite of the pilot results).

Following those results, we completed another new experiment, with another group of 26 participants. These participants discriminated symmetry from random trials and ignored color. We refer to this as the Regularity task. This is an important comparison. It might be that automatic symmetry responses are more vulnerable to alcohol-induced changes. Here, we predicted alcohol would have no effect on the SPN. These predictions were confirmed (https://aspredicted.org/yn3vq.pdf). Results of the Oddball and Regularity tasks are presented together and as separate groups in a mixed design.

2 | METHOD

2.1 | Participants

Separate groups of 26 participants were involved in the Oddball task (Mean age 20.77, range 18–32, 10 males, 2 left-handed) and the Regularity task (Mean age 19.62, range 18–30, 3 males, 0 left-handed). The experiments had local ethics committee approval and were conducted in accordance with the declaration of Helsinki (revised 2008).

Participants completed the Alcohol Use Disorders Identification Task (AUDIT; Saunders et al., 1993) to assess chance differences in drinking behavior and related problems between groups (Oddball task mean = 9.65, Regularity task mean = 12.00, maximum possible score = 40).

Individuals were excluded from participation if they had a current or previous diagnosis of alcohol or other substance use disorder, assessed by self-report. Participants who met the eligibility criteria were asked to consume a low-fat meal approximately an hour before each session and to refrain from consuming caffeine. Participants also had to provide an alcohol breathalyzer reading of zero mg/l upon arrival for their session.

The gap between alcohol and placebo sessions varied from 3 to 69 days in the Oddball task (median = 7), and 3–259 days (median = 8) in the Regularity task. Some participants did not return for their second sessions and were replaced.

2.2 | Apparatus

Participants were seated 57 cm from a 29 × 51 cm 60 Hz LCD monitor. Head position was stabilized with a chin rest. The experiment was programmed in Python on open-source PsychoPy software (Peirce, 2007). EEG data were recorded continuously from 64 scalp electrodes arranged according to the extended international 10–20 system. We used the BioSemi active-two EEG system, sampling at 512 Hz. To control for eye movements and blinks, bipolar HEOG and VEOG were monitored online. These external channels were not included in any analyses.

2.3 | Drink measurements

To obtain the alcoholic dose in a drink, we measured each participant's weight at the beginning of the session. The alcoholic experimental drinks contained a dose of vodka (Smirnoff Red Label) equivalent to 0.65 g of alcohol per kg of body weight for each participant, plus a no-sugar diet lemonade mixer (Schweppes Slimline) to make up a total beverage of 400 mL. Placebo drinks contained the equivalent amount of a no-alcohol substitute drink (Strykk Not Vodka) and the same amount of no-sugar diet lemonade.

2.4 | Measurement of blood alcohol concentration (BAC) levels

BAC was measured throughout the experimental session using an Alco-Sensor IV breath analysis device (Lion Alcometer 500). Participants were not informed of their BAC level during the experimental task. Three BAC measures were obtained from each participant: at the beginning of the session (prior to any drink consumption), 10 min after alcohol consumption (prior to starting the experimental task) and immediately after completion of the experimental task. To aid with participant blinding, BAC measurements were taken during alcohol and placebo sessions. At the beginning of the session for each participant, BAC was always 0 mg/L. In the Oddball task alcohol condition, average BAC was 0.21 (±0.06) mg/L 10 min after drink consumption. This rose to 0.27 (±0.07) mg/L after task completion. For the Regularity task alcohol condition, the average BAC was 0.22 (±0.06) mg/L 10 min after drink consumption. This rose to 0.27 (±0.05) mg/L after task completion.

Mixed ANOVA confirmed that BAC rose significantly during the interval when participants were completing the tasks ($F(1,48) = 14.332$, $p < .001$, $\eta_p^2 = 0.230$). This

did not interact with the between subjects' factors Task ($F(1,48) = 1.113$, $p = .297$) or Gender ($F(1,48) = 0.001$, $p = .971$). There were no other main effects or interactions (largest effect $F(1,48) = 2.136$, $p = .150$).

2.5 | Stimuli

The stimuli were the same as in the pilot experiment. Exemplars are shown in Figure 4. They were comprised 64 small Gaussian-masked dot elements with either 4 axes of reflectional symmetry or randomly arranged. The dot elements were on a gray disk with a diameter of approximately 3.5 degrees of visual angle.

The patterns were generated by an element-positioning algorithm during the experiment, so the same pattern was never repeated. The number of individual dot elements around the axes was either 0% (random) or 100% (symmetrical). Elements were constrained from overlapping or falling at the center of the pattern and were either green (50 CD/M^2) or black (0.15 CD/M^2). Elements were positioned on a gray background disk (40 CD/M^2) and a black background screen (0.15 CD/M^2). The individual dot element diameter was 0.43°.

For half of the participants, normal trials were black and rare oddball trials were green; for half it was the other way around (black oddball, green normal). Participants who had green (black) oddballs in the alcohol condition also had green (black) oddball in the placebo condition (with one exception). Due to complications with sampling and replacing participants, this balancing was not achieved in the Oddball task group (14/26 had green oddballs in the alcohol condition, and 15/26 participants had green oddballs in the placebo condition). Correct balancing was achieved in the Regularity task group.

2.6 | Procedure

Participants attended the EEG laboratory on the University campus between 12:00 and 19:00. They first provided a breath alcohol reading to ensure they had consumed no alcohol prior to the study. Participants then completed a study checklist to make sure they were aware of the conditions of participation and that they have had a meal beforehand. After the pre-screening, participants were fitted with the EEG cap.

Participants were then presented with three glasses containing a drink and were instructed to spend 12 min consuming the drink and approximately 3 min per glass. They completed a Subjective Effects Scale (SES, Morean et al., 2013) before they consumed any drink, in the middle of drink consumption, and after all drinks were consumed.

After drink consumption, the EEG experiment began with 24-trial practice block. Participants then completed the experimental task which lasted approximately 20 min. Finally, they completed another SES after the experiment was finished. Breathalyzer readings were taken at the beginning of each session, 10 min post-drink consumption, and after experimental task completion.

The EEG experiment involved 260 trials. These were broken into 10 blocks of 26. Within each block, there were 10 symmetry normal trials (e.g., black or green symmetry), 10 random normal trials (e.g., black or green random), 3 symmetry oddball trials (e.g., green or black symmetry), and 3 random oddball trials (e.g., green or black random). The trials were presented in a randomized order. Each trial began with a 1.5 s baseline period, followed by a 1.5 s pattern presentation. Participants in the Oddball task judged whether the patterns were green or black. Judgments were entered in a non-speeded fashion after stimulus offset using the

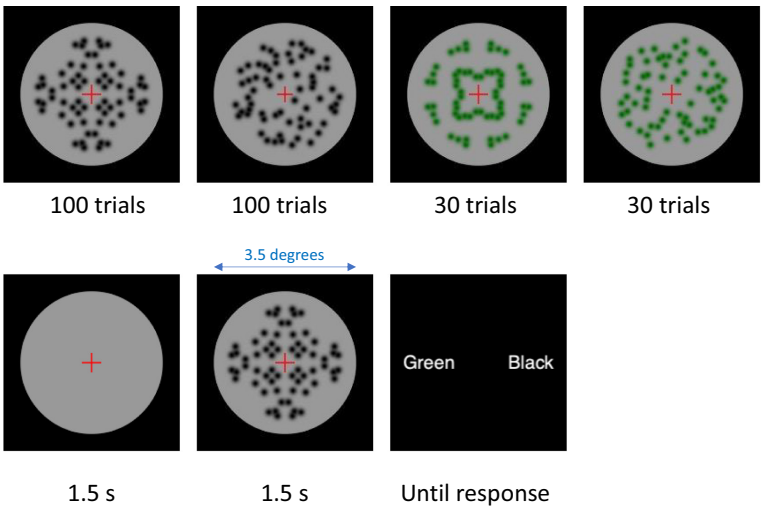

FIGURE 4 The top row shows example of stimuli in each of the four conditions. In these examples, black is normal and green is oddball. For approximately half the participants, the color categories were reversed. The lower row shows structure of a single trial from the Oddball task. The Regularity task was the same, except that participants reported whether patterns were "Regular" or "Random".

left (A) and right (L) keys on a standard keyboard. The response mapping varied unpredictably between trials. In half of the trials, the A key was used to report one option (e.g., Green, as in Figure 4), and the L key was used to report the other option (e.g., Black, as in Figure 4). The Regularity task was identical to the Oddball task, except that participants judged whether the stimuli were Regular or Random.

2.7 | EEG analysis

2.7.1 | EEG pre-processing

EEG data (.bdf files) were processed offline using the eeglab 2022.1 toolbox (Delorme & Makeig, 2004) in Matlab 2022b. All raw data, processed data, and codes for pre-processing and analysis are available on Open Science Framework (*Project 43alldata* in SPN catalog, https://osf.io/2sncj/). Data were re-referenced to scalp average, downsampled to 256 Hz, low pass filtered at 25 Hz, and segmented in −0.5 to +1 s epochs with −200 to 0 ms pre-stimulus baseline (resulting .set files in the 01_original folders). Channels were identified for interpolation with a semi-automated routine and zeroed during cleaning with Independent Components Analysis (ICA, .set files in the 02_ICA folders). ICA components were then removed from the data with the Adjust toolbox, and interpolated channels were then re-introduced (.set files in the 03_Pruned folders). EEG data were then re-referenced to the scalp average again. Any trial where amplitude exceeded ±100 microvolts at any electrode was removed (.set files in the 04_AmpEx folders). Finally, data were separated into conditions (05_epochs folders) and compiled in the SETData.mat structure. We then averaged over remaining trials for each subject and condition (resulting files in *Project 43* folder in the SPN catalog, https://osf.io/2sncj/).

On average, 5.77 ICA components were removed from each participant (min = 1, max = 23). ICA component removal rates were similar for Oddball alcohol (mean = 6.5, min = 1, max = 15), Oddball placebo (mean = 6.15, min = 2, max = 23), Regularity alcohol (mean = 6.15, min = 2, max = 14), and Regularity placebo (mean = 4.15, min = 1, max = 12).

Trial exclusion rate was around 4%–5% per condition in the Oddball task, and around 8% per condition in the Regularity task. Trial exclusion rate was 31% in the worst participant and condition. Therefore, all SPN waves were based on at least averaging over at least 69 trials per participant. One participant from the Oddball task was replaced because trial exclusion rate was over 50% (following the pre-registered criteria).

2.7.2 | ERP analysis

Although the SPN was the focus of this project, we also analyzed P1, N1, and P300 components. The additional components were chosen based on literature citing large alcohol effects on P1, N1, and P300 (Oddy & Barry, 2009; Porjesz & Begleiter, 2003). Analysis of P1, N1, and SPN was based on average amplitude across pre-registered spatiotemporal clusters. Electrodes were PO7, O1, O2, and PO8 (highlighted in Figure 1). P1 was defined as the maximum amplitude between 100 and 200 ms post-stimulus onset in each participant and condition. N1 was the minimum between 150 and 250 ms. N1 drop was then computed as the difference from P1 peak. The SPN was computed as the difference between symmetry and random waves in the 200–1000 ms window. This was split into two sub-windows which differed in the pilot experiment (early 200–400 and late 400–1000). Oddball trials were not included in these analyses. P300 was based on posterior central electrode cluster P1, PZ, and P2 from 300–800 ms post-stimulus onset. This was defined as the Oddball–Normal difference (averaging over symmetry and random trials).

2.7.3 | Frequentist analysis

P1 peak and N1 drop effects were analyzed with separate mixed ANOVAs. These had two within subjects' factors [Regularity (symmetry, random) × Block (alcohol, placebo)], and one between subject's factor [Task (Oddball, Regularity)]. The SPN was first computed as a difference between symmetry and random in two separate time windows. This was analyzed with mixed ANOVA [Interval (early, late) × Block (alcohol, placebo)] and one between subject's factor [Task (Oddball, Regularity)]. The presence of an SPN in all windows was confirmed with one-sample *t* tests (symmetry – random <0).

2.7.4 | Bayesian analysis

We supplemented frequentist analysis with Bayesian analysis, which can confirm the probability of the null hypothesis being true given the data (PH0|D) (Dienes, 2014). This allows us to statistically confirm that ERP amplitudes are similar in two conditions. We used conventional Bayes factor parameters of 1/3 and 3. For Bayesian *t* tests, we used the default Cauchy prior (with *r*-scale of 0.707). For Bayesian ANOVA, we report BF include. This involves parameter estimation and is not completely consistent between re-runs, so we avoid overinterpretation of borderline values. Bayesian

analyses were run in open-source JASP software (JASP Team, 2022).

3 | RESULTS

3.1 | Subjective and cognitive effects of alcohol

Results of the subjective effects scales and behavioral performance are shown in Figure 5. A minority of participants did not complete the subjective effects scales, so this analysis is based on 23/26 participants from the Oddball task and 24/26 from the Regularity task. Compared to placebo, alcohol made our participants feel less alert and more lightheaded. Other subjective effects were much less dramatic, with only a slight increase in contentment post-experiment. This was confirmed by an Effect × Drink × Time interaction ($F(8.324, 374.570) = 9.365$, $p < .001$, $\eta_p^2 = 0.172$) that was not further modulated by Task. The crucial Drink × Time interactions and pairwise differences between alcohol and placebo blocks are highlighted in Figure 5.

Discrimination between green and black stimuli in Oddball task was impaired by alcohol ($F(1,25) = 7.359$, $p = .012$, $\eta_p^2 = 0.227$). Discrimination between symmetrical and random stimuli in the Regularity task was also impaired by alcohol ($F(1,25) = 5.361$, $p = .029$, $\eta_p^2 = 0.177$). However, these differences only correspond to 1–2 additional error trials on average, and performance was typically near ceiling. Evidently, our 0.65 g/kg alcohol dose had modest emotional effects without impairing visual discrimination dramatically.

3.2 | Sustained posterior negativity

The SPN was analyzed in two a priori intervals (200–400 ms and 400–1000 ms). This was justified by the interval × drink interaction in the pilot study. In the Oddball task, SPN was reduced in the alcohol block (opposite of the pilot results). In the Regularity task, the SPN was similar in alcohol and placebo blocks. In both tasks, the SPN peaked at around 300 ms is then declined (Figure 6).

Mixed ANOVA found a strong main effect of Interval ($F(1,50) = 57.519$, $p < .001$, $\eta_p^2 = 0.535$). The main effect of Drink on SPN amplitude was not significant ($F(1,50) = 1.171$, $p = .284$, $\eta_p^2 = 0.023$). There was no Drink × Interval interaction ($F(1,50) = 0.288$, $p = .594$, $\eta_p^2 = 0.006$) and no Interval × Drink × Task interaction ($F(1,50) = 1.676$, $p = .201$, $\eta_p^2 = 0.032$).

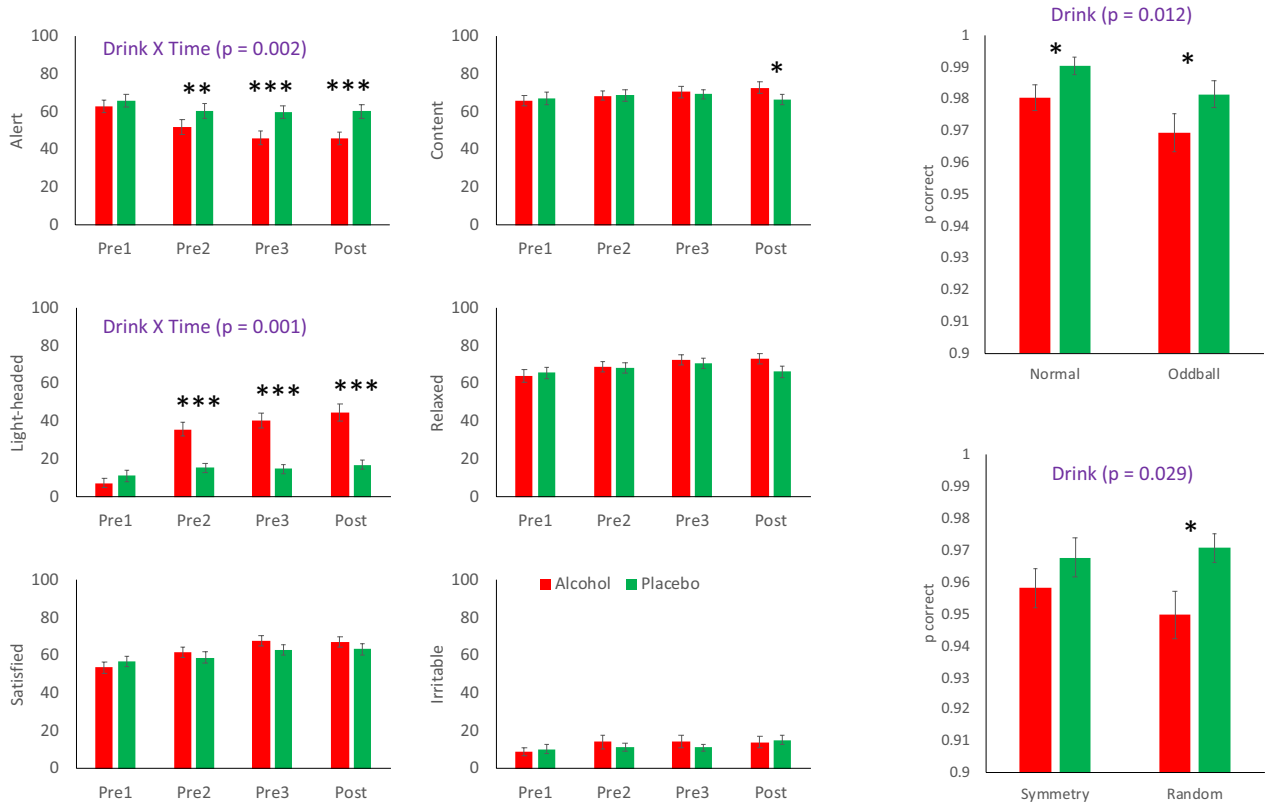

FIGURE 5 Emotional and cognitive differences between the alcohol (red) and placebo (green) conditions. *$p < .05$, **$p < .01$, ***$p < .001$. Error bars = ±1 S.E.M.

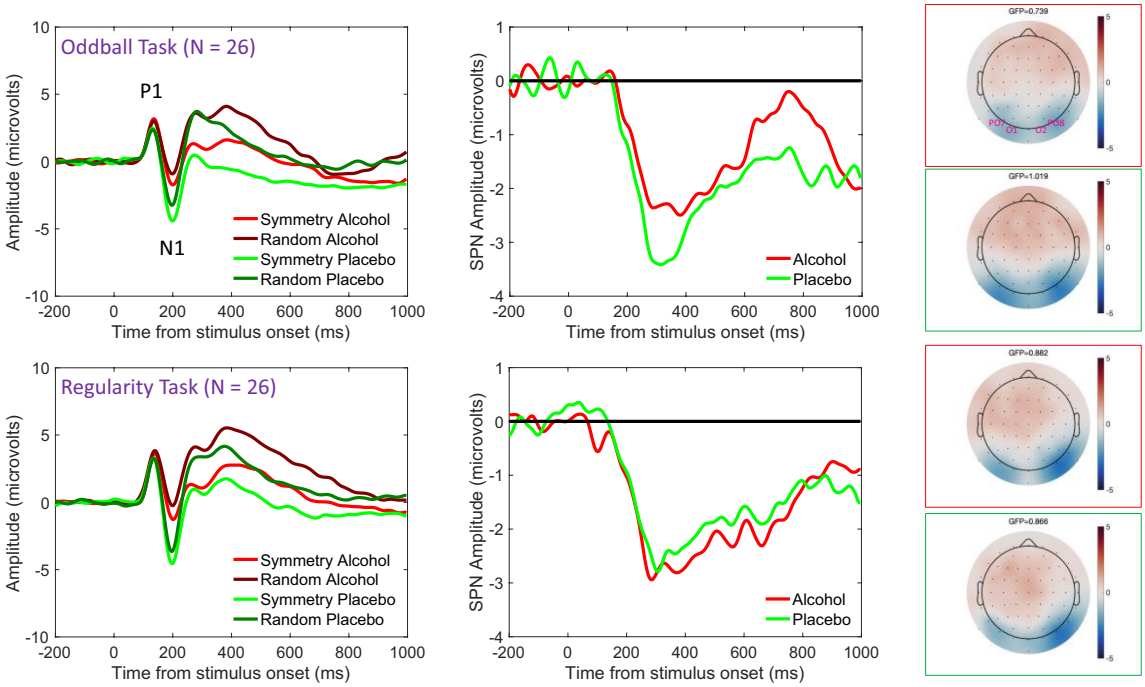

FIGURE 6 Top row: Oddball task. Bottom row: Regularity task. Left column: Grand average ERPs from electrode cluster [PO7, O1, O2, and PO8]. Central column: SPN difference waves (symmetry – random). Right column: Topographic difference maps from alcohol (red outline) and placebo (green outline), averaged over the 200–1000 ms window. GFP, Global Field Power (the standard deviation of amplitude across all 64 electrodes).

There was also no Drink × Task interaction ($F(1,50)=3.601$, $p=.064$, $\eta_p^2=0.067$). Despite this, we report the separate pre-registered analysis on each task: There was a main effect of Drink in the Oddball task ($F(1,25)=5.724$, $p=.025$, $\eta_p^2=0.186$), but not in the Regularity task ($F(1,25)=0.272$, $p=.607$, $\eta_p^2=0.011$).

Bayesian ANOVA confirmed the main effect of interval (BF include >100). Other effects and interactions were mostly inconclusive (BF include between 0.134 and 0.498). Bayesian pairwise comparisons between alcohol and placebo in the late interval are instructive, given that this was the important window in the pilot study. There was no support for presence or absence of an alcohol effect in the Oddball task (BF10 = 0.814). However, analysis supports the *absence* of an alcohol effect in the Regularity task (BF01 = 4.631).

Overall, alcohol has no consistent effect on SPN amplitude. There was no effect of alcohol on SPN amplitude in the Regularity task, and the evidence from Oddball tasks is mixed, especially when considering the contradictory pilot results.

All conditions produced a large SPN signal as compared to zero. This is illustrated in three ways in Figure 7. The top row shows SPN waves with 95% CI, the middle row shows SPN amplitude in violin plots with various descriptive and inferential statistics, and the bottom row shows prior and posterior plots associated with Bayesian one-sample *t* tests. We can also see that SPNs are normally distributed around the grand average and present in most participants (at least 23/26, $p<.001$ Binomial test). Figure 7 allows visualization of individual differences in SPN amplitude. It also shows that our grand average SPNs were very strong signals, so statistical choices are academic in this case.

3.3 | Other ERP components

While the SPN was the focus of this study, other ERP components require comment. Unlike the pilot study, the P1 peak was slightly enhanced by alcohol (red waves) compared to placebo (green waves). This was confirmed as a main effect of Drink ($F(1,50)=5.368$, $p=.025$, $\eta_p^2=0.097$). There was no main effect of Regularity or Task and no interactions (largest effect $p=.262$). Bayesian ANOVA analysis confirmed the absence of these effects (BF include <1/3) but could not confirm the presence of an effect of Drink (BF include approximately 1). Therefore, we do not overinterpret the small and unexpected effect of alcohol on P1 peak.

The N1 drop was larger in the Symmetry than Random conditions, and larger in the Placebo the alcohol conditions. N1 effects were similar in both tasks. This was confirmed by main effects of Regularity ($F(1,50)=38.032$, $p<.001$, $\eta_p^2=0.432$) and Drink ($F(1,50)=53.572$, $p<.001$, $\eta_p^2=0.517$). The Drink × Task interaction was not

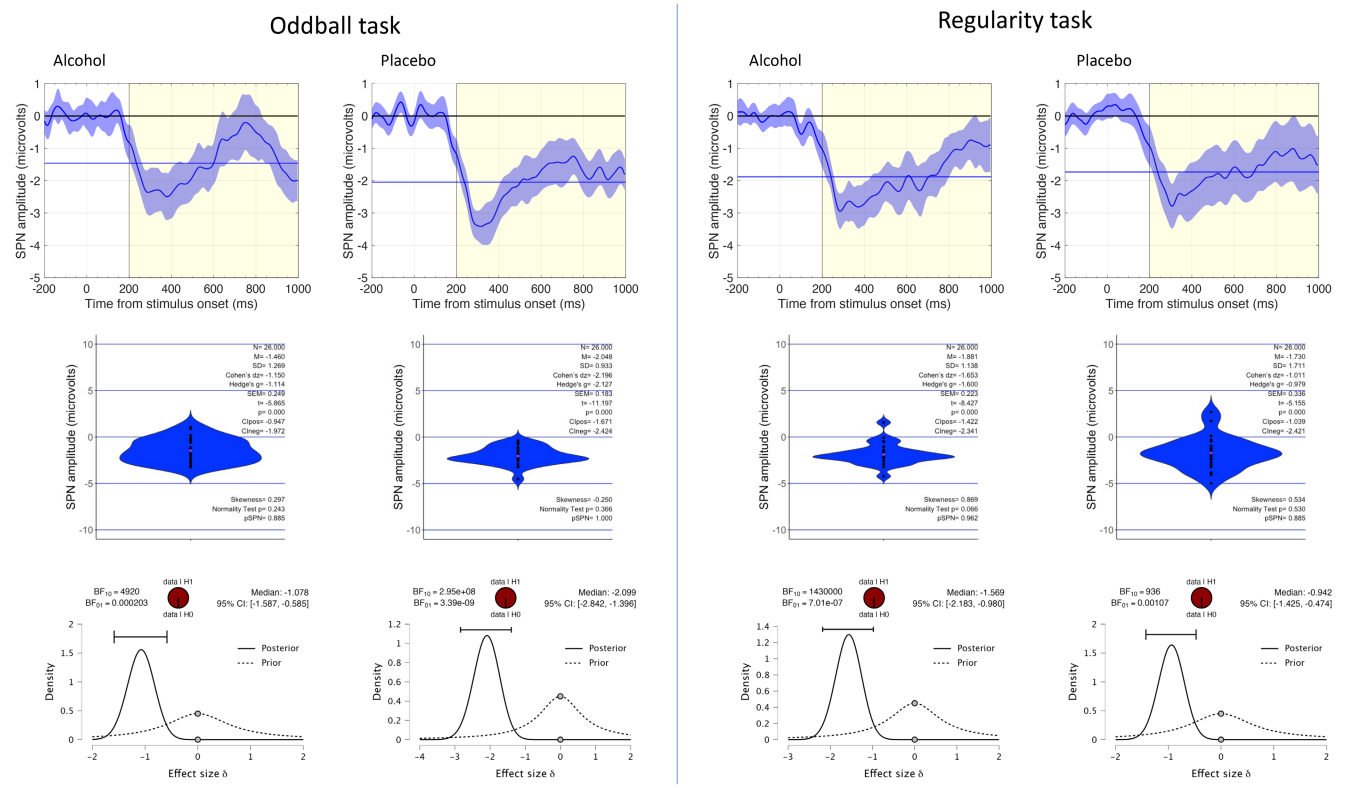

FIGURE 7 Alternative visualizations of the alcohol and placebo SPN waves in the Oddball task (left) and Regularity task (right). Top row shows SPN waves with 95% Confidence interval ribbon. When this falls below zero, difference between symmetry and random is significant at the 0.05 level. The horizontal blue line indicates mean SPN amplitude in the analyzed window (highlighted yellow). The central row shows violin plots with descriptive and inferential statistics. The bottom row shows prior and posterior plots associated with Bayesian one-sample *t* tests. Evidence for an SPN effect is overwhelming in all four conditions.

significant ($F(1,50) = 3.663$, $p = .061$, $\eta_p^2 = 0.068$), and there were no other main effects or interactions (largest effect $p = .413$). Bayesian ANOVA analysis supported the presence of Drink and Regularity effects (BF include >100). However, it did not consistently support the absence of most other effects and interactions.

P300 results are shown in Figure 8. In the Oddball task, the expected P300 effect was found at posterior central electrodes (P1, Pz, and P2). This was apparently larger in the alcohol than placebo condition. There was no P300 in the Regularity task. Mixed ANOVA found main effect of Task ($F(1,50) = 10.383$, $p < .001$, $\eta_p^2 = 0.172$) and Task × Drink interaction ($F(1,50) = 5.037$, $p = .029$, $\eta_p^2 = 0.092$). Additional analysis found no main effects or interactions involving Oddball color (largest effect, $F(1,47) = 1.128$, $p = .294$, $\eta_p^2 = 0.023$).

In the Oddball task, the apparent difference between alcohol and placebo conditions was not significant ($t(25) = 1.884$, $p = .071$), although the P300 was present in the placebo condition ($t(25) = 4.458$, $p < .001$, $d_z = 0.874$), but not alcohol condition ($t(25) = 1.536$, $p = .137$, $d_z = 0.301$). Bayesian analysis was not conclusive here and only confirmed the presence of a main effect of Task on P300 (BF include >5).

While there is a risk of running multiple analyses on multiple ERP components without correcting for familywise error rate, the alcohol-induced reduction in N1 amplitude appears very robust. All other alcohol-induced ERP differences should not be overinterpreted without replication.

3.4 | Individual differences

We also ran several exploratory analyses on individual differences (see supplementary materials on SPN catalog/Project 43). Most importantly, we found that AUDIT scores negatively correlated with the size of alcohol-induced changes to SPN amplitude. Specifically, alcohol increased SPN amplitude in heavy drinkers and reduced SPN amplitude in light drinkers.

The fact that alcohol had no effect on mean SPN amplitude in the Regularity task could be partly due to the fact that the participants had marginally (although

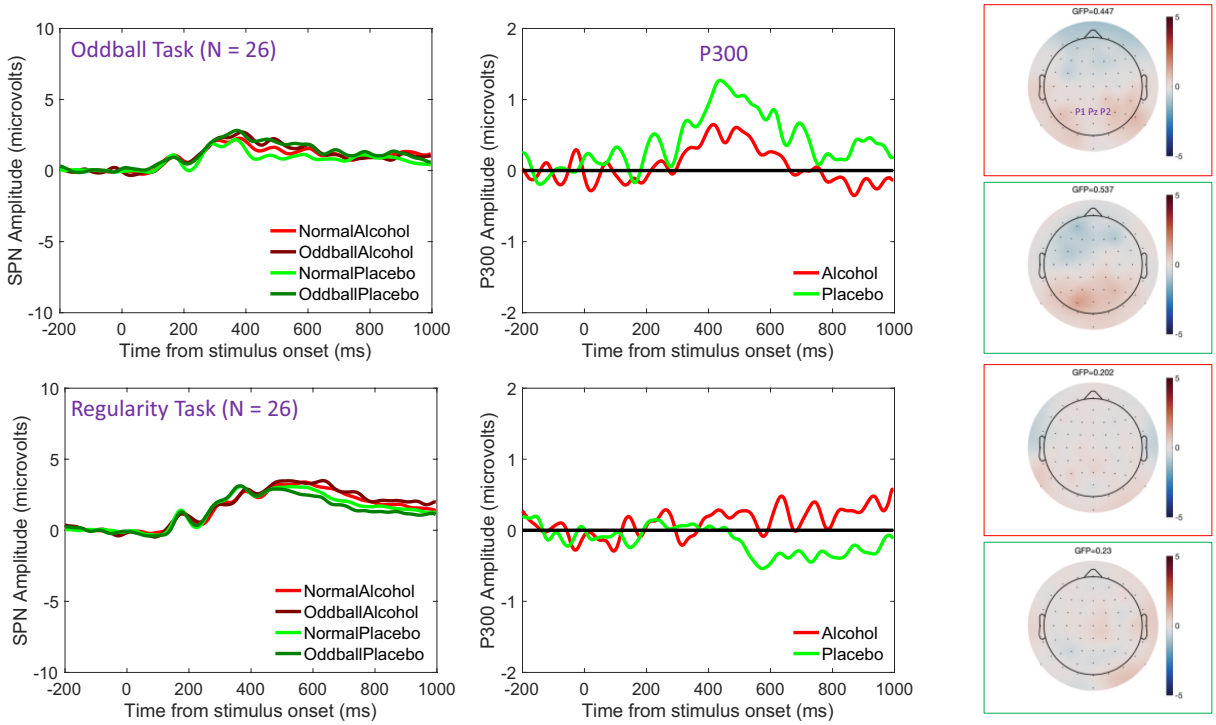

FIGURE 8 P300 analysis. Layout conventions the same as Figure 6. Waves are from electrode clusters P1, Pz, and P2.

non-significantly) higher AUDIT scores in the Regularity task (Mean AUDIT in Oddball task = 9.65 (SD = 4.84); Mean AUDIT in Regularity task = 12.00 (SD = 3.96), $t(50) = 1.911$, $p = .062$, $d_s = 0.530$).

Regression analysis supports this possibility (Figure 9). The DV was alcohol-induced change in SPN amplitude. When entered alone, AUDIT explained 17.8% of variance ($R^2 = 0.178$, $F(1,50) = 10.818$, $p = .002$). Inclusion of Task to the model explained little additional variance (R^2 change = 0.024, $F(1,49) = 1.466$, $p = .232$). Given this analysis, we cannot confidently claim that Task has an independent effect on alcohol-induced changes in SPN amplitude, beyond the effect of AUDIT.

Unfortunately, we did not obtain AUDIT scores from the 13 participants in the pilot study. It could be that the pilot participants were relatively heavy drinkers, explaining why alcohol enhanced the SPN in this sample. This is plausible, considering that the maximum possible AUDIT score is 40, and the pivot from SPN reduction to enhancement happened at around 15 in the Oddball task.

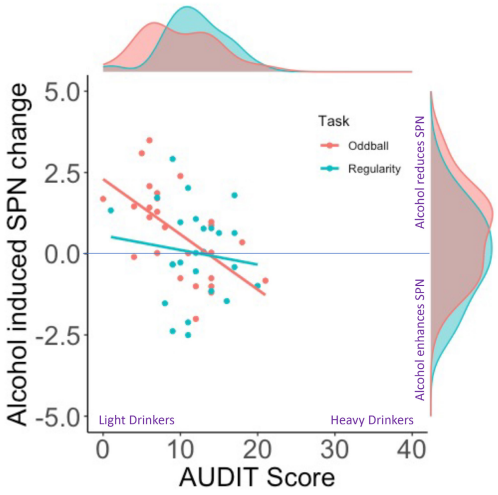

FIGURE 9 Alcohol-induced SPN change as a function of AUDIT score in each task. The marginal difference in alcohol-induced SPN change between tasks may be partly due to marginal differences in drinking behavior between samples.

4 | DISCUSSION

Previous research has shown that the SPN is robust to experimental variations of task, spatial attention, and visual memory load. Here, we investigated whether the SPN is also robust to alcohol-induced changes in mental state. A pilot Oddball task found a surprising alcohol-induced SPN enhancement, particularly in the later part of the interval. In contrast, the new Oddball task showed a small alcohol-induced SPN reduction. There was no effect of alcohol on SPN amplitude in the new Regularity task. It could be that task-relevant symmetry processing in the Regularity task is more robust and can thus survive moderate doses of alcohol.

However, exploratory analysis indicated that task differences are partly explained by individual differences

in drinking behavior. Participants who drink more show an alcohol-induced SPN enhancement, and participants who drink less show an alcohol-induced SPN suppression. By chance, the participants in the Oddball task were lighter drinkers. This means we cannot overinterpret task differences.

Despite the remaining questions, we can confidently state that there was a large SPN in all tasks and conditions. The SPN is probably altered by alcohol in a complicated way, which depends partly on the task and partly on individual differences in drinking behavior, but it was never substantially reduced, let alone abolished.

In contrast, the N1 was consistently reduced by alcohol in the Pilot study, the Oddball task, and the Regularity task. We conclude that alcohol-induced changes in N1 amplitude are more consistent and reliable than alcohol-induced changes in SPN amplitude. Stimulus regularity and alcohol consumption both affected N1 amplitude, but these effects did not interact. This suggests that early symmetry processing during the N1 time window is not strongly altered by alcohol. There was also some weak evidence that the P1 was enhanced by alcohol, and that the P300 in the Oddball task was reduced by alcohol. However, these two effects were much smaller than the alcohol-induced N1 reduction.

This project highlights the importance of replication in cognitive neuroscience. It would probably have been possible to publish the pilot results by rhetorically blurring the distinction between confirmatory and exploratory research. The narrative "alcohol enhances symmetry sensitivity" is readily understandable and leads to interesting discussions about aesthetic experience. For instance, although evidence is mixed, it has been suggested that alcohol makes faces look more beautiful because it makes them look more symmetrical (Halsey et al., 2010; Harvey et al., 2024; Souto et al., 2008). Our pilot results were consistent with this. However, hasty publication of unexpected results from small sample experiments has a net negative effect on reproducibility (Bishop, 2019; Button et al., 2013; Munafò et al., 2017). We were keen to avoid this, and a strength of the current work is that we repeated the pilot experiment and expanded it with a new Regularity task. While we are now less confident that the SPN is systematically altered by alcohol consumption, we have not polluted the literature with an eye-catching fluke!

The current work adds to previous studies that have found an SPN in Oddball tasks (Höfel & Jacobsen, 2007; Makin et al., 2013). Here, we went one step further and found an SPN in an Oddball task when participants were intoxicated. However, this automaticity may be qualified by known boundary conditions: weaker regularities, such as symmetry embedded in noise, might not produce an SPN under these conditions (Makin et al., 2020a). Moreover, SPNs generated by other kinds of visual symmetry, such as rotation, translation, and Glass patterns, might be more vulnerable to task and alcohol manipulations. Most importantly, these results only generalize to symmetry in the retinal image. This is an important limitation: many objects are symmetrical, but they only project a symmetrical image onto the retina when viewed in the frontoparallel plane (Sambul et al., 2013; Sawada & Pizlo, 2008). Symmetrical objects do not project a symmetrical image when viewed in perspective. Perspective symmetry can be detected when it is task relevant (Bertamini et al., 2022) but may not be processed automatically when it is not (Keefe et al., 2018; Makin et al., 2015; Rampone et al., 2019).

5 | CONCLUSIONS

This was the first study to investigate the effect of alcohol on the SPN. We found that the SPN is robust to moderate intoxication. Amplitude may be slightly enhanced or reduced by alcohol, partly depending on task and individual differences in drinking behavior. However, the SPN response to our stimuli is never abolished.

AUTHOR CONTRIBUTIONS

Elena Karakashevska: Data curation; formal analysis; investigation; methodology; writing – original draft; writing – review and editing. **Yiovanna Derpsch:** Conceptualization; methodology; project administration; writing – review and editing. **Andrew Jones:** Formal analysis; methodology; project administration; resources. **Alexis D. J. Makin:** Formal analysis; methodology; project administration; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

None.

DATA AVAILABILITY STATEMENT

All EEG data sets, materials, and analysis codes from SPN research at the University of Liverpool are housed in the Complete Liverpool SPN catalog on Open Science Framework (https://osf.io/2sncj/). As described in Makin et al. (2022), the SPN catalog has folders for each SPN project. The pilot study is Project 26, and the current study is Project 43. Other researchers could assess computational

reproducibility by running an alternative analysis. They could also use raw data for a new analysis (e.g., changes in pre-stimulus alpha power when intoxicated). Materials to validate the statistical tests reported in the results section (such as SPSS files and R codes) are in subfolder called "Results and analysis" in Karakashevska et al. Supplementary materials are also in this folder.

ORCID

Elena Karakashevska https://orcid.org/0000-0002-3423-4716
Yiovanna Derpsch https://orcid.org/0000-0002-4380-5058

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article. **Supplementary Material 1.**

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