The extrastriate symmetry response is robust to alcohol intoxication.

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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 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.

Introduction

Visual symmetry contributes to perceptual organisation 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 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 (ERPs) 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, 2020, Palumbo et al., 2015) The SPN is generated whatever the participant's task, but amplitude is enhanced when symmetry is task relevant (Figure 2).

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Figure 1. Results of Makin et al. (2020). 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).

Derpsch et al. (2019) found that whilst 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 unattended regions. In addition, Derpsch et al. (2021) found that the SPN is not diminished by a concurrent visual working memory task, suggesting the SPN is also robust to variations in visual memory load. In the current work, we extended this research program by investigating the SPN is equally sensitive to alcohol-induced changes in mental state.

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Figure 2. Results from Makin et al. (2020). The parametric SPN response was evident in 5 tasks (from 5 different groups of 26 participants) but selectively enhanced in the regularity task (left). Figure from Makin et al. (2023).

It is likely that all brain functions are altered by alcohol intoxication, directly or indirectly (Field et al., 2010; Fillmore & Van Selst, 2002). Steele and colleagues (e.g., Steele and Josephs, 1988, Steele and Josephs, 1990) proposed a model of alcohol effects that focused on alcohol's influence on attentional processes. According to this attention-allocation model, intoxication restricts the focus of attention to only the most salient cues in the environment, such that other available cues are not fully processed (Sayette, 1999). A different model proposed by Vogel-Sprott and colleagues posits that, rather than restricting attentional focus, alcohol impairs a form of response inhibition (e.g. Vogel-Sprott et al., 2001). This model is based on a theory of cognitive control (Logan and Cowan, 1984) positing that behavioural activation and behavioural inhibition stem from two independent cognitive processes.

Acute and chronic effects of alcohol consumption are well studied (for reviews see Creupelandt et al., 2019; Dry et al. 2012; Stavro et al., 2013; Oscar-Berman et al., 2014). Alcohol has many effects on vision: altering eye movement (Marinkovic et al., 2013), contrast sensitivity (Pearson, & Timney, 1998), colour perception (Brazil et al., 2015; Zrenner et al. 1986), retinal image quality and night vision performance (Castro et al., 2014), as well as deteriorating binocular vision (Hogan & Linfield, 1983).

Alcohol usually supresses ERPs, but there is considerable variability between components. A meta-analysis by Fairbairn et al. (2021) found that alcohol reduces ERP components linked with attention (P3b), automatic auditory processing (MMN), and performance monitoring (ERN/FRN). Alcohol also reduces the P300 response negative feedback (Euser et al. 2011) and a temporoparietal N180 that indexes prelexical pattern-recognition processes. In contrast, alcohol may have little, or no effect on components linked to executive control (N2b) or stimulus classification (N2c). Finally, alcohol significantly *increases*the amplitude of the N450, particularly on trials evoking sympathetic arousal (Marinkovic et al., 2006). The current research tested whether the SPN is reduced by alcohol, like many other ERPs.

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 coloured 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. 2021 for meta-analysis). In one session they drank 0.65g/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. 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, $p < .001$, $d_z = 1.237$).

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

Current study

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

As well as increasing sample size, we increased the number of trials to 100 per condition and increased the number of oddball trials to 60 (from 80 and 32 in the pilot study). This improved signal quality, and the additional oddball trials allowed us to measure a more reliable P300 component. Furthermore, to reduce variability between participants, the absorption period of alcohol was kept to 10 minutes.

Given the pilot study results, we predicted that alcohol would have no effect on positive P1 peak, reduce the negative N1 dip, and enhance the SPN in the 400-1000 ms window (https://aspredicted.org/hj9eh.pdf). We also predicted that alcohol would reduce the P300 response to oddballs. Analysis of the Oddball task confirmed our predictions regarding P1, N1 and P300. 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 colour. 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 P1, reduce N1, and have no effect on the SPN. These predictions were confirmed. We also predicted that the SPN would be larger overall in the Regularity task than the Oddball task, although this was not confirmed (https://aspredicted.org/yn3vq.pdf).

Results of the Oddball and Regularity tasks are presented together and as separate groups in a mixed design.

Method

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 was conducted in accordance with the declaration of Helsinki (revised 2008).

Participants completed the Alcohol Use Disorders Identification Task (AUDIT; Babor et al., 2001) to assess chance differences in drinking behaviour 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 that 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 breathalyser reading of zero mg/l upon arrival for their session.

The gap between alcohol and placebo sessions varied from 3-69 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.

Apparatus

Participants were seated 57 cm from a 29 X 51 cm 60Hz 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 was 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.

Drink measurements

The alcoholic experimental drinks contained a dose of vodka (Smirnoff Red Label) equivalent to 0.65g of alcohol per kg of body weight, plus a no-sugar diet lemonade mixer (Schweppes Slimline) to make up a total beverage of 400 ml. Placebo drinks consisted of 400 ml of mixer of non-alcoholic vodka (Strykk Not Vodka) and the same no sugar diet lemonade.

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 actual BAC level during the experimental task. To aid with participant blinding, BAC measurements were taken during alcohol and placebo sessions.

Stimuli

The stimuli were the same as the pilot experiment. Exemplars are shown in Figure 4. They were comprised on 64 small Gaussian-masked dot elements with either 4 axes of reflectional symmetry, or randomly arranged. The dot elements were on a grey 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 centre of the pattern and were either green (50 Cd/m^2) or black (0.15 Cd/m^2). Elements were positioned on a grey 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 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.

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 3 glasses containing a drink and were instructed to spend 12 minutes consuming the drink and approximately 3 minutes 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 minutes. Finally, they completed another SES after the experiment was finished. Breathalyser readings were taken at the beginning of each session, 10 minutes 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 second baseline period, followed by a 1.5 second 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 left (A) and right (L) keys on a standard keyboard. The response mapping varied unpredictably between trials. On half 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.

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Figure 4. The top row shows example of stimuli in each of the 4 conditions. In these examples, black is normal and green is oddball. For approximately half the participants, the colour 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'.

EEG analysis

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 catalogue, https://osf.io/2sncj/). Data was re-referenced to scalp average, downsampled to 256 Hz, low pass filtered at 25Hz, and segmented in -0.5 to + 1 second 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, Jung, 2000, .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 was then re-referenced to the scalp average again. Any trial where amplitude exceeded +/- 100 microvolts at any electrode were removed (.set files in the 04_AmpEx folders). Finally, data were separated into conditions (05_epochs folders) and compiled in the SET Data.mat structure. We then averaged over remaining trials for each subject and condition (resulting files in*Project 43* folder in the SPN catalogue, 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).

ERP analysis

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 trails 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).

Frequentist analysis

P1 peak and N1 drop effects were analysed with separate mixed ANOVAs. These had two within subjects' factors [Regularity (symmetry, random) X 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 analysed with mixed ANOVA [Interval (early, late) X 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).

Bayesian analysis

We supplemented frequentist analysis with Bayesian analysis, which can confirm the probably 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).

Exploratory individual differences analysis

Additional exploratory analyses were conducted to examine whether individual differences between participants explains the magnitude of alcohol induced effects within the Oddball and Regularity tasks, and whether any chance differences between samples could explain differences between tasks.

For this analysis, we computed the magnitude of *alcohol induced change* for each variable. For instance, P1 peak was measured in alcohol and placebo conditions, and the difference between these is the alcohol induced P1 effect.

For each participant, we measured alcohol-induced changes to P1 peak, N1 drop, P300, SPN, behavioural performance, light-headedness, and alertness. For this analysis, the SPN was defined averaged over early and late windows. We also computed gap between sessions, AUDIT score and age. Correlations between these 10 variables were assessed within Oddball and Regularity tasks separately. Light-headedness and alertness scores were averaged over the final pre-experiment rating and post experiment rating.

Data availability

All EEG datasets, materials, and analysis codes from SPN research at the University of Liverpool are housed in the Complete Liverpool SPN catalogue on Open Science Framework (https://osf.io/2sncj/). As described in Makin et al. (2022), the SPN catalogue has folders for each SPN project. The pilot study is project 26 and new study is project 43. This catalogue is designed for maximum transparency. 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 double check 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 1 is also in here.

Results

Subjective and cognitive effects of alcohol

Results of the subjective effects scales and behavioural 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 X Drink X 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 X 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.

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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.

ERP analysis

Grand average ERPs from our posterior electrode cluster [PO7, O1, O2 and PO8] are shown in Figure 6.

P1 and N1

Unlike the pilot study, the P1 peak was slightly enhanced by alcohol (red waves) compared to placebo (green waves). This was confirmed 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 X Task interaction was not 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.

Sustained Posterior Negativity

The SPN was analysed in two a priori intervals (200-400 ms and 400-1000 ms). 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 then declined.

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

There was also no Drink X Task interaction (F (1,50) = 3.601, p = .064, ηp^2 = 0.067). Despite this, we report the separate pre-registered analysis on each task: There was a main effect Drink in the Oddball task (F (1,25) = 5.724, p = .025, ηp^2 = 0.186), but not in the Regularity task (F (1,25) = 0.272, p = .607, ηp^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. In contrast, alcohol reliably reduces N1 amplitude in all cases.

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Figure 6. ERP and SPN waves. 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).

We also note that 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, 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 < 0.001 Binomial test)

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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 analysed 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.

P300

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, ηp^2 = 0.172) and Task X Drink interaction (F (1,50) = 5.037, p = .029, ηp^2 = 0.092). Additional analysis

found no main effects or interactions involving Oddball colour (largest effect, F (1,47) = 1.128, p = .294, ηp^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).

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Figure 8. P300 analysis. Layout conventions the same as Figure 6. Waves are from electrode cluster P1, Pz and P2, from 300-800 ms post stimulus onset.

Exploratory analysis of Individual differences

An exploratory analysis of individual differences in each task is illustrated in Figure 9. Together the two matrices cover 110 correlations, are not corrected for multiple comparisons. We would expect 5.5 significant correlations by chance. However, some of the larger effects may not be false positives and warrant tentative interpretation and discussion (p < .01, black boxes in correlation matrix and scatterplots in Figure 9).

Within task exploratory analysis

In the Oddball task (Figure 9A) alcohol induced changes to SPN amplitude (illustrated with upward arrow in the ERP inset) correlated with participant's self-reported drinking behaviour (AUDIT) (r = -0.59, p = .002). Alcohol reduced the SPN in participants who drink less, but increased the SPN in participants who drink more. The same trend was present, but non-significant, in the Regularity task (r = -19, p = .347). In the Oddball task, alcohol induced subjective light-headedness correlated with alcohol induced P300 reduction. The direction of this effect is surprising: Participants who were more subjectively affected by alcohol bucked the overall trend for alcohol to reduce P300. However, this was driven by two anomalous participants, with usually high alcohol induced light-headedness.

In the Regularity Task (Figure 9B), alcohol induced change in behavioural performance negatively correlated with alcohol induced change in N1 drop. Also in the Regularity task, alcohol induced changes in behavioural performance correlated with alcohol induced changes in subjective alertness.

We should emphasize that the variables in the correlation analyses were differences between alcohol and placebo conditions. We can also run the same analysis with amplitude averaged across alcohol and placebo conditions. Although the literature suggests N1 and P300 components are altered by chronic drinking, there was no evidence that amplitude of any of our ERP components correlated with AUDIT, and no other correlations were significant at the 0.01 level (see supplementary materials on SPN catalogue /Project 43).

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Figure 9. Correlations between AUDIT score, age, gap between sessions, and alcohol induced changes five ERP components plus subjective light-headedness, subjective alertness, and behavioural performance in A) Oddball Task and B) Regularity Task . Significant correlations are highlighted with bold borders. Scatter plots illustrate the stronger effects.

Between-task exploratory analysis

These observations raise an important possibility: The fact that alcohol had no effect on mean SPN amplitude in the Regularity task could be partly due the fact that the participants had marginally (although 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 10). The DV was alcohol-induced change in SPN amplitude. When entered alone, AUDIT explained 17.8% of variance in the DV (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.

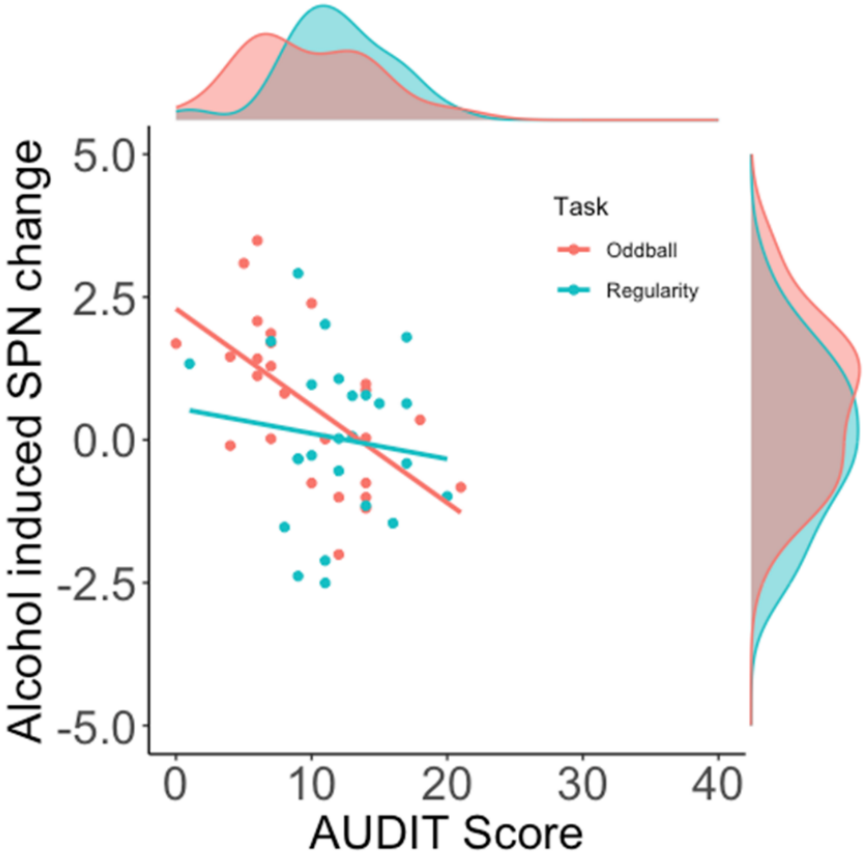

Figure 10 – Alcohol induced SPN change as a function of AUDIT score in each task . The marginal difference in alcohol induced SPN change between tasks may be partly due to marginal differences in drinking behaviour between samples.

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 for the first time. A pilot Oddball task found a surprising alcohol induced SPN enhancement. 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 behaviour. Participants who drink more show an alcohol induced SPN enhancement, participants who drink less show an alcohol induced SPN suppression. By chance, the participants in the Oddball task were lighter drinkers. This aspect of the results requires replication. However, it 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 behaviour, but it was never substantially reduced, let alone abolished.

In contrast, 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. The N1 is also generated by mid-level visual processes involved in perceptual grouping and gestalt formation, and these initial visual operations may be more directly sensitive to alcohol that the subsequent SPN.

Interestingly, 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. Moreover, there was some weak evidence that the P1 was enhanced by alcohol, and that the P300 in the Oddball task was reduced by alcohol. These two effects were much smaller than the alcohol induced N1 reduction.

Our reliable N1 results are broadly consistent with the attention-allocation model (Sayette, 1999). Alcohol may narrow attention, so potential distractions go unnoticed. This lets drinkers live in the moment, untroubled by stressful memories and duties. Distracting visual stimuli are also more likely to remain unconscious if activations that generate the N1 are dampened.

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, it has been suggested that alcohol makes faces look more beautiful because it makes them look more symmetrical (Halsey et al., 2010; 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 replicated 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., 2020). 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 relevant (Keefe et al., 2018; Makin et al., 2015; Rampone et al., 2019).

Conclusions

This was the first ever 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 behaviour. However, the SPN response is never abolished. The alcohol induced N1 reduction is much more consistent.

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