



Registered Report

Cardiac signals and the interference of reward on attention and inhibitory control



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ABSTRACT

Interoceptive responses can modulate cognition and behavior; discrete cardiac signals can shape emotional and motivational adaptation towards reward-related cues, but also affect response inhibition. Novel addiction perspectives posit an interoceptive basis for the interplay between substance-related reward processing and inhibitory control, but there is a lack of behavioral evidence for this relationship. In this registered report, we investigated whether reward cues modulate cardiac-facilitated attention and motor inhibition. Fifty social drinkers completed an attentional visual search task and two instances of a stop signal task, in which alcohol or neutral stimuli were presented as targets or distractors. Stimuli were presented in synchrony with participants' cardiac phase (systole vs. diastole). This design allowed us to test whether cardiac signals amplify attentional biases in the presence of alcohol cues and influences inhibitory control. Overall, our results were predominantly null: alcohol cues did not produce significant attentional interference in any task, limiting conclusions about interoceptive modulation of cognitive abilities by cardiac phase. However, we replicated a previous finding that synchronizing stop signals at systole improved motor inhibition. This provides strong evidence that cardiac phase can facilitate inhibitory processes in the stop signal task. Although more sensitive paradigms are needed to clarify how cardiac rhythms interact with alcohol cues to influence attention and inhibition, our replication of systolic facilitation highlights the promise of cardiac cycle-based approaches in interoception research. Future studies may benefit from refining task design and considering craving states to more effectively capture the potential interoceptive influences on attention and inhibitory control.

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1. Introduction

Interoception is the processing of physiological signals in the brain, incorporating strength of afferent signals and preconscious impact of such signals, through to conscious perception of bodily feelings and beliefs, insight, and attention to body sensations (Suksasilp & Garfinkel, 2022). Interoceptive processing provides a novel perspective to understand the role of body-brain communication in human behavior (Craig, 2003; Khalsa et al., 2018). Interoceptive signals can mediate emotional (Critchley & Garfinkel, 2017), cognitive (Critchley & Garfinkel, 2018) and behavioral responses that underlie different mental health disorders (Khalsa et al., 2018; Suksasilp & Garfinkel, 2022). Dysregulated reward processing is a crucial component of psychopathology (Anderson, 2021; Baskin-Sommers & Foti, 2015), particularly relevant for addiction, which is characterized by an inability to inhibit behavioral responses in the presence of incentive salient rewards (Berridge & Robinson, 1998; Robinson & Berridge, 1993). In this registered report we examine whether interoceptive signals can modulate reward-related effects in inhibitory control and attentional biases in a sample of social alcohol drinkers.

Interoceptive signals generated in the viscera are integrated through afferent nervous pathways in interoceptive neural hubs (Critchley & Harrison, 2013), and can be studied across different interoceptive axes (Garfinkel, Manassei, et al., 2016). Cardiac signals are particularly relevant to understand the role of interoception on cognition and behavior across multiple dimensions (Critchley & Garfinkel, 2017, 2018); from higher-order levels of interoception, such as the conscious perception of individual heartbeats (Garfinkel, Seth, Barrett, Suzuki, & Critchley, 2015), intermediate levels, such as the effect of cardiac signals on behavior and cognition (Garfinkel et al., 2014; Leganes-Fonteneau, Buckman, Suzuki, Pawlak, & Bates, 2020), to lower-order measures of cardiovascular reactivity, such as baroreceptor functioning or heart-rate variability in the 0.1 Hz frequency band (Leganes-Fonteneau, Bates, Muzumdar, et al., 2021). These dimensions allow multimodal approaches to understanding the interoceptive basis for different addictive processes.

Baroreceptors are stretch-sensitive mechano-receptors located in the aortic walls that signal the timing and strength of each individual heartbeat to the brain via vagus nerve. The activity of arterial baroreceptors can be mapped onto the QRS complex of the electrocardiogram, reflecting different phases of the cardiac cycle. During the systolic phase, when the heart pumps blood out of the heart (~300 ms after the R-wave, corresponding with the pulse wave), baroreceptors fire, whereas during the diastolic phase (coinciding with the R-wave), baroreceptors remain silent (Garfinkel, Tiley, et al., 2016; Lewis, Rittogers, Froester, & Boudoulas, 1977; Sato et al., 2019). By pairing stimulus presentation with different phases of the cardiac cycle (i.e. systole vs. diastole) it is possible to observe how intermediate levels of interoception modulate cognitive and behavioral responses (Critchley & Garfinkel, 2018; Garfinkel et al., 2014).

For example, in an emotional visual search task (Leganes-Fonteneau et al., 2020) faces presenting a “happy” or

“disgust” emotion were more easily detected at systole compared to diastole, whereas the detection and identification of “fearful” faces was inhibited. These findings expand previous research (Azevedo, Badoud, & Tsakiris, 2018; Garfinkel et al., 2015), showing that the detection of broadly emotionally salient stimuli can be modulated by afferent cardiac signals. Cardiac signals can also modulate cognitive responses in a variety of paradigms. For example, in a Stop Signal Task, Rae et al. (2018) found that presentation of the Stop signal at systole improved motor control compared to diastole, as evidenced by decreased Stop Signal Reaction Times (SSRT) and increased Stop Signal Delay (SSD). This points towards the ability of afferent cardiac signals to participate in inhibitory motor control processes, understood as the ability to stop, change or delay a response in favour of higher order goals (Logan, Schachar, & Tannock, 1997).

Addictive behaviors can be modelled experimentally by measuring attentional biases towards salient alcohol cues and deficits in inhibitory control (Field & Cox, 2008; Goldstein & Volkow, 2011). Studies with clinical (Field, Mogg, Mann, Bennett, & Bradley, 2013) and non-clinical populations (Jones & Field, 2015; Wilcockson & Pothos, 1975) highlight the significance of such methods as a predictor of alcohol consumption (Nikolaou, Field, & Duka, 2013). In alcohol visual search tasks (Alcohol VST), alcohol cues can trigger attentional responses over neutral control cues in social (Pennington, Qureshi, Monk, & Heim, 2019) and heavy drinkers (Pennington et al., 2020); and scores in the Alcohol VST also positively correlate with levels of alcohol consumption. As opposed to other measures of attentional bias, the Alcohol VST allows observing attentional prioritization of alcohol cues within the environment, more closely matching naturalistic settings (Becker, Anderson, Mortensen, Neufeld, & Neel, 2011; Pennington et al., 2020). In Alcohol Stop Signal Tasks (Alcohol SST) (Jones et al., 2018) alcohol cues are presented as task-irrelevant distractors during Go trials to study their effect on inhibitory control processes. In a clinical sample it was found that, after an alcohol exposure procedure, alcohol cues can generate decreased Go Reaction Times (Go RT) and higher rate of Stop Signal Errors (SSE) (Kreusch, Billieux, & Quertemont, 2017). Alcohol cues can also increase SSRT, partially explaining how the interference of alcohol cues on inhibitory control participates in alcohol consumption (Field & Jones, 2017). The implication is that alcohol cues can act as highly arousing distractors that automatically capture attention and reduce the resources available to effectively exert inhibitory control (Verbruggen & De Houwer, 2007).

Neuroimaging research indicates that the processing of interoceptive signals within the insular cortex can alter executive control and decision making in favour of drug related responses (Naqvi & Bechara, 2009), predicting subsequent relapses (Paulus, 2007). It is thus hypothesized that the interoceptive representation of drug related stimuli can “hijack” cognitive control mechanisms and precipitate drug related behaviors (Gray & Critchley, 2007; Naqvi & Bechara, 2009). There is, however, a lack of research studying this hypothesis from a behavioral perspective. Recent research across dimensions of cardiac interoception shows that high-order interoceptive processes, reflecting the perception of individual heartbeats, is modulated by alcohol administration to

support the perception of subjective alcohol effects and expectancies (Leganes-Fonteneau, Bates, Islam, & Buckman, 2021b; Leganes-Fonteneau, Cheang, Lam, Garfinkel, & Duka, 2019). Low-order interoceptive processes can also be modulated by alcohol administration, and changes in 0.1 Hz heart rate variability can precipitate the development of alcohol cognitive biases (Leganes-Fonteneau et al., 2020) and increase the interference of alcohol stimuli in cognitive control mechanisms (Leganes-Fonteneau, Bates, Vaschillo, & Buckman, 2021). This evidence points towards an interoceptive basis for the development of alcohol attentional biases. Research linking intermediate levels of interoception with the interference of reward-related stimuli in attention and inhibitory control can further clarify the role of interoceptive signals in addictive behaviors.

In this registered report (<https://osf.io/wn96a/>) we recruited a sample of social drinkers to study whether intermediate levels of interoception modulated alcohol attentional biases and inhibitory control mechanisms in the presence of alcohol related stimuli. We used an Alcohol Visual Search Task (Alcohol VST) in which stimulus presentation was synchronized with systolic or diastolic cardiac phase. This task allows measuring the role of cardiac signals in the detection (visual search accuracy–VSA) and identification (stimulus identification accuracy – SIA) of alcohol cues. Across two sessions, we also used two instances of an Alcohol Stop Signal Task in which alcohol or neutral cues appeared as task-irrelevant distractors during the presentation of Go cues. In the first instance of this task, Go cues in Go and Stop trials appeared synchronized with participants' systole or diastole to measure whether cardiac signals modulate the interference of alcohol cues in inhibitory control (Alcohol Cardiac Go task–Alcohol CGT). In a different instance of the task, Stop signals appeared synchronized with participants' cardiac phase to measure whether cardiac signals can inhibit the interference of alcohol cues on inhibitory control (Alcohol Cardiac Stop task–Alcohol CST).

2. Hypotheses

We pre-registered three hypotheses related to the effect of cardiac signals on the Alcohol VST, the Alcohol CGT and the Alcohol CST (see [Design Table in supplementary materials](#)). Initial manipulation checks examined preferential attentional responses towards alcohol cues in the Alcohol VST, expecting increased VSA and SIA in the detection of alcohol targets compared to neutral targets. We also checked in the Alcohol CGT that the presence of alcohol cues interfered with response inhibition (longer SSRT, shorter SSD), regardless of cardiac synchrony (Jones et al., 2018). A failure to replicate previous findings implied that the tasks used to examine attentional biases toward alcohol cues were not sensitive. Manipulation checks also examined the effect of cardiac signals on inhibitory control regardless of the task-irrelevant distractors in the Alcohol CST (Rae et al., 2018). Specifically, we checked whether response inhibition was improved at systole (faster SSRT, longer SSD) compared to diastole, regardless of alcohol stimulus presence. A failure to replicate previous findings (Rae et al., 2018), would have

implied that the main effect of cardiac signals on inhibitory control was less robust than expected, or that the specific population recruited for this experiment showed, overall, an inability to integrate cardiac signals in the processing of inhibitory control. Non-significant manipulation checks, albeit unlikely, would undermine the validity of main hypotheses. However, interaction effects (i.e. counter-effects due to cardiac synchrony) could be responsible for this failure. Such limitations will be acknowledged in the discussion and examined via exploratory analyses.

Hypothesis 1. In the Alcohol VST, presenting alcohol stimuli at systole increases attentional bias over neutral stimuli.

This hypothesis is based on prior evidence for increased attentional salience of alcohol cues in an Alcohol VST (Heitmann, Jonker, & de Jong, 2021; Pennington et al., 2019), and on previous observation that the processing of emotionally salient stimuli in a visual search task can be modulated by cardiac signals (Leganes-Fonteneau et al., 2020). To test this, we computed VSA and SIA for alcohol and neutral cues and computed difference scores (systole–diastole) using planned comparisons. We expected the effect of cardiac signals to be significantly higher for alcohol than neutral cues. This would support a role for cardiac signals in the development of alcohol attentional biases. A non-significant result would imply lack of evidence for an impact of cardiac signals in alcohol attentional biases.

Hypothesis 2. In the Alcohol CGT, presenting Go cues at systole increases alcohol attentional bias and the interference of alcohol cues in inhibitory control.

In the Alcohol CGT, Go cues were always presented at systole or diastole. Decreased Go RT implies higher attentional bias. Increased SSRT, and SSE, as well as lower SSD imply disrupted inhibitory control.

This hypothesis is based on prior evidence for the interference of alcohol cues on inhibitory control (Jones et al., 2018) and that the processing of salient stimuli can be modulated by cardiac signals (Leganes-Fonteneau et al., 2020). To test this, we computed Go RT for Go trials, and SSRT, SSE and SSD for Stop trials separately for alcohol and neutral distractors in systole and diastole trials. Because we expected alcohol cues to generate, overall, higher attentional biases and interference in inhibitory control than neutral cues, we computed difference scores (systole–diastole) for alcohol and neutral trials using planned comparisons. For SSRT, Go RT, and SSE, we expected difference scores for alcohol cues to be higher than for neutral cues. For SSD, we expected difference scores for alcohol cues to be smaller than for neutral cues. This would imply that systolic signals selectively increase the interference of alcohol cues on inhibitory control and attention relative to diastole. A non-significant result would imply lack of evidence for the support of cardiac signals in the interference of alcohol cues on inhibitory control.

Hypothesis 3. In the Alcohol CST, presenting Stop signals at systole reduces the interference of alcohol cues in inhibitory control.

In the Alcohol CST, Stop signals were always presented at systole or diastole. This hypothesis is based on prior evidence for the interference of alcohol cues on inhibitory control (Jones et al., 2018) and for the ability of cardiac signals to improve inhibitory control in the SST (Rae et al., 2018). To test this, we computed SSRT, SSE and SSD in the presence of alcohol and neutral cues for trials in which the Stop signal was presented at systole vs. diastole. Because we expected alcohol cues to generate, overall, a higher interference in inhibitory control than neutral cues, but that response inhibition would be improved at systole, we computed difference scores (alcohol – neutral) for systole and diastole trials using planned comparisons. For SSRT and SSE, we expected difference scores at systole to be smaller than at diastole. For SSD, we expected difference scores at systole to be higher than at diastole. This would imply that, in the presence of alcohol cues, systolic signals can improve inhibitory control relative to diastole, counteracting the deleterious effect of alcohol cues. A non-significant result would imply lack of evidence for the ability of cardiac signals to improve inhibitory control in the presence of alcohol cues.

Exploratory Hypothesis 4. Individual differences in alcohol use correlate with the cardiac amplification of attentional biases and effects on inhibitory control.

Alcohol use scores, binge drinking scores, and AUDIT scores positively correlate with the cardiac amplification of attentional biases in the Alcohol VST and interference in inhibitory control in the Alcohol CGT; and negatively with the reduction of interference in inhibitory control in the Alcohol CST.

3. Methods

Experimental codes are available here <https://osf.io/kr3jg/>

3.1. Ethics

The Ethics Committee of the Psychological Sciences Research Institute at UC Louvain approved this design. Note that there was a change of institution following in principle acceptance of Stage 1. Participants read and signed an informed consent form before the start of the experiment and were compensated 10 Euros/hour for their participation.

3.2. Alcohol visual search task

In the Alcohol VST, participants sat ~60 cm away from the 15" screen and were presented with a visual array consisting of 6 stimuli organized in a circular pattern around the center of the screen (stimulus array, stimulus size = 2.42×2.42 cm, distance from center of the screen = 2.85 cm, screen resolution 1920×1080 px). For each trial, one target odd-one-out stimulus was presented, surrounded by 5 distractors. On half of the trials, the target was an alcohol cue, and distractors were neutral cues, and vice-versa. Alcohol cues consisted of pictures of alcohol beverages (i.e. bottles of liquor, cans of beer) with their content served in a glass. Pictures were taken

against a white background and were presented in black and white. Stimuli were previously used in a modified Flanker task (Nikolaou et al., 2013), although some images were replaced by alcohol brands more common in the Belgian market, using a set of images previously curated by the laboratory (Bollen et al., 2024). Neutral cues presented stationary items with a similar visual content, as previously employed in similar experimental paradigms (Leganes-Fonteneau, Bates, Vaschillo, & Buckman, 2021). In total, participants completed 160 trials (40 per for each cue type and cardiac condition).

Each trial started with the presentation of a red fixation point that remained on screen until the presentation of the stimulus array. The stimulus array remained on screen for 100 ms, followed by a black screen for 20 ms. Next, participants were presented with the visual search response array, in which 6 white squares were presented at the location of the stimuli. Finally, participants saw the stimulus identification response array, which consisted of 2 words in a row (Alcohol, Neutral). Both response arrays remained on the screen until a response was detected. The mouse cursor was reset to the center of the screen at the beginning of each trial. On 50% of the trials, the stimulus array appeared in synchrony with the participant's systole or diastole, in a fully randomized manner.

Participants' were instructed to detect the odd-one-out stimulus by selecting the square corresponding to the location of the target stimulus as quickly and accurately as possible using the computer mouse. The proportion of correct responses was used as an index of visual search accuracy (VSA). In the stimulus identification response array, participants selected which cue type (Alcohol vs. Neutral) they think was presented as an odd-one-out target stimulus using the mouse (stimulus identification accuracy, SIA). Prior to the main task, participants observed two example trials in which the stimulus array remained on screen for 500 ms, and completed a practice block in which 10 trials were presented (5 per cue type), non-synchronized with participants' cardiac phase, see Fig. 1.

3.3. Alcohol stop signal task

In the Alcohol SST participants completed two instances of a modified SST in which an alcohol or neutral cue was presented as task-irrelevant distractor in each trial. For each trial, the stimulus array consisted of a distractor (alcohol or neutral cue, 6.40×6.40 cm) surrounded by the Go cue, a colored frame (blue or yellow, $.173 \times 0.173$ cm). Participants' task was to press one of two buttons on the keyboard depending on the color of the Go cue using the index and middle fingers of their dominant hand, and to ignore the distracting stimulus cue. On a minority of trials (25%), after a variable stop signal delay (SSD), the colored frame was replaced by a red frame, and participants heard a tone (1 kHz, 100 ms duration), indicating that they should withhold their response on the trial. For Go trials, the stimulus array remained on screen for 1000 ms or until response. For Stop trials, the stimulus array remained on screen upon presentation of the stop signal, for a maximum duration of 1000 ms if participant successfully inhibits their response, or disappeared upon incorrect Go response. Inter-trial interval depended on the occurrence of the next R-wave, with a minimum of 200 ms.

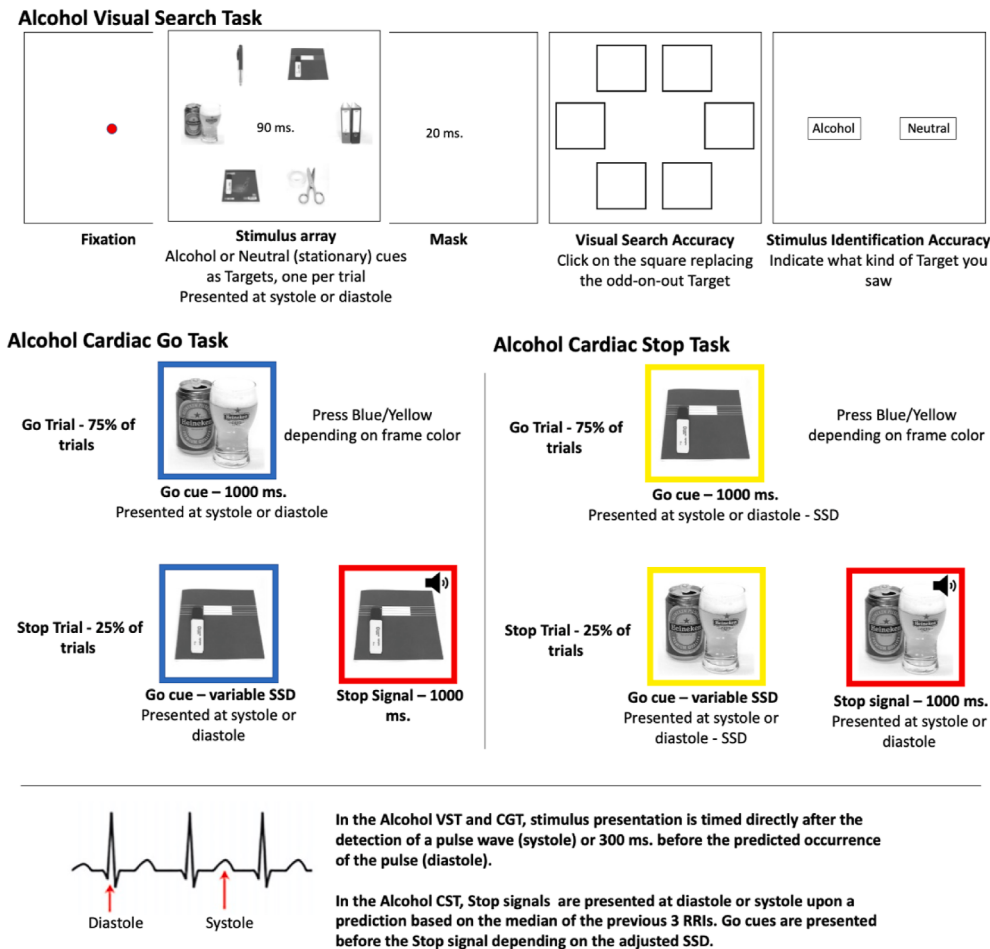


Fig. 1 – Experiment tasks – In the Alcohol VST, participants have to detect and identify an odd-one-out Target (alcohol or neutral cue) presented at systole or diastole (**Hypothesis 1**). In the Alcohol CGT, Go cues (blue or yellow frame) are presented at systole or diastole, together with a task irrelevant distractor (alcohol or neutral cue). A Stop signal replaces the Go cue on 25% of the trials (**Hypothesis 2**). In the Alcohol CST, stimulus presentation is equivalent, but the Stop signal is presented at systole or diastole (**Hypothesis 3**).

We ran two separate instances of the Alcohol SST in each session:

The Alcohol CGT allows studying whether cardiac signals increase the interference of alcohol stimuli on inhibitory control (**Hypothesis 2**). Go cues were always presented synchronized with cardiac signals (50% systole, 50% diastole).

The Alcohol CST allows studying whether cardiac signals improve inhibitory control in the presence of alcohol cues (**Hypothesis 3**). Stop signals were always synchronized with cardiac signals (50% systole, 50% diastole). During Go trials, Go cues were set at the point within the cardiac cycle that they would be delivered on Stop trials, using the values from the SSD staircase trackers, however there was no delivery of a subsequent stop signal. Each task was divided into 4 blocks of 200 trials, and participants were allowed to take a break between blocks to reduce movement during the task and ensure quality of pulse data recording. A practice block of 10 trials with no cardiac synchrony was included at the beginning of each task.

The presence of an alcohol or neutral cue distractor was evenly distributed between all possible trial combinations. To

maintain success at 50%, the SSD was adjusted on each trial by adding or subtracting 50 ms after a correct stop or incorrect response, respectively (initial SSD = 250 ms, minimum SSD = 50 ms). Importantly, in each task, SSD was adjusted independently for the different combinations of Stop or Go trials presented at systole or diastole and for those presenting alcohol or neutral distractors using a 2 (systole vs. diastole) x 2 (alcohol vs. neutral) design. Likewise, SSE was obtained, and SSRT was calculated independently for each of the trial combinations according to the integration method, wherein n go reaction times were rank ordered, and the SSD was subtracted from the go reaction time corresponding to the n *probability of responding on stop trials (Verbruggen et al., 2019).

3.4. Physiological measurements and cardiac synchronization

Participants were connected to a Nonin 8000SM finger pulse-oximeter (Nonin Medical Inc. USA) to collect pulse data at a sampling frequency of 75 Hz. The sensor was placed on the index finger of the non-dominant hand.

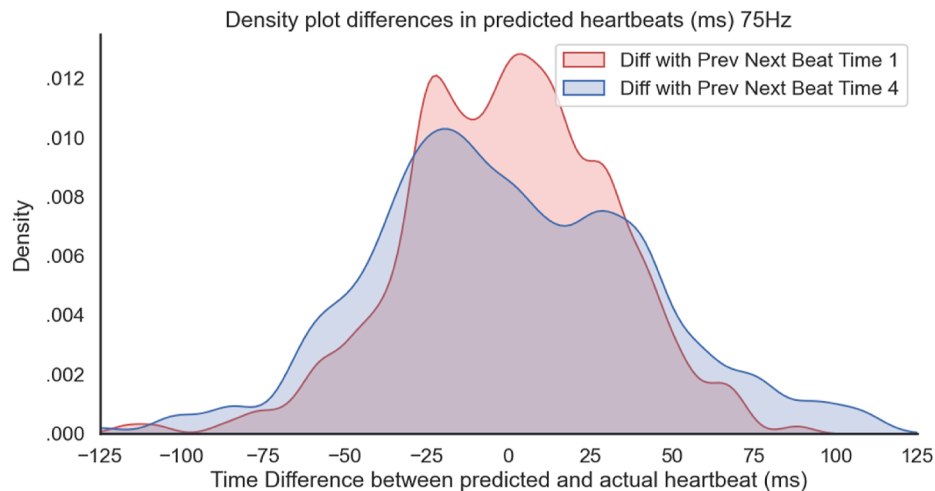


Fig. 2 – Difference between predicted and actual heartbeats – The difference between predicted and actual heartbeats was computed using either the previous PP-interval or the median of the previous 4. Predictions appear to be slightly more reliable using the immediately previous PP-interval to establish a prediction.

Participants' pulse was measured at baseline (t_0) using a Vanilla task (Jennings, Kamarck, Stewart, Eddy, & Johnson, 2007). In this task, participants were comfortably seated and presented with colored squares for 5 min. Each colored square remained on screen for 10 s and participants' task was to silently count how many blue squares they saw. Participants' responses on the Vanilla task were irrelevant but used to avoid mind-wandering. Pulse data were also collected during the Alcohol VST and SST. All pulse data were only used for future secondary data analyses in the context of a broader experimental procedure. Any future use of these data will acknowledge it was collected in the context of this registered report.

In the Alcohol VST, stimulus synchronization with cardiac phase was performed using the Systole package for Python. A trigger signal was automatically sent via UDP protocol into Matlab 2023b whenever a pulse wave was detected, corresponding to the systolic phase. For trials presented at systole, stimulus presentation was immediate upon the detection of the UDP trigger. For trials presented at diastole, stimuli were presented at the predicted occurrence of the next diastole, introducing a waiting period based on the previous PP-interval minus 300 ms. In order to decrease the length of inter-trial-intervals and to prevent biases (i.e. longer ITI for diastole trials) information about the predicted diastole time was available via UDP for 700 ms after the occurrence of the previous pulse, or until a new pulse was detected.

In the Alcohol CGT, for Go trials presented at systole or diastole, stimulus synchronization was analogous to the one used for the Alcohol VST. In the Alcohol CST, for Stop trials in which the Stop signal was presented at systole or diastole, the inter-beat interval was dynamically monitored in Python to predict the occurrence of the next systole or diastole (300 ms before the predicted systole event). This allowed presentation of Stop signals in synchrony with the cardiac events. Go cue appeared prior to the predicted presentation time, accounting for the current SSD (systole-SSD or diastole-SSD), whether or not they were followed by a Stop signal. The method consisting of predicting heartbeats has proven reliable in the past

(Rae et al., 2018), and a post-hoc quality analysis was conducted to verify the precision of cardiac timing procedures. This strategy consisted in a deviation of the previously registered protocol,¹ and although valid, generated a decrease in the accuracy of cardiac synchronization. This decrease in accuracy was due mostly to the lower sampling rate (only 75 Hz with the finger pulse-oximeter) and marginally to the fact that diastole now needed to be predicted instead of being directly measured at the onset of the R-wave. For the rest, both methods require equivalent predictive methods for the Alcohol CST. Upon examination of the reliability of the heartbeat prediction, we found that basing the predictions solely upon the previous PP-interval generated a smaller difference than utilizing, as pre-registered, the median of the 4 previous PP-intervals, see Fig. 2 as an example following a 300 s measurement. To counteract any possible increase in noise in the data we increased the target sample size to 45 participants.

3.5. Questionnaires

Participants completed a basic demographic questionnaire including age, sex and illicit drug use. Participants also completed the Alcohol Use Questionnaire (AUQ) (Mehrabian & Russell, 1978) adapted for use in a French-speaking population using well-validated measures existing in the laboratory. This 13-item questionnaire examines alcohol consumption quantified in alcohol units per week. A binge drinking score was extracted (Townshend & Duka, 2002) accounting for number of drinks consumed per hour, percentage of times participants reach drunkenness after consuming alcohol and number of times drunk in the last 6 months. We also measured alcohol consumption through the alcohol use disorder identification test (AUDIT), a 10-item questionnaire that assesses hazardous

¹ This change was agreed upon with the editorial team and underwent additional peer-review before data collection commenced.

Table 1 – Descriptive statistics for alcohol use measures.

Measure	M	SD
AUDIT	15.57	6.90
AUQ	46.45	39.25
Binge score	30.12	24.95

alcohol consumption (Saunders, Aasland, Babor, De La Fuente, & Grant, 1993).

3.6. Participants

A sample of 50 social drinkers, mean age = 21.7, SD = 2.38, 27 females (see Sampling plan, targeting $n = 45$ instead of 35 to make up for deviations in cardiac synchronization) was recruited using fliers posted on social media or near UC Louvain, Belgium. Participants were included if they consumed more than 4 drinks per week over the last 6 months. Exclusion criteria were reporting a history of learning disability, psychiatric disorders, treatment for substance use disorder, or regular (weekly) illicit or prescription drug use. Patterns of alcohol consumption are presented on Table 1:

3.7. Procedure

Upon successful screening using an online Qualtrics form, participants were invited for two laboratory sessions. In the first session, they completed demographic questions, the AUDIT questionnaire and the full version of the AUQ. Participants then sat in front of the computer screen and were connected to the finger-pulse oximeter. Participants first completed the baseline Vanilla task. Note that beat-to-beat blood pressure was supposed to be collected at this stage, but was not as the equipment was not available. This does not interfere with the hypotheses of this registered report. Then, participants completed the Alcohol VST and one of two iterations of the Alcohol SST (Alcohol CGT or CST, counter-balanced), in that order to avoid priming effects in the Alcohol VST. Pulse data were collected during these tasks, although data were not presented in this report. In the second session, participants completed a baseline Vanilla task, the other iteration of the Alcohol SST.

3.8. Data quality checks

For the Alcohol VST, participants' data were excluded from further analysis if their mean VSA was below 1/6 (chance level).

For each instance of the Alcohol SST, participants' data were excluded from further analysis on the basis of the following exclusion criteria, following guidance in Verbruggen et al. (2019):

- 1) Probability of responding to the stop signal of $<.25$, or $>.75$
- 2) Probability of Go omissions $>.1$
- 3) Longer reaction time for unsuccessful Stop trials than Go trials

Within the limited capabilities of the laboratory in terms of recruitment and data collection, we recruited a sample 43% larger than the one established in the original sampling plan.

3.9. Data analysis plan

Data analysis files and full data set are available on <https://osf.io/kr3jg/>. Considering the specificity of our hypotheses, planned comparisons were conducted for all analyses using difference scores (Keppel, 1991; Ruxton & Beauchamp, 2008). For the Alcohol VST, data were transformed using arcsine square root. Difference scores (systole – diastole) were computed for VSA and SIA for alcohol and neutral cues separately, and compared using a paired-samples t-test (Hypothesis 1). Accuracy scores on VSA and SIA were not conditional on each other. Manipulation checks for VSA and SIA consisted of paired samples t-tests comparing performance for alcohol and neutral cues, collapsing for cardiac synchrony.

For the Alcohol CGT, wherein Go cues were presented in synchrony with cardiac phase, we considered Go RT for Go trials, and SSRT, SSE and SSD for Stop trials. Difference scores (systole – diastole) were computed for alcohol and neutral cues separately and compared using paired-samples t-tests (Hypothesis 2). Manipulation checks for Alcohol CGT consisted of paired samples t-tests comparing SSRT and SSD for alcohol and neutral cues, collapsing for cardiac synchrony.

For the Alcohol CST, wherein Stop signals were presented in synchrony with cardiac phase, we considered SSRT, SSE and SSD. Difference scores (alcohol – neutral) were computed for systole and diastole trials separately and compared using paired-samples t-tests (Hypothesis 3). Manipulation checks for Alcohol CST consisted of paired samples t-tests comparing SSRT and SSD for systole and diastole trials, collapsing for stimulus presented.

For exploratory analyses on the relationship between alcohol use and task performance, correlational analyses were performed between AUDIT score, alcohol use score and binge drinking score, and the difference scores computed for VSA, SIA in the Alcohol VST, Go RT in the Alcohol CGT and SSRT, SSE and SSD in the Alcohol CGT and CST. Corrections for multiple comparisons were performed separately for scores obtained in each task. Descriptive statistics on questionnaire scores were used to characterize the sample of social drinkers.

Data analyses were conducted using R. Alongside frequentist hypothesis testing, exploratory Bayes Factors with default priors (BF_{10}) examined sensitivity of results.

3.10. Pilot data and sampling plan

We conducted a frequentist power analysis based on our main hypotheses relying on observing significant differences. For the Alcohol VST, previous results using complex emotional faces show that the task is sensitive to the effects of cardiac signals (Leganes-Fonteneau et al., 2020), finding a significant interaction between emotional face and cardiac synchrony for VSA, $F(3,490) = 11.880$, $p < .001$, $\eta^2 = .068$ and SIA, $F(3,490) = 6.090$, $p < .001$, $\eta^2 = .036$. A power analysis was performed simulating values according to published data, https://osf.io/s2ew9/?view_only=b0360195d2d3421a9623a3d9684492c9. As opposed to the original experiment in which participants only completed 25 trials per condition (4 emotional faces), here participants completed 40 trials per condition, and SDs were correspondingly adjusted by

Table 2 – Exemplary data used for the power analysis for the Alcohol Visual Search Task. SDs were modulated according to the increase in trial number per condition. Simulated data show that Visual Search Accuracy and Stimulus Identification Accuracy would be higher for alcohol cues presented at systole than diastole. Accuracy scores were arcsine square root transformed. For the Alcohol Cardiac Go Task (H2) and Cardiac Stop task (H3), data was simulated based on previous research. In the case of H3 for the Alcohol CST, we contemplate correction for multiple comparisons by computing the smallest possible critical alpha at which power = .90 would be achieved with $n = 35$, and present Benjamini-Hochberg Adjusted P values following False Discovery Rate. For the Alcohol CGT, critical alphas become negligible with $n = 35$.

H1- VSA	Systole		Diastole		n	Power		
	Mean	SD	Mean	SD				
Alcohol	1.809	.235	1.670	.213	32	.92		
Neutral	1.233	.167	1.199	.159				
H1- SIA								
Alcohol	1.668	.297	1.451	.232	32	.98		
Neutral	1.552	.254	1.492	.238				
H2 - SSRT								
Alcohol	249.6	44	234.2	44	13	.92		
Neutral	223.2	44	221	44				
H2 - SSE								
Alcohol	29.35	17	22	16	21	.91		
Neutral	27.8	24	27	21				
H2- Go RT								
Alcohol	873.9		834	116	13	.93		
Neutral	845.8		840	114				
H2 – SSD								
Alcohol	169.70	95.22	198.26	95.22	24	.91		
Neutral	217.31	95.22	226.83	95.22				
H3 - SSRT							Critical	Adjusted
Alcohol	206.2	64	234.2	44	35	.90	.02	.02
Neutral	203	64	221	44				
H3 - SSE								
Alcohol	19.45	24	33	24	21	.91	8e-04	.0024
Neutral	18.6	17	27	17				
H3 - SSD								
Alcohol	235.54	106.49	198.26	95.22	23	.903	.0016	.0024
Neutral	243.22	106.49	226.83	95.22			Italic: non-simulated values	

accounting for changes in the sampling distribution of the sample mean, see Table 2. Results of the power analysis computed on pwr2ppl for R show that, with 40 trials per condition for alpha = .02, 32 participants would generate a power = .92 for VSA. For SIA, 32 participants would generate power = .98 (see Table 2).

For the Alcohol Stop Signal Tasks, previous results allow simulation of values for the Alcohol CST and SST, showing that performance in Stop trials can be improved by the presentation of Stop signals at systole (Rae et al., 2018). In Rae et al. (2018), SSRT was lower, $t(45) = -2.49$, $p = .016$, Cohen's $d = 0.037$, at systole compared to diastole. Results from a meta-analysis show that alcohol cues have a small effect on inhibitory control (Jones et al., 2018), and values for SSE are input from (Kreusch et al., 2017). Means and SDs for SSRT, SSD, Go RT and SSE were simulated for all independent variable combinations, accounting for a small effect of alcohol cues on inhibitory control ($d = .3$), and further corrected according to the small effect of cardiac signals on inhibitory control ($d = .37$), see Table 1. Results of the power analysis show that, for alpha = .02 and target power = .90, the highest sample size would be necessary for SSRT in Hypothesis 3, $n = 35$. All other required sample sizes would be smaller. In order to correct for multiple comparisons for H3, accounting for variable-specific sensitivities, we computed the smallest possible alpha level compatible with power = .90 and $n = 35$.

These critical alpha values were adjusted using False Discovery Rate to show that, with $n = 35$, results would a-priori survive correction for multiple comparisons (see Table 1). For H2, the critical alphas generated with $n = 35$ yield negligible values.

Regarding manipulation checks, power analysis was computed collapsing mean values for each main effect and accounting for changes in SDs due to doubling the number of trials. For VSA and SIA, $n = 35$ would generate power = 1 and .906 respectively in the detection of a main effect of alcohol vs. neutral cues. For SSRT and SSD in the Alcohol CGT, $n = 35$ would generate power = .96 and .965 respectively in the detection of a main effect of alcohol vs. neutral cues on inhibitory control. For SSRT and SSD in the Alcohol CST, $n = 35$ would generate power = .99 and .92 respectively in the detection of a main effect of systole vs. diastole on inhibitory control. Hypotheses, analysis plan and outcome interpretation are presented in Table 2.

4. Results

4.1. Alcohol VST

Two participants were excluded from all analyses due to being color blind, resulting in 48 participants. Additionally, further

exclusions were applied based on task performance. For VSA analysis, one additional participant was excluded for having a VSA below chance (1/6), leaving a final sample of 47 participants.

The manipulation check for VSA revealed a significant difference between alcohol and neutral cues, collapsing for cardiac synchrony, $t(46) = -9.00$, $p < .001$, $BF_{10} = 2.37 \times 10^8$, indicating extreme evidence in favour of the alternative hypothesis. For SIA, the paired-samples t -test indicated a significant difference between alcohol and neutral cues, with SIA being lower for alcohol cues than neutral cues, collapsing for cardiac synchrony, $t(47) = -3.59$, $p < .001$, $BF_{10} = 8.91$, indicating moderate evidence in favour of the alternative hypothesis. Contrary to our hypothesis, VSA and SIA scores were significantly lower for alcohol cues compared to neutral cues. We believe that this might reveal a failure of the task in measuring alcohol attentional biases. Neutral stimuli, being more perceptually varied, stood out and were detected more quickly among standardized alcohol-related objects, whereas alcohol-related stimuli were more difficult to identify among diverse neutral objects.

For the planned t -tests, results showed no effect in VSA difference scores (systole – diastole) between alcohol and neutral, $t(46) = 1.39$, $p = 0.172$, $BF_{10} = .39$. Similarly, there was no effect in SIA difference scores between alcohol and neutral cues, $t(47) = .73$, $p = 0.470$, $BF_{10} = .21$ (see Fig. 3).

4.2. Alcohol CGT

Four participants were excluded from the analysis due to experimental error. Additionally, one participant was excluded based on percentage of Go omissions $>10\%$ (see Data Quality Checks in Methods above). These exclusions resulted in a final sample of 43. Full descriptives for CGT and CST task performance are presented on Table 3 (Verbruggen et al., 2019).

The manipulation checks revealed no difference between alcohol and neutral cues, for SSRT, $t(42) = -.52$, $p = 0.608$, $BF_{10} = .19$, or SSD, $t(42) = .35$, $p = .728$, $BF_{10} = .17$. This implies that alcohol cues did not interfere with response inhibition relative to neutral cues, when collapsing across cardiac

synchrony. A non-planned manipulation check examined the effect of cardiac synchrony, collapsing for stimulus. Results revealed some evidence for a significant difference between systole and diastole on SSD, $t(42) = 2.31$, $p = .026$, $BF_{10} = 1.76$, with SSD being higher for systole compared to diastole. For all other variables, results were sensitively null, $BF_{10} < 1/3$.

For the planned t -tests, results showed evidence towards a lack of effect in SSRT difference scores between alcohol and neutral cues, $t(42) = -1.29$, $p = .203$, $BF_{10} = .36$. Similarly, there was evidence for no effect in SSE difference scores, $t(42) = -.96$, $p = .341$, $BF_{10} = .25$, and tentatively for no effect in SSD difference scores, $t(42) = 1.54$, $p = .131$, $BF_{10} = .49$. Additionally, no effect was found for Go reaction time difference scores between alcohol and neutral cues, $t(42) = .75$, $p = .457$, $BF_{10} = .22$. This implies a lack of evidence for the support of cardiac signals in the interference of alcohol cues on inhibitory control (see Fig. 4), although results need to consider the lack of main effect of alcohol cues.

4.3. Alcohol CST

Six participants were excluded from the analysis due to experimental error. Data quality checks based on performance criteria (see Data Quality Checks in Methods above) did not reveal any violations. These exclusions resulted in a final sample of 42. Full descriptives for task performance are presented on Table 3 (Verbruggen et al., 2019).

The manipulation check revealed a significant difference between systole and diastole trials for SSRT, $t(41) = -3.14$, $p = .003$, $BF_{10} = 11.06$, suggesting strong evidence in favour of the alternative hypothesis, with SSRT being significantly lower during systole than diastole. Similarly, there was a significant difference for SSD, $t(41) = 4.86$, $p < .001$, $BF_{10} = 1193.67$, suggesting extreme evidence in favour of the alternative hypothesis, with SSD being higher during systole compared to diastole (see Fig. 5). The implication is that there is an effect of cardiac signals on inhibitory control, regardless of the distractor cue.

A non-planned manipulation check examined the effect of stimulus (alcohol vs. neutral) on SSRT and SSD, collapsing by

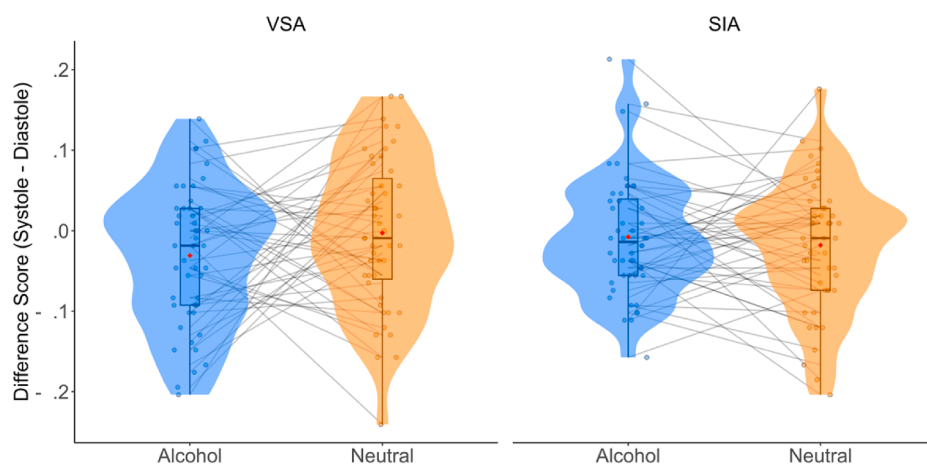


Fig. 3 – Raincloud plots revealing no effect of cue on difference scores (systole – diastole) for visual search accuracy and stimulus identification accuracy in the Alcohol Visual Search Task.

Table 3 – Descriptive task performance data for the Cardiac Go and Stop tasks.

Stimulus	Synchrony	Probability Go Omission		Probability Choice Error		RT Go Trials		Stop Signal Delay		Stop Signal RT		RT Stop Trials	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Cardiac Go Task													
Neutral	Diastole	.0340	.1005	5.37	4.05	460.53	91.38	303.42	138.50	161.25	77.24	417.98	86.65
Neutral	Systole	.0343	.0900	5.67	4.71	460.80	85.80	306.62	138.49	161.91	70.67	417.22	94.86
Alcohol	Diastole	.0295	.0894	5.23	4.55	462.20	88.17	301.32	132.87	163.49	83.15	416.81	85.52
Alcohol	Systole	.0343	.0958	5.23	3.87	460.94	85.57	310.90	137.78	158.61	76.89	415.38	90.81
Cardiac Stop Task													
Neutral	Diastole	.0206	.0316	5.43	4.69	458.17	72.00	295.41	108.25	162.16	49.86	409.26	80.05
Neutral	Systole	.0183	.0300	5.41	4.78	468.02	74.13	313.72	111.46	148.79	51.56	422.55	87.57
Alcohol	Diastole	.0203	.0336	5.51	5.71	460.49	71.31	302.87	115.85	154.85	54.47	407.53	82.39
Alcohol	Systole	.0164	.0271	5.84	5.34	470.33	75.30	314.02	105.85	148.14	47.31	415.04	74.41

synchrony. Results were sensitively null for both variables, with $BF_{10} < .17$.

For the planned t-tests, one participant was removed as an outlier based on graphical observation, as being more than 3 standard deviations from the mean across both conditions. Results showed no effect of cardiac synchrony in SSRT difference scores (alcohol – neutral), $t(40) = .83$, $p = .411$, $BF_{10} = .23$, nor in the mean SSE difference scores, $t(40) = -.12$, $p = .907$, $BF_{10} = .17$ or SSD difference scores, $t(40) = -.99$, $p = .330$, $BF_{10} = .27$. This implies a lack of evidence for the ability of cardiac signals to improve inhibitory control in the presence of alcohol cues, although results need to consider the lack of main effect of alcohol cues (see Fig. 6).

4.4. Exploratory analyses

Exploratory correlational analyses examined the relationship between AUDIT score, alcohol use score and binge drinking score, and the difference scores computed for VSA, SIA in the Alcohol VST, Go RT in the Alcohol CGT and SSRT, SSE and SSD in the Alcohol CGT and CST. The results across the Alcohol VST, CGT, and CST tasks were not sensitive for or against the null hypothesis: across all variables, correlations were weak, with P values $> .5$ and $1/3 < BF_{10} < 1$.

5. Discussion

This registered report aimed to investigate whether cardiac signals modulate attentional biases towards alcohol cues and their interference on inhibitory control in social drinkers. Using multiple tasks (Alcohol VST, CGT, and CST), we explored how reward cues interact with cardiac signals to impact attention and control inhibition. As outlined in the manuscript, we made one deviation from our pre-registered protocol, which was peer-reviewed before data collection. Due to a change in laboratory, we used pulse oximetry instead of ECG for the cardiac synchrony procedure, a modification pre-registered and reviewed before data collection. To ensure robustness, we recruited a larger sample size, and results from the CST support the validity of this approach.

The first hypothesis was that in the Alcohol VST, presenting alcohol stimuli at systole will increase attentional biases over neutral stimuli. Manipulation checks revealed increased accuracy in the detection and identification of neutral cues when

presented as odd-one out target stimuli compared to alcohol cues. Although the mechanisms underlying this unexpected effect are difficult to establish, we interpret this as a failure of the VST in measuring alcohol attentional biases, perhaps because neutral stimuli, composed of perceptually varied stationary items, were more easily detected among standardized alcohol-related objects. Following the failure of this manipulation check, it is unsurprising to observe a lack of effect of cardiac synchrony in the detection and identification of targets. We are therefore unable to determine whether cardiac signals impact alcohol attentional biases, since the task was not suitable to measuring these effects in the first place.

The second hypothesis proposed that presenting Go cues at systole in the Alcohol CGT would heighten attentional bias towards alcohol cues and amplify their interference with inhibitory control. Contrary to these expectations, results revealed no effect of cardiac signals on either alcohol-related attentional biases or their interference with inhibitory processes. These null findings must be interpreted in the context of unsuccessful manipulation checks, which again failed to demonstrate differences in SSRT or SSD between alcohol and neutral cues, irrespective of cardiac synchrony. This lack of validation contrasts with previous studies (see Jones et al., 2018), which reported that alcohol cues can significantly impair inhibitory control. Importantly, most tasks previously employed SST paradigms in which responding to the cue was an integral part of the task. In this experiment, however, the alcohol and neutral cues were task-irrelevant, requiring participants to focus solely on processing the color of the frame. As participants only needed to process the frame to respond, they might have voluntarily avoided processing the stimuli, thereby leading to an absence of attentional interference. Consequently, this paradigm again does not allow us to conclusively determine whether cardiac signals interact with alcohol cue interference in inhibitory control.

The third hypothesis posited that in the Alcohol CST, presenting Stop signals at systole would reduce the interference of alcohol cues on inhibitory control. While manipulation checks revealed a strong main effect of cardiac phase, evidenced by significantly lower SSRT and higher SSD during systole compared to diastole, unplanned manipulation checks show, as in the CGT, no main effect of stimulus type. Consequently, we failed to observe a sensitive effect of cardiac signals on inhibitory control specifically in the presence of alcohol cues, and we remain unable to address the central

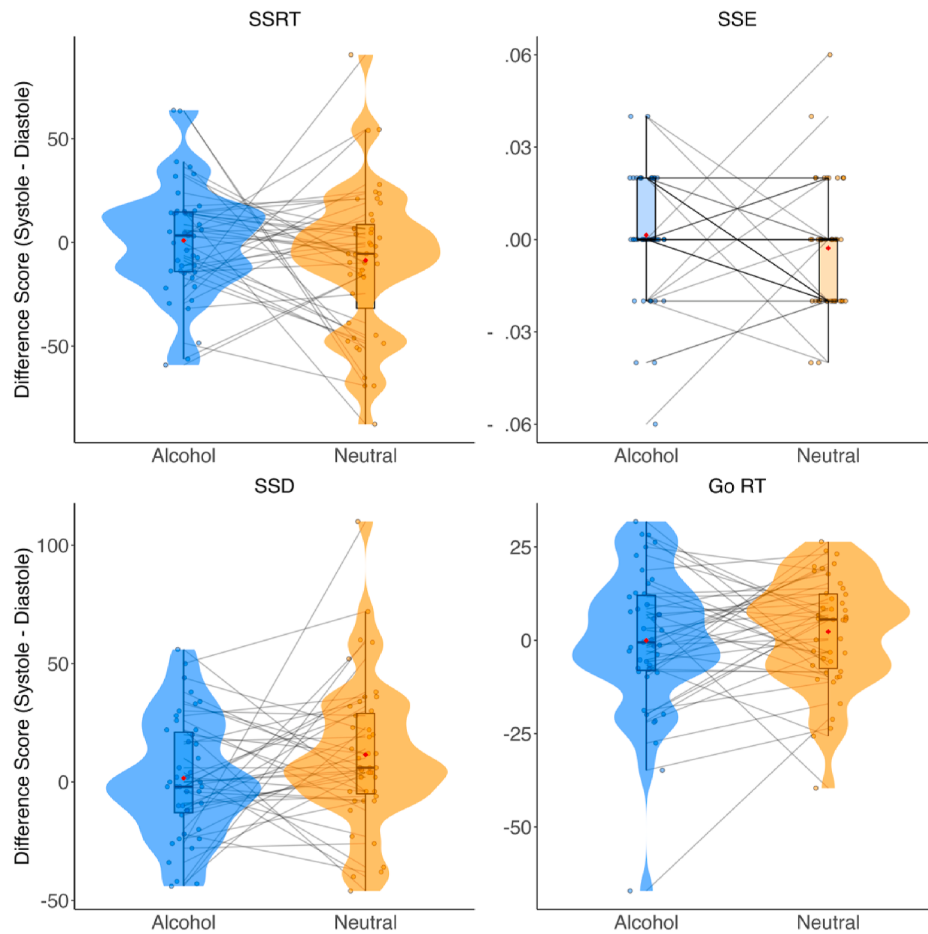


Fig. 4 – Raincloud plots revealing no effect of cue on difference scores (systole – diastole) for stop signal reaction time, stop signal error, stop signal delay, and Go reaction time in the Cardiac Go Task. No violin plot was produced for stop signal error due to its clustered distribution.

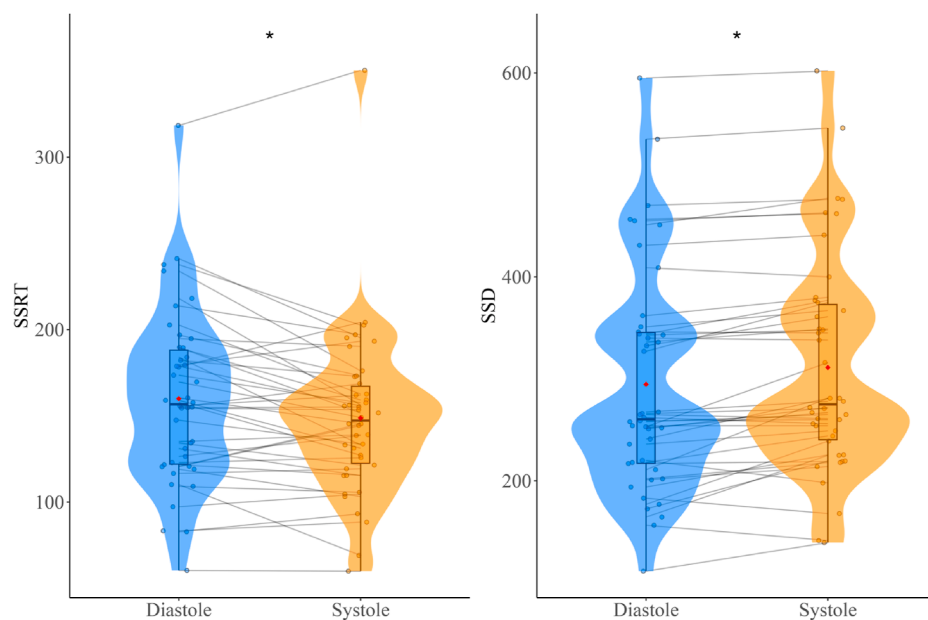


Fig. 5 – Raincloud plots of stop signal reaction time and stop signal delay between systole and diastole, collapsing for stimulus. * For both measures we found strong evidence for an effect of cardiac synchrony in the Cardiac Stop Task.

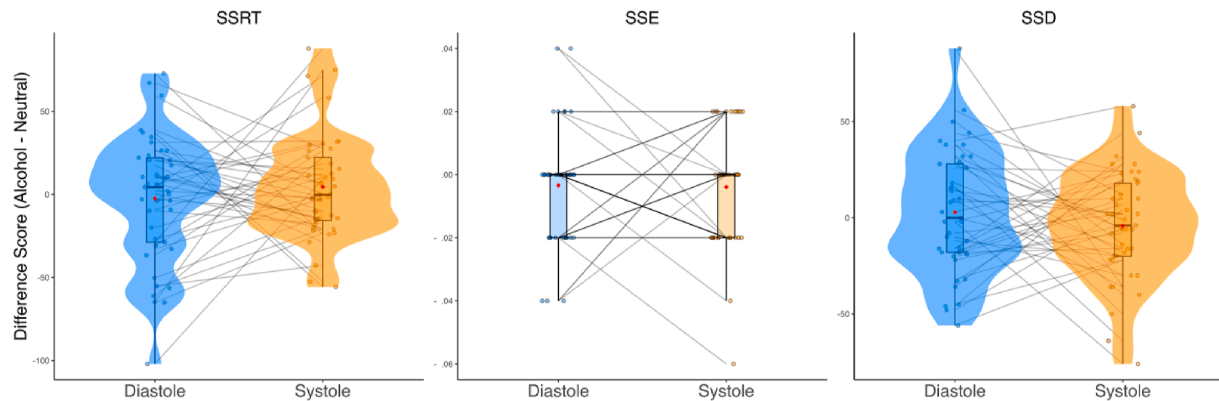


Fig. 6 – Raincloud plots revealing no effect of cue on difference scores (alcohol–neutral) for stop signal reaction time, stop signal error and stop signal delay in the Cardiac Stop Task. No violin plot was produced for stop signal error due to its clustered distribution.

question of this registered report: can systolic signals help reduce the interference of alcohol cues on inhibitory control?

The main effect of cardiac phase on motor inhibition is consistent with earlier findings by [Rae et al. \(2018\)](#), which showed enhanced motor inhibition during systole through reduced SSRT and increased SSD. Replicating this effect is significant, given mixed findings in more recent research, such as [Ren, Marshall, Kaiser, and Schütz-Bosbach \(2022\)](#), who reported prolonged SSRTs (instead of shorter) when Stop signals were presented at systole. Our findings reinforce the notion that presenting Stop cues at systole can enhance inhibitory control, at least within the parameters of this version of the SST. This aligns with interoceptive theories of fear processing ([Garfinkel & Critchley, 2015](#)), which propose that during systole, the detection of salient stimuli such as threat indicators is enhanced, along with associated aversive action, such as withdrawal behaviours. During states of high cardiovascular arousal, when the heart is beating hard and fast, and therefore proportionally spending more time in systole, an enhanced ability to rapidly stop actions is evolutionarily advantageous. As the literature studying this specific cardiac cycle effect grows, future meta-analyses will be useful to more conclusively determine the direction and magnitude of effect.

Finally, our exploratory hypothesis aimed to determine whether individual differences in alcohol use would correlate with the cardiac amplification of attentional biases and effects on inhibitory control. However, we found no significant associations between alcohol consumption measures and cardiac effects in any of the tasks. Given the limitations in task design, due to a lack of control of perceptual factors in the alcohol VST and the fact distractors were completely avoided in the SSTs, these null findings are unsurprising.

A key limitation in these studies is that we did not control for participant position relative to the screen, varying visual angles in a way that could have generated inconsistent results, by either changing the capacity to detect distractors in the VST, or the interference of alcohol cues in the SST. Future studies should target this and other limitations present in this experiment. Herein we chose to adapt existing paradigms

based around cardiac synchrony to incorporate the interference of alcohol cues. Other attentional paradigms, such as spatial cueing ([Azevedo et al., 2018](#)) or attentional blink ([Garfinkel et al., 2014](#)) have shown sensitivity to cardiac signals. Notably, those same paradigms were previously used, without cardiac synchrony, to assess alcohol attentional biases yet failed to produce reliable effects (i.e. [Garland, Franken, Sheetz, & Howard, 2012](#); [Tibboel, De Houwer, & Field, 2010](#)). An alternative approach could be to select paradigms that are proven to generate attentional biases or interference of alcohol cues on attention and inhibitory control ([Bollen, Field, Billaux, & Maurage, 2022](#)), and adapt them to use with cardiac synchrony protocols. For example [Jones and Field \(2015\)](#) used a paradigm in which cues were slightly rotated around the vertical axis. Participants' task was to indicate the direction of the rotation, measuring the interference of alcohol cues in motor inhibition. This kind of paradigm could be easily adapted to establish the effect of cardiac signals in stop response.

Participants' state and trait characteristics could also impact results. While the sample represented a subclinical population of alcohol drinkers, with high AUDIT scores revealing high probabilities of harmful alcohol consumption or even dependence, it is unclear whether they would display alcohol attentional biases even if using more sensitive tasks. In this regard, subjective craving can increase attentional biases towards alcohol cues, in both clinical ([Bollen et al., 2024](#)) and subclinical populations ([Bollen, Masson, Salvaggio, D'Hondt, & Maurage, 2020](#)) an effect that can be modulated experimentally (i.e. via stress induction, [Field & Powell, 2007](#)). Considering the interaction between bodily states and the attentional salience of alcohol cues ([Leganes-Fonteneau, Bates, Vaschillo, & Buckman, 2021](#); [Leganes-Fonteneau et al., 2020](#)), and the growing evidence for a bodily dimension to craving ([Billaux et al., 2025a](#)), future studies should at least measure, if not induce ([Billaux, et al., 2025b](#)), craving to examine the interplay between bodily signals and addictive processes.

Despite these limitations, our findings contribute to broader perspectives on how disruptions in higher-order interoceptive processes may impact inhibitory control and

impulsive behaviors (Herman, 2023). Preliminary evidence suggests that lower interoceptive accuracy correlates with greater impulsivity, a relationship that could be exacerbated by states like craving, stress, or intoxication, further driving maladaptive consumption behaviors (Herman, 2023). We propose that paradigms like those used in this study could help explore whether preconscious bodily signals underlie inhibitory control deficits in clinical populations.

The replicated effect of systolic signals on response inhibition underscores the potential of Cardiac Stop Signal Tasks in future clinical studies. Further research should clarify the neural and physiological bases of these effects (Marshall, Ren, Enk, Liu, & Schütz-Bosbach, 2024) and explore how interoceptive processes interact with other cognitive domains relevant in addiction (e.g., working memory and reflective processing; Wiers, Gladwin, Hofmann, Salemink, & Ridderinkhof, 2013, 2021), to inspire novel interventions. Approaches targeting interoception, such as mindfulness, non-invasive neural or vagus nerve stimulation (Herman, 2023), resonance breathing biofeedback (Leganes-Fonteneau, Bates, Muzumdar, et al., 2021) or sonoception (Di Lernia et al., n.d.) could be integrated with inhibitory and attentional retraining tasks to capitalize on bodily fluctuations and improve decision-making. These strategies offer promising avenues for strengthening inhibitory control and promoting healthier behavioral alternatives in addiction contexts, which could be strengthened with the inclusion of more ecologically valid active sensing approaches (Lopez Townshend and Duka, 2002. et al., Under review). In this context, the Registered Report retains its prospective value by allowing us to report experimental failures and the successful replication of cardiac-synchrony effects on motor inhibition, and it will hopefully guide future efforts to optimise paradigms that more effectively induce alcohol-oriented attention.

6. Conclusion

In summary, this registered report failed at providing evidence for or against the effect of cardiac signals on attentional biases or inhibitory control in the presence of alcohol cues because the experimental paradigms were not sensitive to the effects of those cues. However, we obtained strong evidence replicating previous findings that cardiac signals affect participants' capacity for motor inhibition. The robustness of these findings highlights the potential of cardiac-based paradigms as tools for advancing our understanding of the interoceptive foundations of inhibitory control deficits. Future research involving clinical populations and refined methodologies is needed to fully explore the therapeutic potential of leveraging cardiac signals in addressing inhibitory control impairments.

CRedit authorship contribution statement

Mateo Leganes-Fonteneau: Writing – original draft, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Annelise Theis:** Project administration. **Irene Dolfini:** Formal analysis. **Reinout**

W. Wiers: Writing – review & editing. **Maurage Pierre:** Writing – review & editing. **Charlotte L. Rae:** Writing – review & editing, Software, Methodology, Investigation, Data curation, Conceptualization.

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Scientific transparency statement

DATA: All raw and processed data supporting this research are publicly available: <https://osf.io/kr3jg/>

CODE: All analysis code supporting this research is publicly available: <https://osf.io/kr3jg/>

MATERIALS: All study materials supporting this research are publicly available: <https://osf.io/kr3jg/>

DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.

PRE-REGISTRATION: At least part of the study procedures was pre-registered in a time-stamped, institutional registry prior to the research being conducted: <https://osf.io/wn96a/> At least part of the analysis plans was pre-registered in a time-stamped, institutional registry prior to the research being conducted: <https://osf.io/wn96a/> The analyses that were undertaken deviated from the preregistered analysis plans. All such deviations are fully disclosed in the manuscript.

For full details, see the *Scientific Transparency Report* in the supplementary data to the online version of this article.

Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cortex.2025.06.013>.

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