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A dual-process exploration of binge drinking: Evidence through behavioral and electrophysiological findings

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Abstract

The dual-process model, describing addictive disorders as resulting from an imbalance between increased automatic approach behaviors towards the substance and reduced abilities to control these behaviors, constitutes a sound theoretical framework to understand alcohol-use disorders. The present study aimed at exploring this imbalance at behavioral and cerebral levels in binge drinking, a pattern of excessive alcohol consumption frequently observed in youth, by assessing both reflective control abilities and automatic processing of alcohol-related stimuli. For this purpose, 25 binge drinkers and 25 comparison participants performed a Go/No-Go task during electrophysiological recording. Inhibition abilities were investigated during explicit (ie, distinguishing alcoholic versus nonalcoholic drinks) and implicit (ie, distinguishing sparkling versus nonsparkling drinks, independently of their alcohol content) processing of beverage cues. Binge drinkers presented poorer inhibition for the explicit processing of beverage cues, as well as reduced N200 amplitude for the specific processing of alcohol-related stimuli. As a whole, these findings indicated inhibition impairments in binge drinkers, particularly for alcohol cues processing and at the attentional stage of the cognitive stream. In line with the dual-process model, these results support that binge drinking is already characterized by an underactivation of the reflective system combined with an overactivation of the automatic system. Results also underlined the influence of explicit processing compared with implicit ones. At the clinical level, our findings reinforce the need to develop intervention methods focusing on the inhibition of approach behaviors towards alcohol-related stimuli.

KEYWORDS

alcohol cues, binge drinking, dual process, explicit, implicit, inhibition

1 | INTRODUCTION

As they are constantly facing stimuli from their environment, human beings have to propose accurate behavioral responses, ensuring their well-being and social integration. Influential theories[1] postulate that adapted responses to these environmental stimuli can be conducted through a deliberate way, involving a cognitive evaluation of the stimuli (based on the reflective system) and relying on prefrontal regions,[2] or alternatively, through an intuitive way, involving an affect-based evaluation of the stimuli (based on the automatic system) and relying on limbic regions.[3] Crucially, this dual-process framework stipulates that addictive disorders result from an imbalance between reflective and automatic systems[4] and conceptualizes severe alcohol-use disorders as relying on an underactivated reflective system, leading to an inability to inhibit alcohol consumption, coupled with an overactivated automatic system, leading to increased appetence towards alcohol

cues.[5,6] This theoretical perspective has also been proposed to understand excessive drinking habits in youth, and especially binge drinking,[7] a pattern of alcohol consumption defined by repeated alternations between large alcohol intakes and withdrawals. Actually, excessive alcohol use is a central public health issue in Western societies[8] but, while the consequences of severe alcohol-use disorders have been largely explored, binge drinking is a quite recent research topic. Yet, studies focusing on binge drinking indicate that this consumption pattern is widespread in youth,[9,10] associated with various consequences.[11,12] Besides, binge drinking has been recognized as an important risk factor for the development of severe alcohol-use disorders.[13,14] Indeed, although binge drinking presents lower intensity than severe alcohol-use disorders, it would lead to similar cognitive and affective difficulties.[15] Nevertheless, only few studies exploring the dual-process model have focused on such preclinical populations, and very little targeted binge drinking pattern. Particularly, little is known regarding how automatic and controlled activations interact in binge drinkers (BD) and at which level of the cognitive stream. Applying this model in binge drinking would allow a better understanding of the mechanisms involved in this hazardous habit.

Deficits of the reflective system in binge drinking have been recently documented, results showing impairments in executive processes such as working memory, planning, or inhibition.[16-19] Concerning inhibition, most studies have focused on prepotent response inhibition, namely, the ability to control an automatic response,[20] notably because this inhibitory process can be directly related to uncontrolled alcohol consumption. In particular, modified electrophysiological activity was observed during working memory[21] and inhibition,[22,23] indexing that BD, even in the absence of detectable behavioral modification, need more brain resources to perform executive tasks. Regarding the automatic system, binge drinking is also characterized by strong alcohol-related biases and associations.[16,24-26] Increased brain activations were also found when confronted with alcohol cues[27,28] particularly for early attentional[29] and decisional[30] electrophysiological processes.

Nevertheless, beyond these disjoint explorations of each system, few studies have investigated their interactions in binge drinking. Evidence has been gathered among young hazardous or heavy drinkers, but behavioral results are currently contrasted,[31,32] although neuroimaging and neurophysiological studies consistently indicated a need for increased brain resources to inhibit automatic responses towards alcohol cues[33,34] and reduced cognitive processing when facing alcohol-related stimuli.[35] However, only one study has specifically focused on binge drinking habits, showing impairments for the prepotent response inhibition of alcohol-related stimuli, but in comparison with neutral forms.[36]

Therefore, beyond their valuable contribution, three main limitations can be identified in previous studies, which hamper to confirm the validity of the dual-process model's main assumption (ie, imbalance between reflective and automatic systems) in binge drinking. First, the current literature presents a large variation in the determination of alcohol consumption patterns. Indeed, only one study specifically focused on key binge drinking characteristics, while others rather considered the number of drinks consumed[35] or used a general alcohol screening tool (ie, AUDIT) to categorize excessive drinkers.[34] Second, on the basis of current findings, it remains difficult to understand which type of processing (ie, explicit or implicit) impairs inhibition abilities. Indeed, previous studies evaluated either explicit[34,36] or implicit[32,35] processing of alcohol cues, but no research has jointly explored these two processing types. Finally, earlier results might be related to a general perceptive difference (eg, in visual complexity) between alcohol-related and nonalcohol-related cues (eg, geometrical forms[36]), rather than to a specific influence of the alcohol-related nature of the stimuli.

The aim of this study was to explore, at behavioral and electrophysiological levels, the modulation of inhibition abilities by alcohol-related stimuli in BD, to overcome the limitations described above. First, regarding alcohol consumption patterns, we focused on binge drinking by examining the occurrence of binge drinking episodes (more than six alcohol doses in a unique occasion[22]) and computing a binge drinking score,[36,37] which focuses on binge drinking pattern's specificities. Second, the explicit processing of alcohol cues was evaluated (alcohol/soft) and compared with an implicit condition presenting the same stimuli but requiring a processing nondirectly associated with alcohol-related content (sparkling/nonsparking). Third, we precisely matched alcohol cues with neutral ones (soft drinks) regarding visual complexity (size, color, and luminance) to explore the specific influence of alcohol cues on cognitive processing. These two last points have been investigated by distinct statistical analyses, respectively comparing explicit versus implicit processing, and the influence of alcohol cues versus neutral cues on inhibitory control. Importantly, in reference to the existing literature and as we were particularly interested in the abilities directly involved in the control of alcohol consumption, we evaluated prepotent response inhibition using a Go/No-Go paradigm. This task allows a reliable evaluation of the interaction between reflective (ie, inhibition performance) and automatic (ie, alcohol cues processing) systems, as proposed in the dual-process model. Besides, we used event-related potentials (ERPs) to overcome the limits identified in earlier studies: The role played by visual complexity and the comparison between explicit and implicit processing can indeed be more thoroughly understood by this electrophysiological tool, presenting high temporal resolution (eg, exploration of the successive steps related to controlled and automatic processes[38]). We hypothesized that BD will present inhibition difficulties (at behavioral and electrophysiological levels), mainly when (1) confronted with alcohol-related cues and (2) an explicit processing of alcohol-related cues is requested.

2 | MATERIALS AND METHOD

2.1 | Participants and procedure

A preliminary screening was performed on 4173 students of the Université catholique de Louvain (Belgium), among which 2927 accepted to take part in experimental studies. Participants were then contacted according to their alcohol consumption pattern, namely, (1) binge drinking score,[37] focusing on consumption speed and drunkenness frequency in the last six months, using the following

formula: ([4 × Consumption speed] + Number of drunkenness episodes + [0.2 × Percentage of drunkenness episodes]); and (2) other alcohol variables, ie, number of occasions per week (two to four for BD and up to four for comparison participants [CP]) and number of drinks per occasion (higher than or equal to six doses for BD and up to three for CP; an alcohol dose containing 10 g of pure ethanol) (Table 1). Participants also had to meet the following criteria: fluent French speakers, at least 18 years old, no severe alcohol-use disorders and no family history of alcohol-use disorders, no psychological/neurological disorders, and absence of past/current drug consumption (except alcohol and tobacco). All exclusion criteria were assessed by binary items. Particularly, for drug use, three questions were proposed: Are you smoking cigarettes, and at which frequency/intensity? Are you smoking cannabis? Are you consuming other drugs? Finally, the 50 participants who better matched those criteria were included: 25 BD (binge drinking score $\geq$ 16) and 25 CP (binge drinking score $\leq$ 12). Cutoff binge drinking scores were adapted from those proposed by Townshend and Duka,[37] to take into account the difference between Belgium and England regarding the number of ethanol grams per standard alcohol unit. Cutoffs were thus defined according to our previous studies[39] and supported by comparisons with other alcohol variables (Table 1). Finally, several psychopathological variables were evaluated before starting the experiment: anxiety (STAI[40]), depressive symptoms (BDI-II[41]), impulsivity (UPPS-P[42]), and general alcohol consumption pattern (AUDIT[43]). Participants were also asked to abstain from drinking alcohol in 3 days before testing, and this was controlled by self-reported measures before starting the experiment. All participants (54% women) were between 18 and 24 years old (M = 21.28, SD = 1.79). This study is part of a wider research project aiming at investigating the inhibitory control of affective stimuli in binge drinking. The ethical committee of the Psychological Science Research Institute (Université catholique de Louvain) approved this study, which followed the recommendations of the Declaration of Helsinki.

TABLE 1 Demographic and psychological measures for binge drinkers (BD) and comparison participants (CP): mean (SD)

Variable	BD (n = 25)	CP (n = 25)
Demographic measures		
Age[ns]	20.88 (1.69)	21.68 (1.82)
Gender ratio (female/male)[ns]	15/10	12/13
Psychological measures		
Beck depression inventory[ns]	4.72 (2.75)	3.96 (3.14)
State anxiety inventory (STAI-A)[ns]	31.12 (7.98)	31.60 (8.52)
Trait anxiety inventory (STAI-B)[ns]	38.84 (7.44)	36.08 (9.16)
Impulsivity (UPPS-P)		
*Negative urgency**	9.83 (3.20)	8.00 (2.65)
*Positive urgency**	11.38 (2.32)	9.04 (3.59)
*Lack of premeditation**	8.83 (2.60)	6.91 (2.45)
*Lack of perseverance**	7.96 (2.18)	6.45 (2.09)
Sensation seeking[ns]	10.00 (2.62)	10.09 (2.49)
Alcohol consumption measures		
Alcohol Use Disorder Identification Test**	17.20 (5.32)	7.56 (4.17)
Binge drinking score**	38.36 (22.32)	6.16 (3.37)
Total alcohol units per week**	22.52 (11.69)	6.30 (5.15)
Number of occasions per week[ns]	2.76 (0.88)	3.56 (2.42)
Number of alcohol units per occasion**	8.11 (3.14)	1.44 (0.85)
Consumption speed (units per hour)**	3.32 (0.90)	1.24 (0.63)

Abbreviation: ns, nonsignificant.

*$P < 0.05$.

**$P < 0.001$.

Regarding the experimental procedure, an informed consent was first provided, and participants then filled in the online questionnaires (LLC, Qualtrics Software) during the electroencephalogram (EEG) setting up. The Go/No-Go task was presented using E-Prime 2 Professional (Psychology Software Tools, Pittsburgh, Pennsylvania). Each session was administrated individually in a quiet room, and participants were placed at 60 cm from the screen (Dell E176FP, resolution: 1280 × 1024 pixels). Students were debriefed at the end of the experiment and were paid for their participation (€20).

2.2 | Experimental task

The experimental task assessed the ability to stop a dominant response (ie, prepotent response inhibition) and evaluated the effect of alcohol cues on this ability. A first condition evaluated the explicit processing of alcohol cues (ie, alcohol being the dimension to process, as participants had to distinguish alcoholic and nonalcoholic drinks), and a second condition evaluated their implicit processing (ie, alcohol not being the dimension to process, as participants had to distinguish sparkling and nonsparkling beverages, independently from their alcohol content), each being presented in separate conditions as "Go" and "No-Go" targets. Conditions were presented in pseudo-randomized order across participants (Latin square). First, to evaluate the explicit processing of alcohol cues, participants had to respond regarding the content of the drink (alcoholic drink versus soft drink). In the first part, participants answered to "Go" trials, Go-stimuli being alcoholic drinks (144 trials) and refrained from answering in "No-Go" trials, No-Go–stimuli being soft drinks (48 trials). In a reverse second part, Go-stimuli were soft drinks (144 trials) and No-Go–stimuli were alcoholic drinks (48 trials). Then, to compare explicit and implicit alcohol-related processing, another condition was introduced, in which participants had to distinguish sparkling versus nonsparkling stimuli. In line with the first condition, the first part used sparkling drinks as Go-stimuli (144 trials) and nonsparkling drinks as No-Go–stimuli (48 trials), the second part proposing the reverse response pattern (ie, 144 "Go" nonsparkling stimuli and 48 "No-Go" sparkling stimuli). The Go/No-Go paradigm contained 384 trials per condition (total: 768 trials).

During a trial, a central cross was first presented (500 milliseconds), followed by the stimulus, presented until the participant answered or for a maximum of 900 milliseconds. Participants had to press the correct button with their dominant hand, response keys differing across conditions. Stimuli depicting alcohol and nonalcohol drinks were selected from the Amsterdam Beverage Picture Set[44]: Eight alcoholic and eight soft drinks, matched for color, size, and luminance, with four sparkling and four nonsparkling beverages, were selected. A behavioral pretest was conducted on 26 psychology students (who did not take part in the subsequent study) to

ensure the correct matching between conditions, and results showed that inhibition performance (ie, percentage of correct "No-Go" responses) did not differ between explicit and implicit processing (t_{25} = 0.89, P = 0.382).

2.3 | EEG acquisition and preprocessing

Electrophysiological data were recorded with a 128-channel Biosemi ActiveTwo system referenced to the CMS-DRL ground (http://www.biosemi.com) at 1024 Hz (0- to 208-Hz bandwidth), and EEG processing was performed using BrainVision Analyzer (version 2). A first band-pass filter between 0.1 and 30 Hz (Butterworth Zero Phase Filters, 12 dB/oct) was applied, followed by a notch filter at 50 Hz. Independent component analysis was then conducted to remove ocular artifacts.[45] All signals were rereferenced to average, and EEG segments were constructed on the basis of a 200-milliseconds baseline and 800 milliseconds following stimulus onset; a baseline correction was applied regarding the mean activity during the 200 milliseconds prior to response onset. Algorithmic artifact rejection of voltage exceeding ±100 μV was conducted; segments with artifacts were inspected and manually rejected. Finally, individual participant averages were built separately for correct responses in each condition. According to previous studies[34,46] and visual inspection of the data, each ERP component was quantified at central, left, and right electrodes in adapted time intervals, namely, 80 to 130 milliseconds at Oz-O1-O2 for P100, 200 to 300 milliseconds at Fz-F3-F4 for N200, and 300 to 600 milliseconds at Pz-P3-P4 for P300.

2.4 | Statistical analyses

The correct group matching was supported by independent samples t tests for age and psychopathological comorbidities and by a chi-squared test for gender. Repeated measure analyses of variance (ANOVAs) were then conducted to explore group differences, separately for correct "Go" responses and inhibition performance (correct "No-Go" responses). At the behavioral level, a first analysis was computed to compare the effect of explicit and implicit beverage cues processing on inhibition, by a 2 × 2 ANOVA with group (BD and CP) as between-subjects factor and condition (explicit processing and implicit processing) as within-subjects factor. Then, a second analysis investigated the effect of stimulus type (alcohol versus soft) by a 2 × 2 ANOVA with group as between-subjects factor and stimulus (alcohol and soft) as within-subjects factor. At the electrophysiological level, similar ANOVAs were performed separately for amplitude and latency and included electrodes as a supplemental within-subjects factor: Oz, O1, and O2 for P100; Fz, F3, and F4 for N200; and Pz, P3, and P4 for P300. For all analyses, significant effects between groups were explored by post hoc two-tailed t tests (P < 0.05) and adjusted for multiple comparisons through Bonferroni correction. According to our a priori hypotheses, the following significant thresholds were thus applied: (a) the initial P value was divided by two (ie, significant when P < 0.025) when exploring double interactions (post hoc were performed separately for explicit and implicit processing in the first analysis and for alcohol and soft stimuli in the second analysis) and (b) the initial P value was divided by four (ie, significant when P < 0.012) when exploring triple interactions (for electrophysiological analyses). As we were centrally interested in group comparisons, only the main effects and interactions involving groups are presented (see the Supporting Information for other results and all ERP waveforms). Finally, correlational analyses were performed, separately for BD and CP, between the behavioral and electrophysiological measures that significantly differed across groups (see the Supporting Information). Moreover, to support the hypothesis that prepotent response inhibition is specifically involved in binge drinking habits, and to determine the specific mechanisms implicated in this relationship, we performed correlations between inhibition performance and alcohol consumption, as well as a regression with binge drinking score as dependent variable.

3 | RESULTS

3.1 | Demographic and psychopathological measures

No difference emerged between groups for age (t_{48} = 1.61, P = 0.114), gender ($\chi^2_{1,N\,=\,50}$ = 0.73, P = 0.395), depressive symptoms (t_{48} = 0.91, P = 0.367), state anxiety (t_{48} = 0.21, P = 0.838), or trait anxiety (t_{48} = 1.17, P = 0.248). However, higher impulsivity (ie, negative and positive urgency, lack of premeditation, lack of perseverance) and alcohol consumption were observed in BD (Table 1).

3.2 | Behavioral analyses

Percentage of correct responses and reaction times (RT) for behavioral data are reported in Table 2 for BD and CP.

TABLE 2 Percentage of correct answers for "Go" and "No-Go" trials and reaction times (RT, in milliseconds) for "Go" trials among binge drinkers (BD) and comparison participants (CP) in each experimental condition: mean (SD)

		Explicit Processing		Implicit Processing	
Variable	Group	Alcohol	Soft	Sparkling	Nonsparkling
"Go"	BD	98.39 (1.80)	98.31 (1.69)	97.89 (3.12)	96.42 (4.49)
	CP	98.39 (1.93)	97.75 (2.86)	96.92 (3.87)	96.89 (2.73)
RT "Go"	BD	446 (35)	433 (45)	436 (33)	464 (44)
	CP	441 (39)	441 (47)	445 (43)	473 (41)
"No-Go"	BD	73.00 (13.32)	72.00 (16.45)	73.50 (15.85)	78.83 (18.70)
	CP	78.00 (8.76)	76.50 (12.67)	74.58 (12.22)	76.75 (15.08)

3.3 | Percentage of correct "Go" responses

Regarding the type of processing, there were no main group effect ($F_{1,48}$ = 0.23, P = 0.632, η^2_p = 0.005) and no group × condition interaction ($F_{1,48}$ = 0, P = 0.968, η^2_p = 0).

Regarding the type of stimuli, there were no main group effect ($F_{1,48}$ = 0.28, P = 0.600, η^2_p = 0.006) and no group × stimulus interaction ($F_{1,48}$ = 0.92, P = 0.342, η^2_p = 0.019).

3.4 | Percentage of correct "No-Go" responses

Regarding the type of processing, there was no main group effect ($F_{1,48}$ = 0.23, P = 0.632, η^2_p = 0.005) but a significant group × condition interaction ($F_{1,48}$ = 4.62, P = 0.037, η^2_p = 0.088), showing lower inhibition performance for explicit than implicit processing in BD (t_{24} = 2.64, P = 0.014) but not in CP (t_{24} = 0.79, P = 0.439) (Figure 1).

Regarding the type of stimuli, there were no main group effect ($F_{1,48}$ = 1.97, P = 0.167, η^2_p = 0.039) and no group × stimulus interaction ($F_{1,48}$ = 0, P = 0.869, η^2_p = 0.001).

3.5 | RT for "Go" trials

Regarding the type of processing, there were no main group effect ($F_{1,48}$ = 0.23, P = 0.632, η^2_p = 0.005) and no group × condition interaction ($F_{1,48}$ = 2.80, P = 0.101, η^2_p = 0.055).

Regarding the type of stimuli, there were no main group effect ($F_{1,48}$ = 0.01, P = 0.912, η^2_p = 0) and no group × stimulus interaction ($F_{1,48}$ = 2.68, P = 0.108, η^2_p = 0.053).

3.6 | Electrophysiological analyses

3.6.1 | Percentage of correct "Go" responses

Mean peak amplitudes and latencies of ERP components for "Go" trials are reported in Table 3.

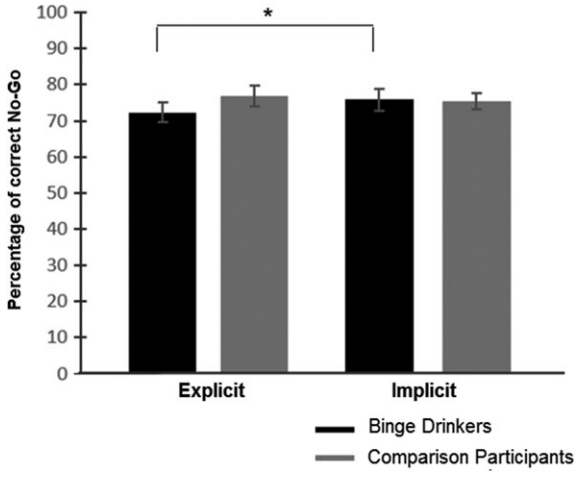

FIGURE 1 Performance of binge drinkers (black line) and comparison participants (grey line) in "No-Go" trials. Bar charts depict the percentage of correct inhibition during explicit and implicit processing, showing reduced performance for explicit processing in binge drinkers

- P100 amplitude: For the types of processing and stimuli, respectively, there were no main group effect ($F_{1,48}$ = 0.04, P = 0.848, η^2_p = 0.001; $F_{1,48}$ = 0.06, P = 0.804, η^2_p = 0.001) and no interaction (all $F \leq 0.60$, all $P \geq 0.550$).
- P100 latency: For the type of processing, there was no main group effect ($F_{1,48}$ = 0.12, P = 0.727, η^2_p = 0.003) but a group × condition interaction ($F_{1,48}$ = 4.55, P = 0.038, η^2_p = 0.087). Longer latencies for implicit than explicit processing were observed in CP (t_{24} = 2.29, P = 0.031) and not in BD (t_{24} = 0.45, P = 0.658), but this effect did not reach the corrected threshold (P < 0.025). Other interactions were not significant (all $F \leq 0.36$, all $P \geq 0.701$). For the type of stimuli, no main group effect ($F_{1,48}$ = 1, P = 0.322, η^2_p = 0.012) and no interaction (all $F \leq 1.01$, all $P \geq 0.368$) were found.
- N200 amplitude: For the type of processing, there was no main group effect ($F_{1,48}$ = 0.52, P = 0.474, η^2_p = 0.011) but a group × electrode interaction ($F_{2,96}$ = 3.62, P = 0.030, η^2_p = 0.070). Higher N200 amplitudes at F3 than F4 were found in CP (t_{24} = 2.08, P = 0.048) and not in BD (t_{24} = 1.26, P = 0.220), but this effect did not reach the corrected threshold (P < 0.025). Other interactions were not significant (all $F \leq 2.35$, all $P \geq 0.132$). For the type of stimuli, no main group effect ($F_{1,48}$ = 1.14, P = 0.290, η^2_p = 0.023) and no interaction (all $F \leq 1.81$, all $P \geq 0.170$) were found.
- N200 latency: For the types of processing and stimuli, respectively, no main group effect ($F_{1,48}$ = 0.31, P = 0.860, η^2_p = 0.001; $F_{1,48}$ = 0.01, P = 0.915, η^2_p = 0) and no interaction (all $F \leq 1.05$, all $P \geq 0.355$) were found.
- P300 amplitude: For the types of processing and stimuli, respectively, there were no main group effect ($F_{1,48}$ = 0.46, P = 0.501, η^2_p = 0.009; $F_{1,48}$ = 0.36, P = 0.550, η^2_p = 0.008) and no interaction (all $F \leq 2.52$, all $P \geq 0.086$).
- P300 latency: For the types of processing and stimuli, respectively, no main group effect ($F_{1,48}$ = 1.35, P = 0.251, η^2_p = 0.027; $F_{1,48}$ = 0.56, P = 0.457, η^2_p = 0.012) and no interaction (all $F \leq 2.94$, all $P \geq 0.058$) were found.

3.6.2 | Percentage of correct "No-Go" responses

Mean peak amplitudes and latencies of ERP components for "No-Go" trials are reported in Table 4.

- P100 amplitude: For the types of processing and stimuli, respectively, there were no main group effect ($F_{1,48}$ = 0.04, P = 0.848, η^2_p = 0.001; $F_{1,48}$ = 0.17, P = 0.679, η^2_p = 0.004) and no interaction (all $F \leq 0.90$, all $P \geq 0.348$).
- P100 latency: For the types of processing and stimuli, respectively, no main group effect ($F_{1,48}$ = 0.36, P = 0.550, η^2_p = 0.007; $F_{1,48}$ = 0, P = 0.987, η^2_p = 0) and no interaction (all $F \leq 2.30$, all $P \geq 0.136$) were found.
- N200 amplitude: For the type of processing, there was no main group effect ($F_{1,48}$ = 0.24, P = 0.627, η^2_p = 0.005) but a group × electrode interaction ($F_{2,96}$ = 4.75, P = 0.011, η^2_p = 0.090), indicating higher N200 amplitude at F3 than F4 in

TABLE 3 Amplitude (in microvolts) and latency (in milliseconds) [mean (SD)] of the event-related potential (ERP) components (ie, P100, N200, and P300) elicited at "Go" trials for each variable, condition, and at each electrode for binge drinkers (BD) and comparison participants (CP)

			Explicit Processing				Implicit Processing			
			Alcohol		Soft		Sparkling		Nonsparkling	
Variable	Electrode	Group	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency
P100	Oz	BD	5.88 (4.06)	116 (15)	6.61 (4.67)	109 (22)	4.86 (4.19)	105 (33)	6.03 (4.25)	117 (16)
		CP	6.04 (5.28)	106 (33)	6.37 (4.86)	105 (28)	5.24 (4.68)	111 (29)	5.82 (4.83)	114 (22)
	O1	BD	7.53 (3.93)	121 (9)	7.61 (4.76)	116 (23)	6.35 (3.84)	115 (22)	7.23 (4.35)	118 (18)
		CP	6.99 (5.59)	117 (16)	7.11 (5.46)	112 (24)	6.36 (5.26)	112 (27)	6.76 (5.19)	120 (18)
	O2	BD	7.00 (5.32)	116 (22)	7.68 (4.42)	112 (23)	6.29 (5.77)	112 (32)	7.37 (5.52)	117 (25)
		CP	6.67 (4.15)	115 (23)	7.20 (4.43)	106 (35)	6.26 (3.75)	119 (17)	6.76 (3.96)	119 (20)
N200	Fz	BD	−5.97 (4.10)	281 (18)	−5.64 (3.76)	279 (24)	−6.15 (3.60)	280 (17)	−7.31 (4.57)	281 (18)
		CP	−6.78 (3.04)	282 (19)	−6.80 (2.60)	279 (22)	−6.82 (2.78)	283 (21)	−7.44 (2.55)	282 (19)
	F3	BD	−4.02 (3.05)	278 (23)	−3.96 (3.12)	275 (27)	−3.99 (2.53)	281 (19)	−5.10 (3.89)	279 (21)
		CP	−5.17 (2.92)	281 (20)	−5.27 (2.81)	272 (23)	−5.36 (2.84)	281 (21)	−5.55 (2.36)	276 (19)
	F4	BD	−4.30 (3.45)	282 (18)	−4.12 (3.60)	279 (23)	−4.46 (3.70)	276 (20)	−5.85 (4.78)	282 (14)
		CP	−4.48 (1.99)	282 (22)	−4.59 (1.86)	275 (21)	−4.24 (1.75)	273 (21)	−4.96 (1.71)	277 (20)
P300	Pz	BD	8.37 (3.63)	370 (55)	8.14 (3.92)	352 (41)	8.37 (3.88)	374 (50)	8.40 (3.53)	386 (60)
		CP	8.76 (3.41)	370 (51)	8.61 (4.20)	370 (51)	8.80 (3.98)	367 (49)	8.65 (3.33)	373 (63)
	P3	BD	5.77 (3.33)	349 (38)	5.29 (3.21)	350 (51)	5.88 (3.10)	355 (38)	5.83 (3.44)	359 (55)
		CP	5.33 (2.37)	341 (34)	5.18 (2.42)	335 (34)	5.71 (2.38)	354 (45)	5.65 (2.60)	357 (56)
	P4	BD	6.76 (3.16)	376 (61)	5.98 (3.23)	360 (57)	6.50 (3.50)	386 (73)	6.48 (3.39)	380 (65)
		CP	8.01 (3.85)	350 (46)	7.25 (3.42)	349 (52)	7.96 (3.18)	352 (62)	8.19 (3.42)	353 (64)

TABLE 4 Amplitude (in microvolts) and latency (in milliseconds) [mean (SD)] of the event-related potential (ERP) components (ie, P100, N200, and P300) elicited at "No-Go" trials for each variable, condition, and at each electrode for binge drinkers (BD) and comparison participants (CP)

			Explicit Processing				Implicit Processing			
			Alcohol		Soft		Sparkling		Nonsparkling	
Variable	Electrode	Group	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency	Amplitude	Latency
P100	Oz	BD	6.16 (4.19)	100 (39)	7.58 (4.97)	104 (30)	7.12 (4.74)	106 (32)	5.22 (3.90)	88 (45)
		CP	5.37 (4.28)	101 (38)	7.85 (6.49)	106 (26)	6.13 (4.43)	101 (39)	4.64 (3.72)	103 (33)
	O1	BD	7.59 (4.11)	110 (33)	8.63 (4.86)	114 (19)	8.23 (4.41)	105 (41)	5.90 (3.41)	98 (42)
		CP	6.73 (4.71)	107 (34)	8.77 (7.38)	111 (24)	7.66 (4.89)	111 (29)	5.76 (4.11)	112 (27)
	O2	BD	7.56 (5.80)	111 (29)	8.77 (6.61)	107 (29)	8.38 (6.44)	104 (38)	6.71 (5.50)	106 (33)
		CP	6.22 (3.84)	108 (40)	8.14 (4.59)	111 (23)	6.97 (3.56)	114 (24)	5.33 (3.05)	112 (25)
N200	Fz	BD	−7.04 (4.47)	282 (26)	−5.85 (4.90)	281 (21)	−6.91 (4.86)	279 (19)	−5.80 (3.64)	285 (19)
		CP	−6.69 (3.16)	278 (24)	−7.55 (5.51)	278 (23)	−7.27 (2.89)	284 (17)	−6.13 (2.26)	281 (18)
	F3	BD	−4.20 (3.30)	274 (28)	−4.33 (3.54)	284 (21)	−4.53 (3.48)	280 (17)	−4.70 (4.16)	280 (22)
		CP	−5.51 (2.66)	273 (29)	−5.9 (4.44)	270 (26)	−5.89 (3.56)	278 (20)	−4.83 (2.26)	281 (19)
	F4	BD	−5.36 (4.50)	280 (22)	−4.80 (3.63)	280 (17)	−5.67 (4.04)	277 (22)	−4.08 (3.92)	278 (24)
		CP	−4.25 (2.57)	273 (23)	−5.26 (4.76)	276 (23)	−4.45 (2.32)	280 (18)	−4.43 (2.22)	280 (18)
P300	Pz	BD	9.48 (3..71)	425 (85)	9.08 (4.23)	392 (67)	8.27 (3.51)	420 (85)	7.70 (3.60)	415 (83)
		CP	8.17 (4.29)	392 (67)	9.92 (6.55)	397 (64)	8..53 (4.14)	422 (83)	8.32 (3.42)	398 (80)
	P3	BD	6.83 (2.98)	384 (74)	5.26 (3.35)	380 (73)	5.76 (3.50)	408 (95)	5.36 (3.09)	388 (85)
		CP	4.85 (2.47)	374 (80)	5.56 (4.59)	386 (82)	5.70 (2.68)	407 (79)	5.07 (2.34)	396 (88)
	P4	BD	7.55 (3.87)	378 (74)	7.01 (3.53)	399 (81)	7.16 (3.97)	361 (57)	5.77 (3.28)	376 (82)
		CP	7.62 (3.87)	363 (78)	8.81 (7.73)	371 (61)	7.89 (3.71)	355 (53)	7.07 (2.77)	361 (67)

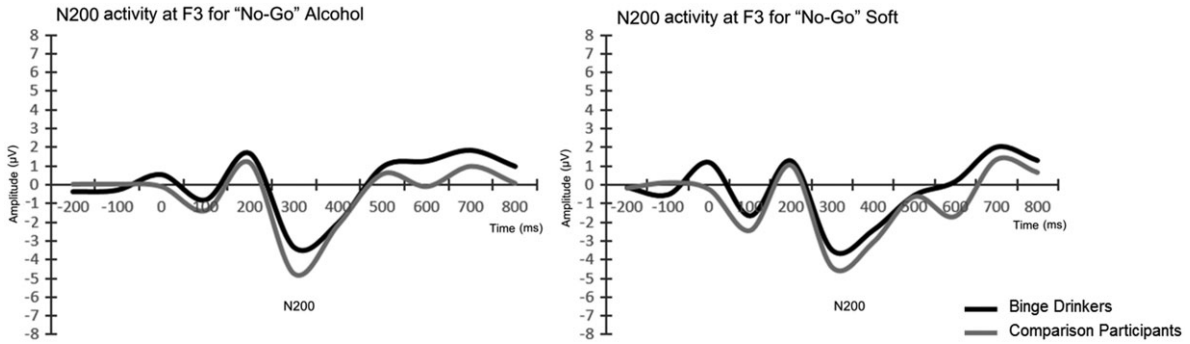

FIGURE 2 Illustration of the grand average event-related potential (ERP) waveforms among binge drinkers (black line) and comparison participants (grey line), showing a reduced N200 amplitude in binge drinkers for the processing of alcohol-related stimuli at F3

CP (t_{24} = 2.56, P = 0.017) but not in BD (t_{24} = 1.36, P = 0.187). Other interactions were not significant (all $F \leq 1.12$, all $P \geq 0.295$). For the type of stimuli, no main group effect ($F_{1,48}$ = 0.45, P = 0.508, η^2_p = 0.009) but a group × electrode interaction ($F_{2,96}$ = 4.42, P = 0.015, η^2_p = 0.084) was found and qualified by a group × stimulus × electrode interaction ($F_{2,96}$ = 3.52, P = 0.030, η^2_p = 0.068), showing higher amplitude at F3 than F4 for the processing of alcohol-related stimuli in CP (t_{24} = 3.35, P = 0.003) but not in BD (t_{24} = 1.57, P = 0.130), where N200 amplitude was reduced (Figure 2). Moreover, the processing of soft stimuli led to the same amplitude at F3 and F4 in both groups (CP: t_{24} = 1.19, P = 0.246; BD: t_{24} = 1.08, P = 0.291). The group × stimulus interaction was not significant (F = 1.24, P = 0.270).

- N200 latency: For the types of processing and stimuli, respectively, there were no main group effect ($F_{1,48}$ = 0.28, P = 0.601, η^2_p = 0.006; $F_{1,48}$ = 1.04, P = 0.314, η^2_p = 0.021) and no interaction (all $F \leq 2.07$, all $P \geq 0.157$).
- P300 amplitude: For the types of processing and stimuli, respectively, no main group effect ($F_{1,48}$ = 0.05, P = 0.818, η^2_p = 0.001; $F_{1,48}$ = 0, P = 0.958, η^2_p = 0) and no interaction (all $F \leq 2.71$, all $P \geq 0.072$) were found.
- P300 latency: For the types of processing and stimuli, respectively, there were no main group effect ($F_{1,48}$ = 0.53, P = 0.471, η^2_p = 0.011; $F_{1,48}$ = 0.93, P = 0.340, η^2_p = 0.019) and no interaction (all $F \leq 1.82$, all $P \geq 0.168$).

3.6.3 | Correlational analyses

- Higher N200 amplitude was related to better inhibition performance in CP but not in BD (see the Supporting Information).
- The percentage of correct "No-Go" responses was not related to alcohol consumption in BDs or CPs (all $r \leq 0.32$, all $p \geq 0.115$). However, while no relations were found in CP, the RT for "Go" trials was negatively correlated with alcohol consumption in BD. For alcohol cues explicit processing, correlations were observed with AUDIT (r = −0.47, P = 0.017) and binge drinking (r = −0.78, P < 0.001) scores, the total weekly consumption (r = −0.70, P < 0.001), and the mean number of doses consumed per occasion (r = −0.60, P = 0.002). Moreover, for implicit processing, correlations were found with binge drinking score (r = −0.50, P = 0.011) and total weekly consumption (r = −0.41, P = 0.040). These findings were supported by a regression analysis with binge drinking score as dependent variable (the RT for "Go" trials in all conditions was entered as independent variable in a stepwise linear model), highlighting that the RT for alcohol "Go" trials was the only significant binge drinking predictor (R^2 = 0.61, P < 0.001).

4 | DISCUSSION

This study evaluated the relevance of the dual-process proposal to account for binge drinking pattern in young adults. This framework has provided convincing evidence in explaining the onset and maintenance of severe addictive disorders, but its validity in preclinical populations still lacks experimental support. The current study investigated prepotent response inhibition abilities during the explicit and implicit processing of alcohol-related stimuli, at behavioral and electrophysiological levels.

First, our behavioral findings highlight that BD present higher difficulties to inhibit a prepotent response. Poorer "No-Go" performance for the explicit condition is found in BD: When explicit processing of alcohol and nonalcohol content is required, BD are less efficient than when implicit processing is performed. Therefore, beyond the impact of alcohol cues on inhibitory control, as proposed in the dual-process model, these findings emphasize a difficulty to process alcoholic and nonalcoholic beverage cues in binge drinking. It could thus be hypothesized that the implicit presentation of alcohol cues activates positive alcohol-related representations and potentially influences future alcohol and binge consumption[47] but does not directly impact self-control abilities. Regarding the first research question focusing on the type of processing, it thus appears that inhibition of explicit beverage cues is particularly impaired in BD. Compared with implicit one, explicit processing might thus be considered as a central mechanism involved in the automatic system's overactivation. Particularly, this higher reactivity during explicit processing of beverage cues, combined with a reduced ability to control a dominant response, might be involved in excessive alcohol use in BD.

Second, findings indicate a specific impact of alcohol cues processing on inhibitory control, specifically observed at the electrophysiological level and indexed by reduced N200 amplitude in BD. The N200 component is related to attentional focus and particularly, during "No-Go" processing, to conflict detection.[48] At the cerebral level, BD thus present reduced attentional resources to detect "No-Go" stimuli, mostly in the presence of alcohol cues. In line with the dual-process model, it appears that electrophysiological attentional processes are impacted in BD only when alcohol cues are displayed, which could be accounted for by an imbalance between underactivated reflective and overactivated automatic systems.[5] These findings are consistent with earlier studies showing inhibition impairments during explicit alcohol cues processing in binge drinking[36] but confirm this effect with a strict control condition and extend its understanding by showing a specific attentional difficulty at the electrophysiological level. Moreover, the present results also relate to those of Kreusch et al[34] who highlighted an absence of anterior dominance in N200 amplitude among young drinkers. Actually, our findings indicate a reduced N200 at left frontal electrode (F3) for "No-Go" trials in BD whereas a larger N200 is clearly observed in CP. Therefore, these results support, beyond amplitude changes, a modification in the topography of brain activity in binge drinking. Eventually, contradictory to previous studies,[33,35] the present results do not illustrate an increase of brain activity during alcohol cues processing, potentially explained by the fact that we conducted separate analyses for "Go" and "No-Go" trials. Indeed, while earlier works compared these two types of trials and thus showed a need for higher brain resources to inhibit than to categorize, the current research aimed to target these two processing separately and to point out the specific prepotent response inhibition performance (correct responses in "No-Go" trials). Focusing on inhibition, our results are quite consistent with earlier research

showing reduced electrophysiological activities together with behavioral impairments.[49] Altogether, and regarding the second research question focused on stimulus type, it appears that an overactivation of the automatic system is observed during alcohol cues presentation and impacts inhibition abilities in BD. These results support the dual-process proposal and suggest that the specific difficulty to explicitly inhibit alcohol-related content is involved in the persistence of binge drinking habits (eg, difficulty to refrain from consuming alcohol drinks in a context where alcohol is present, typically in student parties). Concerning this secondary aim targeting the relationship between inhibition performance and alcohol consumption, correlations were performed and showed no significant results in BD. However, it has been found that the faster participants answered to "Go" trials, the higher was their alcohol consumption. This relation was further confirmed by a regression analysis showing that the RT for alcohol "Go" trials was the unique predictor of binge drinking habits. This association between binge drinking and faster RT is in accordance with the higher self-reported impulsivity observed in BD and may be in line with previous studies, proposing that BD present motor impulsivity.[37,50] Therefore, beyond the reduced performance for inhibition in BD compared with CP, it would seem that an increase motor impulsivity for alcohol-related stimuli would specifically predict binge drinking. Yet, as evaluating the relationship between binge drinking and inhibition was not the primary aim of this study, this specific issue should be further addressed.

It is worth noting that this study not only offers several perspectives for future research but also presents some limitations. In accordance with previous works, the current findings suggest that binge drinking is characterized by an imbalance between reflective and automatic systems, and specify the importance of distinct mechanisms. Therefore, future studies should explore the role of attentional processes, as well as the influence of explicit processing on inhibitory control. Indeed, while we suggest the involvement of attentional processes based on electrophysiological findings, it would be interesting to directly investigate the possible role of attentional abilities in the imbalance between systems. Importantly, the current study has focused on prepotent response inhibition to better understand binge drinking habits, as excessive consumption episodes can be directly associated with the difficulty to inhibit an approach and/or dominant automatic behavior (eg, drinking alcohol). However, future studies should not only explore other inhibitory processes, particularly cognitive inhibition or the ability to control distractor interference, but also support this assumption by investigating the relationship between inhibition and binge drinking in longitudinal designs. Beyond these perspectives, and although the sample size of the current study has allowed identifying significant group differences (strictly controlled for multiple comparisons), subsequent research should confirm these results with larger samples. It is indeed important to mention that the current sample size may have limited the power to detect significant group differences. Future works should also examine the influence of potential confounding factors (eg, psychopathological comorbidities and genetic factors), as the present study limited their influence at the group selection level but did not directly assess their role in the inhibition impairments presented by BD. In this regard, genetic influence has to be considered. Moreover, the nature of the relation between behavioral performance and electrophysiological modifications has not been supported by a correlational approach in BD and should be further examined. Finally, the issue of gender difference regarding the interaction between reflective and automatic systems should be addressed in subsequent works.

As a whole, this study aimed at disentangling the mechanisms involved in the persistence of binge drinking habits by focusing on the recognized dual-process model. Particularly, the current research has targeted specific binge drinking pattern, selected precisely matched cues, and compared the differential influence of explicit and implicit processing. Results highlighted inhibition deficits in binge drinking related to both (1) a stronger impairment for the explicit processing of beverage cues, observed at behavioral and cerebral levels, and (2) a specific difficulty to inhibit alcohol cues, evidenced at the cerebral level. These results are consistent with the dual-process model of addictive disorders[4] and reinforce the interest to consider the effect of alcohol-related stimuli on cognitive abilities and to specify which type (explicit) and level (attentional) of processing is related to binge drinking. At the clinical level, this study also strengthens the need for developing neurocognitive programs focusing on inhibitory control during the direct processing of alcohol cues,[51] with a specific stress on attentional abilities.

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AUTHORS CONTRIBUTION

SL, JB, MB, FD, and PM were responsible for the study concept and design. SL achieved the data acquisition. SL, VD, and FD preprocessed the data, and SL and VD performed the analyses. SL, VD, and PM worked on the interpretation of the findings. SL drafted the manuscript under the supervision of PM. All authors provided critical revision of the manuscript and approved the final version.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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