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Electrophysiological correlates of performance monitoring in binge drinking: Impaired error-related but preserved feedback processing

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HIGHLIGHTS

- Performance monitoring (PM), while crucial for adapted actions, is unexplored in binge drinking (BD).
- Brain correlates of error-related and feedback-related PM were measured by event-related potentials.
- BD showed a dissociation between PM subcomponents (preserved feedback but abnormal error processing).

ABSTRACT

Objective: Performance monitoring, which allows efficient behavioral regulation using either internal (error processing) or external (feedback processing) cues, has not yet been explored in binge drinking despite its adaptive importance in everyday life, particularly in the regulation of alcohol consumption. Capitalizing on a theoretical model of risky behaviors, the present study aimed at determining the behavioral and electrophysiological correlates of the cognitive (inhibition) and motivational (reward sensitivity) systems during performance monitoring.
Methods: Event-related potentials were recorded from 20 binge drinkers and 20 non-binge drinkers during two experimental tasks, a speeded Go/No-Go Task [investigating internal error processing by Error-Related Negativity (ERN) and error positivity (Pe)] and a Balloon Analogue Risk Task [investigating external feedback processing by Feedback-Related Negativity (FRN) and P3].
Results: While no group differences were observed at the behavioral level, electrophysiological results showed that binge drinkers, despite having intact feedback-related components, presented modified error-monitoring components (i.e. larger ERN amplitude, delayed Pe latency).
Conclusions: Internal performance monitoring is impaired in binge drinkers, showing an abnormal automatic processing of response errors (ERN) and a decreased processing of their motivational significance (Pe).
Significance: These results suggest that the electrophysiological correlates of inhibitory control allow identifying the specific binge drinking consumption pattern.

1. Introduction

Binge drinking is an alcohol consumption pattern characterized by alternations between intense alcohol intakes and abstinence periods (Courtney and Polich, 2009). Despite the lack of consensus regarding its definition, this habit is now widespread in young people in Western countries (e.g., Kanny et al., 2013). As a matter of fact, binge drinking is no longer considered as a recreational and occasional alcohol consumption activity, but rather as a risky behavior with major cognitive consequences (e.g., in memory or attentional processes; Hartley et al., 2004; Heffernan and O'Neill, 2012). Electrophysiological studies have also provided important findings in binge drinking, showing the modification

Abbreviations: BD, binge drinkers; ERN, Error-Related Negativity; FA, false alarms; FH, fast hits; FRN, Feedback-Related Negativity; PM, performance monitoring; SH, slow hits.

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of information processing at multiple levels following stimulus onset, from early perceptual and attentional stages to response selection, decision, and cognitive control (Lopez-Caneda et al., 2013; Maurage et al., 2009, 2012). Therefore, it appears crucial to target the specific processes involved in the development of this hazardous alcohol consumption habit. In this respect, several models have been proposed to better understand risky behaviors in adolescents and young adults. Widely prevalent theories have emerged from neurodevelopmental research (Casey et al., 2008, 2010), highlighting immature impulse control and heightened activation to incentives. Risk taking in youth can thus be explained by elevated impulsivity, which was defined by two different factors: (1) rash impulsiveness, reflected by poor inhibitory control relying on the cognitive system and (2) reward sensitivity, reflected by poor decision-making relying on the motivational system (Dawe and Loxton, 2004; Dawe et al., 2004). Impulsivity indeed appears as a vulnerability factor for several substance-use disorders (Boog et al., 2013; Peeters et al., 2017; Verdejo-García et al., 2008) but these two specific systems have rarely been explored in a simultaneous way in young adult binge drinkers.

On the one hand, regarding the cognitive system, binge drinking is characterized by impaired executive functions (Parada et al., 2012), particularly for inhibition (e.g., VanderVeen et al., 2013; Poulton et al., 2016), the ability to stop automatic response or inappropriate information processing. A recent study also specifies that binge drinking is associated with altered response monitoring, indexed by a weak post-error slowing effect (Bø et al., 2016a). On the other hand, regarding the motivational system, binge drinking is characterized by disadvantageous decision-making, the ability to accurately react to sensory stimulations (e.g., Goudriaan et al., 2007; Johnson et al., 2008). These impairments are driven by high reward seeking (see Stautz and Cooper, 2013 for a meta-analysis) and more risky choices (Bø et al., 2016b; Worbe et al., 2014). These data are not only correlational in nature as elevated impulsivity appears involved in the etiology and even the maintenance of binge drinking (Field et al., 2008). Indeed, while research mainly focused on the cognitive component, poor inhibitory control and risky decision-making were both identified as reliable predictors of future binge drinking habits in adolescents and young adults (Carlson et al., 2010; Peeters et al., 2015; Xiao et al., 2009). Actually, whereas other behavioral disorders are mainly predicted by inhibition deficits, binge drinking appears specifically associated with reward-related disinhibition measures (Castellanos-Ryan et al., 2011). Moreover, within the specific binge drinking pattern, higher binge drinkers demonstrated more impulse control and decision-making deficits than low binge drinkers (Townshend et al., 2014).

Consequently, the picture emerges that binge drinking could be associated with impairments in both inhibitory control and decision-making. Nevertheless, few studies have simultaneously measured cognitive and motivational systems and none has specifically investigated the electrophysiological processing related to these systems. A reliable way to consider their cerebral correlates is to focus on performance monitoring (PM), a crucial cognitive process enabling to detect mismatches between goal/intention and action, as well as to trigger subsequent remedial processes upon their timely detection (Ullsperger et al., 2014). Indeed, in line with neurodevelopmental findings in youth's risky behaviors (e.g., Casey et al., 2008), many studies have demonstrated that efficient PM is yielded by the integrity of prefrontal cortex in concert with deeper subcortical brain structures. Research in binge drinking moreover indicated abnormal activations in prefrontal cortex (e.g., Schweinsburg et al., 2011) and increased subcortical activity (e.g., Xiao et al., 2013), leading to the hypothesis that binge drinking might be explained by PM deficits. Centrally, PM is based on the information available at a given moment in time, by using internal (i.e. motor) or external (i.e. feedback) cues, to update action's value and adjust behavior. These processing have been related to two distinctive event-related components: (1) The *Error-Related Negativity* (ERN or Ne; Falkenstein et al., 2000; Gehring et al., 1993) corresponds to an early and likely automatic error-specific brain activity peaking between 0 and 100 ms at prefrontal sites, based on the processing of internal and motor cues, and presumably generated in the dorsal anterior cingulate cortex (Dehaene et al., 1994; Nieuwenhuis et al., 2001). The ERN is followed by the error positivity component (Pe), reflecting the conscious appraisal of response errors or the processing of their distinctive motivational significance (Nieuwenhuis et al., 2001; Ridderinkhof et al., 2009). Usually, a negative component is also generated after correct responses at the same latency, namely the correct-related negativity (CRN), also followed by a positive deflection, namely the correct positivity (Pc). Both ERN and CRN are thus implicated in performance monitoring (Roger et al., 2010), although this study particularly focuses on error processing, necessary to prevent rash impulsiveness by further implementing good inhibition or error correction; (2) The *Feedback-Related Negativity* (FRN; Gehring and Willoughby, 2002) reflects the same monitoring process but based on external evaluative information (Pfabigan et al., 2015; Talmi et al., 2013), elicited by negative and/or unexpected feedback on task performance. The FRN peaks 250–300 ms post-feedback onset at prefrontal sites and is also best explained by a main intracranial generator in the dorsal anterior cingulate cortex. The FRN is followed by the P3, sharing similarities with the Pe, and which would reflect the processing of the feedback motivational salience, as opposed to its valence in the case of the FRN (Polich, 2007). Therefore, feedback processing is needed to promote efficient decision-making, with a correct balance between rewards and risks.

At the electrophysiological level, most studies have shown amplitude reduction of PM components in people with substance use or behavioral disorders compared to controls, for ERN (Anokhin and Golosheykin, 2015; Steele et al., 2014; Zhou et al., 2013) and Pe (Franken et al., 2010) as well as for FRN (Torres et al., 2013; Yau et al., 2015). Interestingly, female heavy drinkers also displayed a smaller ERN amplitude, accompanied with a delayed Stop Signal Reaction Time (Smith and Mattick, 2013). However, contrary to previous findings, recent studies did not confirm this smaller ERN in heavy drinkers, one with marginal results (Smith et al., 2016) and others with no group difference for ERN (Franken et al., 2017; Smith et al., 2017), although Pe amplitude was reduced in heavy drinkers (Franken et al., 2017). Moreover, unlike other substance use or behavioral addictions (see Luijten et al., 2014 for a review), alcohol-dependence was characterized by larger, and not smaller, ERN amplitude. Indeed, without behavioral impairments, an increased ERN was reported during a Flanker task in individuals with both post-traumatic stress disorder (PTSD) and alcohol use disorder compared to individuals with only PTSD (Gorka et al., 2016). These findings were also shown among abstinent alcohol-dependent individuals (Padilla et al., 2011; Schellekens et al., 2010) and were interpreted as influenced by comorbid anxiety disorders (Schellekens et al., 2010) or as reflecting the increased resources needed by participants with alcohol use disorders to perform efficient error monitoring (Padilla et al., 2011). By comparison, to our knowledge, FRN component has not been evaluated in people with alcohol abuse or dependence.

The main goal of the current study was thus to propose a within-subject design to simultaneously assess the two PM components and evaluate the specific cerebral correlates of both inhibitory control (cognitive system) and decision-making (motivational system), as proposed in influential models (Dawe et al., 2004). Particularly, we assessed possible PM impairments in binge drinkers compared to non-binge drinkers and observed whether deficits appear equally strong or with dissociable effects. For this

purpose, we used a validated speeded Go/No-Go task (Vocat et al., 2008), evaluating inhibitory control and enabling to assess the integrity of error monitoring. To assess decision-making and PM based on external cues, we capitalized on the Balloon Analogue Risk Task (Fein and Chang, 2008; Lejuez et al., 2002), where a clear FRN is elicited. As previous studies showed PM impairments in alcohol-related disorders, with an increased ERN reported in alcohol-dependence, we hypothesized a larger ERN in binge drinkers, in line with the proposal that binge drinking and alcohol-dependence belong to the same continuum (e.g., Sanhueza et al., 2011). By comparison, as there is no available literature regarding FRN component in alcohol-use disorders, we relied on previous behavioral and neuroimaging studies targeting decision-making processes and reward sensitivity in binge drinking (e.g., Worbe et al., 2014) to hypothesize an increased FRN in binge drinkers.

2. Methods

2.1. Participants

A first screening interview was proposed to 3014 undergraduate students from the Université catholique de Louvain (Belgium). The screening was anonymous, participants answered by using a code (i.e. first letter of the mother's surname, first letter of the mother's first name, day and month of personal birth) and were directed to another online link at the end of the survey if they agreed to provide email address and take part in the experimental phase of the study. Only one investigator had access to these data and email addresses were only used for contacting participants. The screening assessed socio-demographic (age, gender, education level, and native language), psychological (pre-screening using binary assessment), and alcohol consumption (mean number of alcohol units per drinking occasion, mean number of drinking occasions per week, consumption speed in number of units per hour, mean number of alcohol units per week, drunkenness frequency) variables, an alcohol unit corresponding to 10 g of pure ethanol in Belgium. To be included in the study, participants had to meet the following criteria: native or fluent French speakers, at least 18 years old, no alcohol-dependence and no family history of alcohol-dependence, no positive psychological or neurological disorders, no current medication, no major medical problems, normal or corrected-to-normal vision, total absence of past or current drug consumption (excepting alcohol and tobacco). A subsample of 90 students was contacted and 40 accepted to take part in the study. They were recruited on the basis of a binge drinking score (Townshend and Duka, 2005) computed by using the following formula: [(4 ∗ consumption speed) + drunkenness frequency + (0.2 ∗ drunkenness percentage)]. Then, the sample was split into two groups by means of adapted cutoffs to categorize binge drinking habits: Twenty non-Binge Drinkers (non-BD; score $\leq$ 16; M = 7.22; SD = 4.17) and 20 Binge Drinkers (BD; score > 16; M = 25.90; SD = 14.11). Group comparisons were also performed on all alcohol-related variables and clearly supported the distinction between groups regarding alcohol consumption and binge drinking pattern (see Table 1). All participants (25 women) were aged between 18 and 27 years old and reported no alcohol consumption for the three days before the experiment. Before starting, participants filled in questionnaires assessing state-trait anxiety (State-Trait Anxiety Inventory, STAI; French validation: Bruchon-Schweitzer and Paulhan, 1993), depression (Beck Depression Inventory, BDI-II; French validation: Beck et al., 1998), and alcohol-related disorders (Alcohol Use Disorder Identification Test, AUDIT; French validation: Gache et al., 2005). The study protocol was approved by the ethics committee of the Université catholique de Louvain, and carried out according to the Declaration of Helsinki.

Table 1
Demographic and psychological measures [mean (SD)] for Binge Drinkers (BD) and non-Binge Drinkers (non-BD).

Variable	BD (n = 20)	non-BD (n = 20)
Demographic measures		
Age [ns]	20.25 (1.62)	21.20 (2.59)
Gender ratio (female/male) [ns]	12/8	13/7
Psychological measures		
Beck Depression Inventory [ns]	5.10 (3.35)	3.95 (3.24)
State anxiety inventory [ns]	30.60 (6.58)	31.05 (7.02)
Trait anxiety inventory [ns]	35.65 (7.18)	35.40 (6.46)
Alcohol consumption measures		
Alcohol Use Disorder Identification Test [*]	16.10 (5.29)	8.35 (5.53)
Total units per week [**]	18.85 (10.06)	9.6 (7.61)
Number of occasions per week [**]	2.60 (0.88)	1.65 (1.09)
Number of units per occasion [**]	7.15 (1.87)	4.45 (3.20)
Consumption speed (units per hour) [**]	4.25 (1.12)	1.60 (0.88)

Note. [ns] = Non-significant.
[*] $p < 0.01$.
[**] $p < 0.001$.

2.2. General procedure

The experiment was conducted in a unique experimental session. Participants provided written informed consent to take part in the study and were tested individually in a quiet laboratory. The questionnaires were administered first, using Qualtrics software (Qualtrics, LLC), followed by the two experimental tasks, in a counterbalanced order across participants. Participants were seated in front of a Dell E176FP (resolution: 1280 × 1024 pixels) at a 60 cm viewing distance and responded with their dominant hand. Participants were asked to minimize their eye blinks and movements while performing the tasks. The speeded Go/No-Go task was presented using E-Prime 2 Professional® (Psychology Software Tools, Pittsburgh, PA, USA) and started with a preliminary practice session (one trial of each condition) and the Balloon Analogue Risk Task was presented using Presentation software® (v. 17.2, Neurobehavioral Systems). Finally, participants were debriefed at the end of the experiment and received a compensation for their participation (20€).

2.3. EEG acquisition

Continuous EEG data were recorded at 1024 Hz (0–208 Hz bandwidth) using a 128-channel (pin-type) Biosemi ActiveTwo system referenced to the CMS-DRL ground (http://www.biosemi.com). All EEG processing was performed in BrainVision Analyzer (version 2). EEG data were band-pass filtered between 0.1 and 30 Hz (Butterworth Zero Phase Filters, 12 dB/oct). An additional notch Filter at 50 Hz was applied. Ocular artifact removal was carried out through an independent component analysis (ICA)-based strategy (Jung et al., 2000). EEG data were then re-referenced to the average of all signals. Due to artefacts in the EEG signal, there are missing data for three participants in the Balloon Analogue Risk Task.

2.4. Speeded Go/No-Go task

2.4.1. Stimuli

Visual stimuli (subtending a visual angle of 11.4° × 0.05°) were arrow symbols presented centrally on a white background (see Fig. 1). They always first appeared with a black color, and oriented either upward or downward. Then, they could turn either green or turquoise (these two colors were matched for luminance), with a similar or opposite orientation.

2.4.2. Procedure

During a trial, the black arrow was shown on the screen for a variable duration ranging from 1000 to 2000 ms. Then, the colored arrow remained on the screen until the response (for the go trials) or for a maximum of 1500 ms (for the no-go trials). Between the trials, a blank screen was presented during 500 ms, followed by a central fixation cross presented for another 500 ms. Participants had to respond as fast and accurately as possible each time the black arrow became green and kept the same orientation (go trials) while they had to refrain from responding when the black arrow became turquoise and/or changed its orientation (no-go trials). The experiment was divided into three sessions, each starting with a calibration block (14 trials; 10 go) following by two consecutive test blocks, each containing 60 trials (40 go). The full task included a total of 402 stimuli and trials presentation was randomized within blocks. During each calibration block, the mean reaction time (RT) for go trials was computed online and used to define an upper limit above which correct responses were considered as errors (slow hits) in the following test blocks. Participants were not informed about this procedure. The upper limit was set to 80% (for the first block) and to 90% (for the second and third blocks) of the mean RT of the corresponding calibration block. During the test blocks, participants received a feedback about their speed when they were too slow (“too late” displayed on the screen for 500 ms) and the percentage of correct responses was displayed on the top right of the screen. This procedure allowed the elicitation of a high number of errors and thus a reliable measure of error monitoring components. Therefore, the task induced two types of errors, the false alarms (i.e. classical inhibition failures) and the slow hits (i.e. too slow responses). Previous studies indeed showed that the slow hits were processed as errors and elicited a clear ERN (Scheffers and Coles, 2000), confirming the reliability of this slow hits procedure (e.g., Luu et al., 2000; Vocat et al., 2008). Fig. 1 illustrates the stimuli and task procedure (for more details, see Vocat et al., 2008). The experimental measures were: the number of false alarms (FA; when the participant answered to a no-go trial), the number of slow hits (SH; when the participant responded after the time limit), the number of fast hits (FH; when the participant responded before the time limit), and RT for the FA, SH, and FH.

2.4.3. EEG preprocessing

EEG segments were constructed that consisted of a 500 ms baseline and 500 ms following motor response onset, and were then baseline-corrected on the basis of the mean activity during the 500 ms prior to response onset. Algorithmic artifact rejection of voltage exceeding ±100 μV was followed by visual data inspection of segmented data in which segments with artifacts were manually rejected. Individual subject averages were constructed separately for each condition (i.e. FA, SH, and FH). ERN/CRN and Pe/Pc component peaks were quantified at three fronto-central electrodes: Cz, Fz, and FCz (Kóbor et al., 2015; Vocat et al., 2008).

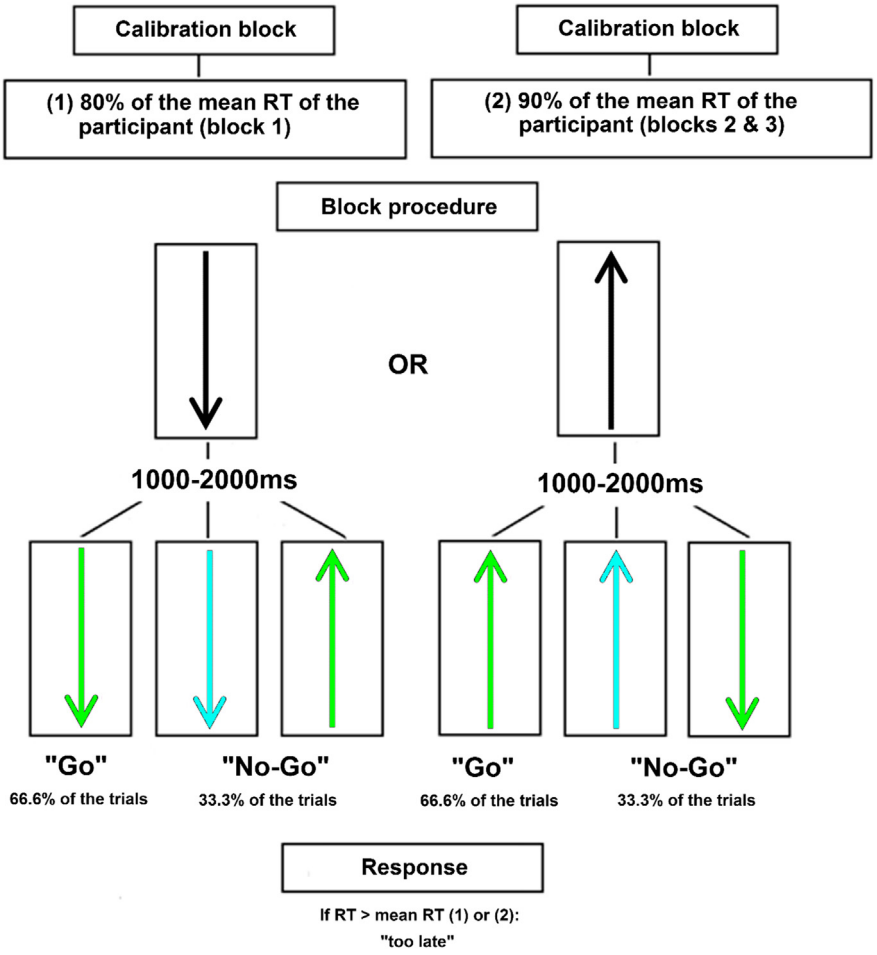

Fig. 1. Illustration of the Speeded Go/No-Go task, presenting stimuli types (black or color targets, oriented upward or downward) and the procedure, starting with a calibration block (calculating individual mean RT), followed by the test block structured as follows: (1) black arrow (1000–2000 ms); (2) variable inter-stimulus interval (500 ms); (3) central fixation cross (500 ms); (4) second color arrow (until the participant's response or 1500 ms), either green or turquoise and with similar or opposite orientation; (5) participant's response (i.e. pressing the space bar as fast as possible for the go trials, namely the green arrow with the same orientation). Adapted from Vocat et al. (2008). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

On the basis of the literature described in the introduction, as well as visual inspection of the grand-averaged data and the data from individual participants, the maximum negative peak value was detected from −50 to 80 ms for the ERN amplitude and latency, and the maximum positive peak value was identified from 80 to 300 ms for the Pe amplitude and latency.

2.5. Balloon analogue risk task

2.5.1. Stimuli

The visual stimulus (subtending a visual angle of 4.5°) consisted in a balloon appearing at the center of the screen. This balloon inflated each time participants pressed a response key (Y) on the keyboard, and the balloon may burst following each inflation.

2.5.2. Procedure

Participants were asked to pump a balloon in order to obtain a reward. A pump could either increase the size (i.e. visual angle increasing by 0.3°–0.7° depending on the amount of points earned) and value of the balloon (positive feedback) or burst the balloon (i.e. visual angle of 11.5° × 16.5°) leading to the loss of the cumulated points (negative feedback). Importantly, the probability of balloon burst increased with each pump and the maximum pump was set to 20 (the bursting probability was 1/18 after the third pump, 1/17 after the fourth pump and so on until the 20th pump). The cumulated score was computed as follows: 1 point for the first pump, 2 points for the second pump, 3 points for the third pump and so on. Participants could also decide to collect the earned points by pressing another response key (C). The task included 90 trials. When the balloon was shown on the screen, participants had unlimited time to pump or collect the accumulated points. A random delay of 1000–1200 ms was inserted between response (C or Y) and feedback stimulus. The negative (bursting) and positive (collecting) feedbacks appeared for 3000 ms on the screen and the following balloon was displayed 10 ms after. Fig. 2 illustrates the task (Kóbor et al., 2015). Behavioral measures (Fein and Chang, 2008; Kóbor et al., 2015; Lejuez et al., 2002) were: the mean adjusted number of pumps (the mean number of pumps for which the balloon did not explode), the number of bursting, the mean score before bursting, and the total score.

2.5.3. EEG preprocessing

EEG segments consisted of a 200 ms baseline and 800 ms following feedback onset, and were then baseline-corrected on the basis of the mean activity during the 200 ms prior to feedback onset. Algorithmic artifact rejection of voltage exceeding ±100 μV was followed by visual data inspection of segmented data in which segments with artifacts were manually rejected. Individual subject averages were constructed separately for each condition and difference waves were created by subtracting the positive feedback-locked waveform from the negative feedback-locked waveform (Fein and Chang, 2008; Kóbor et al., 2015). FRN and P3 component peaks were quantified at three fronto-central electrodes: Cz, Fz, and FCz (Kóbor et al., 2015). On the basis of the literature described in the introduction, as well as visual inspection of the grand-averaged data and the data from individual participants, the maximum negative peak value was detected for each participant separately from 200 to 300 ms for the FRN amplitude and latency, and the maximum positive peak value was identified from 300 to 600 ms for the P3 amplitude and latency.

2.6. Statistical analyses

All statistical analyses were performed using SPSS software package (version 21.0) and the following analytic plan was used. First, between-group comparisons were performed on demographic and psychological characteristics. Second, for the behavioral data from the speeded Go/No-Go task, a 2 × 2 repeated measures analysis of variance (ANOVA) with Group (non-BD, BD) as between-subjects factor and Error type (FA, SH) as within-subjects factor was computed separately for RT and Number of trials in each condition. Independent samples *t*-tests were also performed to compare correct answers (FH) between groups. Concerning electrophysiological data, two 2 × 2 × 3 ANOVAs (one for amplitude and one for latency) were performed for each components related to error monitoring (ERN and Pe) with Group (non-BD, BD) as between-subjects factor and Error type (FA, SH) as well as Electrode (Cz, Fz, FCz) as within-subjects factors. Moreover, two 2 × 3 ANOVAs (one for amplitude and one for latency) were performed for the components related to correct response (CRN and Pc), with Group (non-BD, BD) as between-subjects factor and Electrode (Cz, Fz, FCz) as within-subjects factor. For the Balloon Analogue Risk Task, four independent samples *t*-tests were performed on the behavioral data (the mean adjusted number of pumps, the number of bursting, the mean score before bursting, and the total score) and two 2 × 3 ANOVAs (one for amplitude and one for latency) were performed for each components related to feedback monitoring (FRN and P3), with Group (non-BD, BD) as between-subjects factor and Electrode (Cz, Fz, FCz) as within-subjects factor. As this study particularly focuses on the difference between BD, Student *t*-tests were performed when interactions with group were found. The post-hoc *t*-tests were computed on each electrode when appropriate. For the Go/No-Go task, comparisons were made on group for each error type (FA, SH) and on error type for each group (non-BD, BD).

3. Results

3.1. Demographic and psychopathological measures

Sociodemographic, psychological, and alcohol consumption data are reported in Table 1. No significant group difference was found for age [$t(38)=1.39$, $p = 0.172$], gender [$\chi^2(1, N = 40)=0.11$, $p = 0.744$], depressive symptoms [$t(38)=1.10$, $p = 0.277$], and anxiety (state: [$t(38)=0.21$, $p = 0.835$], trait: [$t(38)=0.12$, $p = 0.908$]).

3.2. Speeded Go/No-Go task

3.2.1. Behavioral data

Mean number of trials and reaction time for each condition are reported in Table 2.

3.2.1.1. Errors.

- Accuracy: There was a significant main effect of Error type [$F(1,38)=238.31$, $p < 0.001$] with more SH (M = 105.13, SD = 26.01) than FA (M = 42.08, SD = 18.86), but no significant main Group effect [$F(1,38)=0.92$, $p = 0.343$] or Group × Error interaction [$F(1,38)=0.42$, $p = 0.520$].
- RT: There was a main effect of Error type [$F(1,38)=287.24$, $p < 0.001$] with longer RT for SH (M = 369, SD = 37) than FA (M = 287, SD = 33), but no significant main Group effect [$F(1,38)=0.42$, $p = 0.520$] or Group × Error interaction [$F(1,38)=0.42$, $p = 0.524$].

3.2.1.2. Correct responses.

- Accuracy: No significant group effect was found for FH [$t(38)=0.96$, $p = 0.342$].
- RT: No significant group effect was found for FH [$t(38)=0.75$, $p = 0.456$].

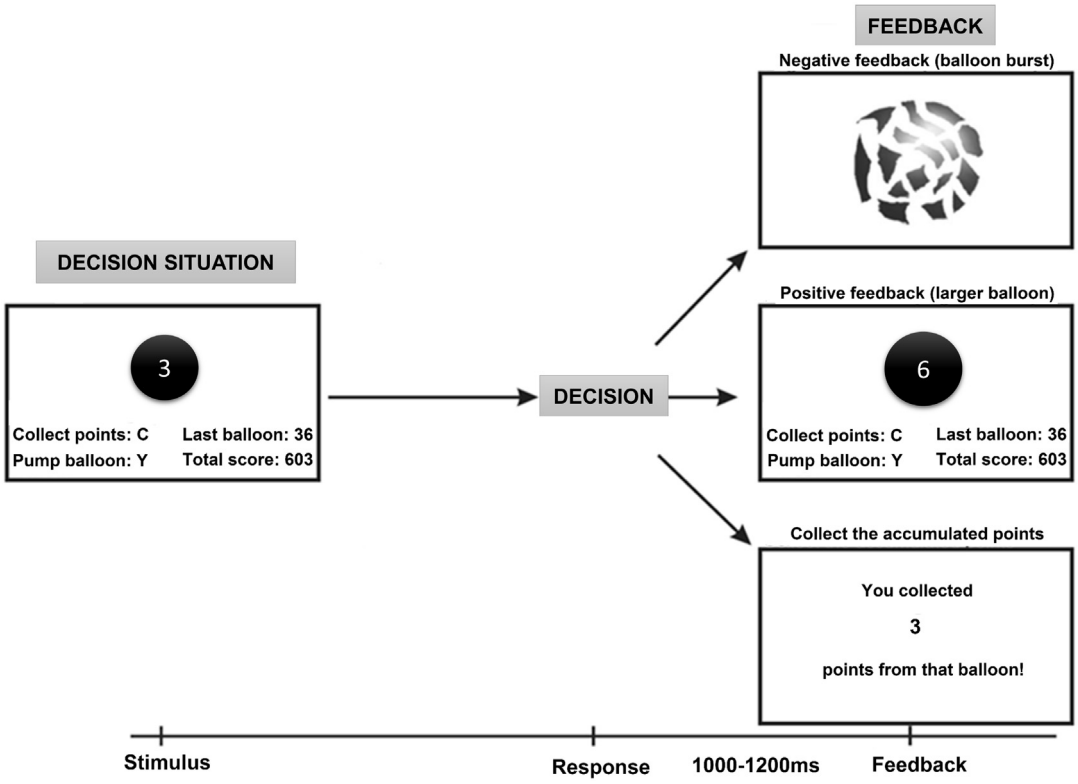

Fig. 2. Illustration of the Balloon Analogue Risk Task, presenting the different elements displayed on the screen: the central balloon with the number of points collected (zero at the beginning of each trial); the "Total score" depicting the points accumulated throughout the task; the "Last balloon" depicting the points collected from the previous balloon. Participant can choose to collect the accumulated points from the current balloon (response key "C"), or to pump the balloon further (response key "Y"). This second choice could have two consequences: a negative (balloon burst) or a positive (larger balloon with more points inside) feedback. Both points' accumulation or balloon burst end the trial. Adapted from Kóbor et al. (2015).

Table 2
Behavioral results [Mean (SD)] for the Go/No-Go task [number of trials and Reaction Times (RT; in ms) in each experimental condition] and the Balloon Analogue Risk Task (performance for each index) for Binge Drinkers (BD) and non-Binge Drinkers (non-BD).

	BD (n_1 = 20; n_2 = 19)		non-BD (n_1 = 20; n_2 = 18)	
Go/No-Go task	Number of trials	RT	Number of trials	RT
Slow Hits	109.30 (25.63)	364 (33)	100.95 (26.37)	374 (40)
False Alarms	43.60 (19.48)	285 (28)	40.55 (18.59)	289 (38)
Fast Hits	130.05 (25.91)	276 (25)	138.10 (27.02)	283 (34)
Balloon Analogue Risk Task	Score		Score	
Mean adjusted number of pump	7.39 (2.11)		7.26 (1.97)	
Number of bursting	33.83 (12.26)		31.32 (12.95)	
Mean score before bursting	21.93 (8.22)		20.51 (8.13)	
Total score	1798.44 (464.72)		1705.00 (570.42)	

Note. n_1 represents the number of participants in the Go/No-Go task and n_2 represents the number of participants in the Balloon Analogue Risk Task.

3.2.2. Electrophysiological data

Mean ERN and Pe peak amplitudes and latencies for each condition are reported in Table 3. All waveforms are illustrated in Fig. 3.

3.2.2.1. ERN.

- Amplitude: Main effects of Error type [$F(1,38)$=15.32, $p < 0.001$] and Electrode [$F(2,76)$=59.74, $p < 0.001$] were qualified by Error × Electrode [$F(2,76)$=4.84, $p = 0.010$] and Group × Error × Electrode [$F(2,76)$=3.80, $p = 0.027$] interactions. Post-hoc *t*-tests showed no significant difference between groups both for FA and SH (all $p \geq 0.188$) whatever the electrode, but ERN amplitude was larger for FA (M = −6.23, SD = 3.41) than SH (M = −5.06, SD = 3.11) at Fz electrode for BD [$t(19)$=2.39, $p = 0.027$] but not for non-BD (FA: M = −5.25, SD = 3.20; SH: M = −5.25, SD = 2.38) [$t(19)$ =0.01, $p = 0.996$]. There was no main effect of Group [$F(1,38)$ =0.51, $p = 0.481$] and other interactions were not significant (all $p \geq 0.915$).
- Latency: There was a main effect of Error type [$F(1,38)$=5.66, $p = 0.023$], showing longer latency for FA (M = 10, SD = 17) than SH (M = −2, SD = 30), and of Electrode [$F(2,76)$=3.62, $p = 0.031$], showing longer latency at Fz (M = 10, SD = 23) than FCz (M = 1, SD = 21) [$t(39)$=2.40, $p = 0.021$] and at Fz than Cz (M = 0, SD = 28) [$t(39)$=2.07, $p = 0.045$]. There was no main effect of Group [$F(1,38)$=0.71, $p = 0.791$] and other interactions were not significant (all $p \geq 0.438$).

Table 3
Amplitude (in microvolts) and Latency (in ms) [mean (SD)] of the ERN (−50 to 80 ms) and Pe (80–300 ms) components in each experimental condition at each electrode for Binge Drinkers (BD) and non-Binge Drinkers (non-BD).

ERN	Variable	Group	False alarms	Slow hits	Fast hits
Cz	Amplitude	BD	−1.02 (3.52)	0.19 (3.29)	0.61 (3.26)
		non-BD	−0.82 (2.54)	1.35 (2.05)	1.74 (2.29)
	Latency	BD	11 (27)	−11 (46)	−12 (49)
		non-BD	7 (27)	−6 (3)	−13 (37)
Fz	Amplitude	BD	−6.23 (3.41)	−5.06 (3.12)	−4.60 (3.23)
		non-BD	−5.25 (3.20)	−5.25 (2.38)	−4.55 (2.13)
	Latency	BD	15 (21)	11 (38)	17 (42)
		non-BD	15 (34)	0 (22)	0.3 (24)
FCz	Amplitude	BD	−4.69 (3.11)	−3.13 (3.31)	−1.95 (3.00)
		non-BD	−4.17 (4.27)	−2.62 (3.91)	−1.93 (3.82)
	Latency	BD	6 (17)	−4 (40)	−10 (25)
		non-BD	5 (27)	−2 (35)	−12 (21)
Pe					
Cz	Amplitude	BD	11.54 (5.58)	3.93 (3.26)	5.55 (4.23)
		non-BD	12.44 (3.95)	5.29 (2.27)	7.11 (2.35)
	Latency	BD	169 (28)	175 (59)	164 (71)
		non-BD	187 (38)	136 (58)	141 (63)
Fz	Amplitude	BD	8.36 (6.23)	1.20 (2.25)	2.79 (2.57)
		non-BD	8.52 (4.65)	2.10 (2.27)	3.71 (2.56)
	Latency	BD	158 (36)	195 (61)	233 (64)
		non-BD	168 (37)	236 (76)	225 (81)
FCz	Amplitude	BD	12.55 (5.53)	2.95 (2.31)	5.06 (3.21)
		non-BD	12.59 (4.44)	4.06 (2.55)	5.78 (2.87)
	Latency	BD	175 (33)	189 (68)	195 (73)
		non-BD	179 (49)	174 (80)	199 (89)

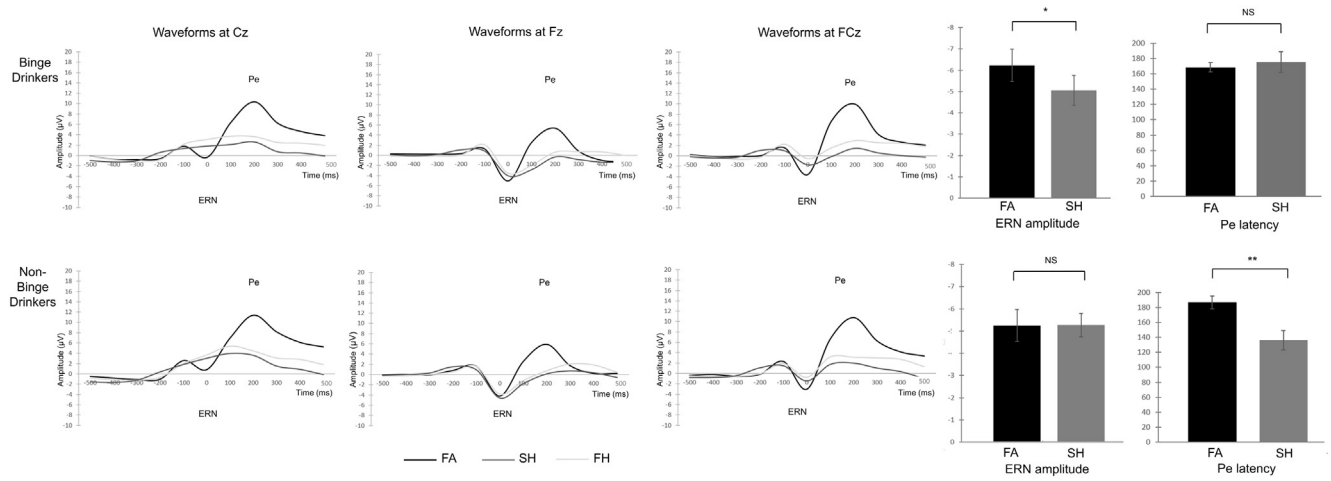

Fig. 3. Go/No-Go Task. Illustration of ERN and Pe components in the FA (black line), SH (dark grey line), and FH (light grey line) conditions for binge drinkers (upper part) and non-binge drinkers (lower part). This Figure illustrates the grand average ERP waveforms at electrodes Cz, Fz, and FCz respectively. The bar-charts depict: (1) the mean amplitude (μV) of the ERN component for FA and SH at Fz electrode (left part), showing a larger ERN amplitude for FA than for SH in binge drinkers, while no such difference was observed in non-binge drinkers and (2) the mean latency (ms) of the Pe component for FA and SH at Cz electrode (right part), showing a delayed Pe latency for SH than FA in non-binge drinkers while both errors types, failures and slow responses, are processed slowly in binge drinkers. $^{*}p < 0.05$, $^{**}p < 0.01$.

3.2.2.2. Pe.

– Amplitude: There was a main effect of Error type [$F(1,38)$ =199.17, $p < 0.001$], showing larger amplitude for FA (M = 10.00, SD = 4.26) than SH (M = 3.25, SD = 2.23), and Electrode [$F(2,76)$ =26.63, $p < 0.001$], showing lower amplitude for Fz (M = 5.04, SD = 3.38) than Cz (M = 8.30, SD = 3.61) [$t(39)$=7.26, $p < 0.001$] and FCz (M = 8.04, SD = 3.30) [$t(39)$=5.28, $p < 0.001$], which did not differ between them [$t(39)$=0.63, $p = 0.531$]. An Error type × Electrode interaction [$F(2,76)$=6.98, $p = 0.002$] was also found. There was no main Group effect [$F(1,38)$=0.64, $p = 0.428$] and other interactions were not significant (all $p \geq 0.494$).

– Latency: There was no main effect of Error type [$F(1,38)$=2.50, $p = 0.122$] nor Error × Group interaction [$F(1,38)$=1.13, $p = 0.294$]. There was no main effect of Group [$F(1,38)$=0.06, $p = 0.803$]. The main effect of Electrode [$F(2,76)$=5.87, $p = 0.004$] was qualified by Group × Electrode [$F(2,76)$=4.27, $p = 0.017$], Error × Electrode [$F(2,76)$=17.31, $p < 0.001$], and Group × Error × Electrode [$F(2,76)$=5.88, $p = 0.004$] interactions. Post-hoc *t*-tests between groups showed that Pe latency for SH at Cz was delayed in BD (M = 175, SD = 59) compared to non-BD (M = 136, SD = 58) [$t(38)$=2.13, $p = 0.040$]. Other group comparisons were not significant (all $p \geq 0.077$).

Table 4
Amplitude (in microvolts) and Latency (in ms) [mean (SD)] of the FRN (200–300 ms) and P3 (300–600 ms) components in the difference waves between positive and negative feedback at each selected electrode for Binge Drinkers (BD) and non-Binge Drinkers (non-BD).

Electrodes	Variable	Group	FRN	P3
Cz	Amplitude	BD	−8.47 (8.96)	24.48 (34.55)
		non-BD	−5.72 (3.84)	15.77 (4.13)
	Latency	BD	228 (10)	348 (55)
		non-BD	228 (13)	354 (26)
Fz	Amplitude	BD	−11.04 (9.52)	20.31 (25.37)
		non-BD	−7.43 (3.93)	12.27 (4.90)
	Latency	BD	239 (14)	332 (20)
		non-BD	240 (7)	338 (22)
FCz	Amplitude	BD	−12.48 (14.42)	27.63 (35.47)
		non-BD	−7.80 (5.40)	17.50 (5.85)
	Latency	BD	230 (9)	335 (31)
		non-BD	231 (9)	344 (25)

3.2.2.3. CRN.

- Amplitude: A main effect of Electrode was found [$F(2,76)=72.79$, $p<0.001$], showing larger amplitude at Fz (M = −4.57, SD = 2.70) than FCz (M = −1.94, SD = 3.39) [$t(39)=6.77$, $p<0.001$] and Cz (M = 1.18, SD = 2.84) [$t(39)=10.43$, $p<0.001$], as well as at FCz than Cz [$t(39)=6.56$, $p<0.001$], but Group effect [$F(1,38)=0.27$, $p=0.608$] and Group × Electrode interaction [$F(2,76)=0.89$, $p=0.416$] were not significant.
- Latency: There was a main effect of Electrode [$F(2,76)=6.17$, $p=0.003$], displaying longer latency for Fz (M = 9, SD = 35) than FCz (M = −11, SD = 23) [$t(39)=3.54$, $p=0.001$] and Cz (M = −13, SD = 43) [$t(39)=2.64$, $p=0.012$], with no difference between Cz and FCz [$t(39)=0.26$, $p=0.794$], but there was no Group effect [$F(1,38)=0.76$, $p=0.389$] nor Group × Electrode interaction [$F(2,76)=0.83$, $p=0.441$].

3.2.2.4. Pc.

- Amplitude: A main effect of Electrode was found [$F(2,76)=26.26$, $p<0.001$], showing larger amplitude at Cz (M = 6.33, SD = 3.47) than FCz (M = 5.42, SD = 3.03) [$t(39)=2.13$, $p=0.039$] and Fz (M = 3.25, SD = 2.57) [$t(39)=6.02$, $p<0.001$], as well as at FCz than Fz [$t(39)=6.26$, $p<0.001$], but there was no Group effect [$F(1,38)=1.72$, $p=0.198$] nor Group x Electrode interaction [$F(2,76)=0.50$, $p=0.609$].
- Latency: There was a main effect of Electrode [$F(2,76)=21.12$, $p<0.001$], showing longer latency for Fz (M = 229, SD = 72) than FCz (M = 197, SD = 80) [$t(39)=3.03$, $p=0.004$] and Cz (M = 152, SD = 67) [$t(39)=6.01$, $p<0.001$] and between FCz and Cz [$t(39)=3.75$, $p=0.001$], but there was no Group effect [$F(1,38)=0.24$, $p=0.629$] nor Group × Electrode interaction [$F(2,76)=0.67$, $p=0.516$].

3.3. Balloon analogue risk task

3.3.1. Behavioral data

Mean performance for each behavioral measure are reported in Table 2.

There was no main Group effect for the mean adjusted number of pumps [$t(35)=0.18$, $p=0.858$], the number of bursting [$t(35)=0.61$, $p=0.548$], the mean score before bursting [$t(35)=0.53$, $p=0.599$], and the total score [$t(35)=0.55$, $p=0.590$].

3.3.2. Electrophysiological data

Mean amplitudes and latencies for FRN and P3 are reported in Table 4. All waveforms are illustrated in Fig. 4.

3.3.2.1. FRN.

- Amplitude: A main effect of Electrode was found [$F(2,70)=8.53$, $p<0.001$], showing larger amplitude at FCz (M = −10.21, SD = 11.11) than Cz (M = −7.13, SD = 7) [$t(36)=3.80$, $p=0.001$] and at Fz (M = −9.28, SD = 7.48) than Cz [$t(36)=3.40$, $p=0.002$], with no difference between FCz and Fz [$t(36)=1.14$, $p=0.262$], but the main effect of Group [$F(1,35)=1.86$, $p=0.181$] and the Group × Electrode interaction [$F(2,70)=0.81$, $p=0.448$] were not significant.
- Latency: A main effect of Electrode was found [$F(2,70)=23.20$, $p<0.001$], showing longer latency for Fz (M = 240, SD = 11) than FCz (M = 230, SD = 9) [$t(36)=4.94$, $p<0.001$] and Cz (M = 228, SD = 11) [$t(36)=6.18$, $p<0.001$], with no difference between FCz and Cz [$t(36)=1.58$, $p=0.124$]. There was no main effect of Group [$F(1,35)=0.05$, $p=0.819$] nor Group × Electrode interaction [$F(2,70)=0.05$, $p=0.950$].

3.3.2.2. P3.

- Amplitude: A main effect of Electrode was found [$F(2,70)=11.08$, $p<0.001$], showing larger amplitude at FCz (M = 22.70, SD = 25.91) than Cz (M = 20.24, SD = 24.99) [$t(36)=3.32$, $p=0.002$] and Fz (M = 16.40, SD = 18.70) [$t(36)=4.35$, $p<0.001$], and at Cz than Fz [$t(36)=2.36$, $p=0.024$]. However, the main Group effect [$F(1,35)=1.43$, $p=0.241$] and the Group × Electrode interaction [$F(2,70)=0.31$, $p=0.732$] were not significant.
- Latency: There was a main effect of Electrode [$F(2,70)=4.82$, $p=0.011$], showing a longer latency at Cz (M = 351, SD = 43) than Fz (M = 335, SD = 21) [$t(36)=2.69$, $p=0.011$] but other comparisons were not significant (all $p \geq 0.066$). There was no main Group effect [$F(1,35)=0.68$, $p=0.417$] nor Group × Electrode interaction [$F(2,70)=0.04$, $p=0.964$].

4. Discussion

This study simultaneously investigated the electrophysiological correlates of two crucial processes involved in binge drinking, namely inhibitory control and decision-making, through the exploration of internal and external PM sources. Results revealed no behavioral difference between BD and non-BD. At the electrophysiological level, the early detection of errors, as indexed by ERN and Pe components, was modified among BD, while they demonstrated preserved feedback processing (FRN-P3). Globally, these results highlighted changes in the electrophysiological activities related to the cognitive component, namely when BD performed inhibitory control.

Regarding the results obtained in the speeded Go/No-Go task evaluating inhibitory control, corresponding to the first factor of

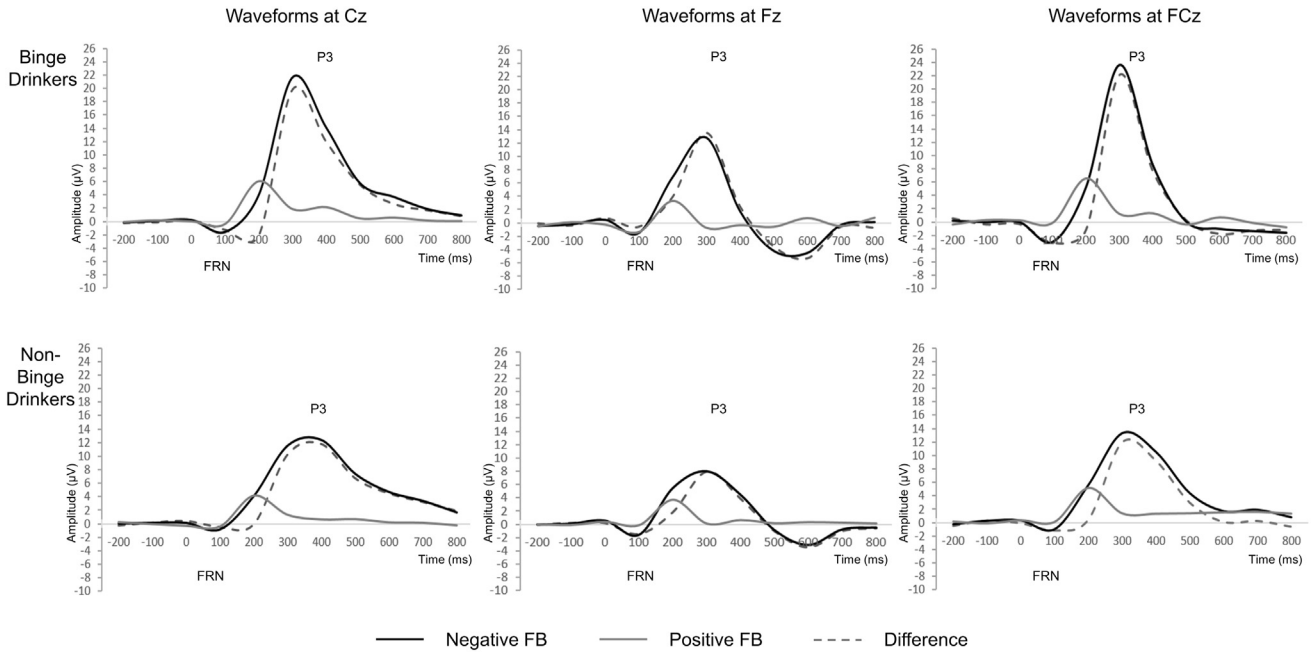

Fig. 4. Balloon Analogue Risk Task. Illustration of FRN and P3 components in the negative (black line) and positive (grey line) feedback conditions, as well as in the difference waveform (dotted line; i.e. negative feedback – positive feedback) for binge drinkers and non-binge drinkers. This Figure illustrates the grand average ERP waveforms at electrodes Cz, Fz, and FCz respectively.

the impulsivity model (Dawe and Loxton, 2004; Dawe et al., 2004), and especially internal error processing, both groups displayed the classical activation pattern associated with internal sources processing. Group differences were observed at the electrophysiological level and related to FA and SH, the two conditions associated with error processing (Luu et al., 2000; Vocat et al., 2008). Analyses on correct responses (i.e. FH) showed no significant difference between groups, suggesting that performance of BD and non-BD only differed during error processing. The first error type (FA) was the classical commission error (i.e. producing a motor response when requested to inhibit it) which elicited a clear ERN. It required detecting the error made (as no indication was given on the screen), and activated subsequent remedial processes to better inhibit the dominant response. The second error type (SH) represented a correct categorization but beyond the response time limit. Participants were precisely informed that too slow responses would be classified as errors and experienced a decrease of their performance when responding too slowly. SH were clearly indicated during the task, which ensured a correct detection of errors and elicited a clear ERN. SH mobilized remedial processes to increase speed in order to get back in the requested time window. The current findings show that BD presented modified cerebral activity related to the processing of both error types.

First, converging with our hypotheses, binge drinking was associated with an increased ERN amplitude in the absence of behavioral counterpart. This is not indexed by a direct group difference but by a more important activity for FA compared to SH in the BD group. BD thus demonstrated a stronger cerebral activity during the detection of FA, in line with previous results in abstinent alcohol-dependent patients (Padilla et al., 2011), while studies conducted among heavy drinkers compared with control ones showed mixed results. Findings also support the continuum hypothesis (Lannoy et al., 2014; Maurage et al., 2013; Sanhueza et al., 2011) suggesting that binge drinking and alcohol-dependence would constitute two successive steps of the same phenomenon, thus leading to analogous impairments and similar brain deficits. Earlier studies conveyed that this increased amplitude could reflect a compensatory activity, which is in line with earlier data in binge drinking (Campanella et al., 2013). This compensatory hypothesis has been described in previous neuroimaging (Maurage et al., 2013; Schweinsburg et al., 2010) and electrophysiological studies, these latter reporting larger N2 (Crego et al., 2012, 2009; Smith et al., 2015) and P3 (Lopez-Caneda et al., 2013) amplitudes in binge drinking, related to the higher mobilization of attentional and executive resources to efficiently perform an experimental task. Regarding the current findings, this interpretation would suggest that BD might compensate inhibition difficulties by increasing brain activity during error detection (ERN), thus leading to a preserved behavioral outcome. Nevertheless, research in the two last decades has demonstrated that PM encompasses more complex processes, notably indicating that the ERN not only reflects error detection but also emotional significance of errors (Luu et al., 2000). A recent review (Koban and Pourtois, 2014) supported that PM constantly interacts with affective and social mechanisms. It thus involves specific appraisal monitoring processes aiming at "automatically" assigning a valence to actions (i.e. qualifying an incorrect response as negative and a correct response as positive) in order to correctly adjust subsequent behaviors (Aarts et al., 2012, 2013). Interestingly, a larger ERN amplitude can be observed when this assignation is not appropriately performed, suggesting reduced affective labelling of incorrect and correct actions (Aarts and Pourtois, 2015). In this perspective, BD would have a difficulty to consider their errors as bad when they respond to a no-go trial, which could therefore impair the following learning and adjustment process. This interpretation is consistent with the fact that higher ERN is observed only for FA, the errors which are not explicitly signaled during the task. Finally, an increased ERN in alcohol-related disorders was also observed among individuals presenting higher anxiety, therefore proposed as a possible explanation of this increased activity (Schellekens et al., 2010). However, in the current study, the first analyses confirmed that groups did not differ for anxiety or depression, which can thus not explain the increased ERN amplitude.

Second, complementarily to these ERN results, BD demonstrated delayed Pe latency for the processing of late answers (i.e. SH), indicating slower error processing than non-BD. Therefore, while behavioral results attest that BD can perform as efficiently as non-BD, electrophysiological findings suggest that the conscious appraisal of errors or the processing of their motivational significance are slower, reflecting that BD may need more time to adjust their behavior after a late answer. This result is consistent with the study of Franken et al. (2017), interpreting the Pe effect as an abnormal attribution of salience to errors made. This altered Pe latency, representing slower brain activity during PM, is in line with the slowing down observed for classical event-related components in previous studies among BD (Maurage et al., 2009) and alcohol-dependent individuals (Campanella et al., 2010).

Altogether, this slower Pe processing and this increased ERN amplitude suggest that BD do not have inhibition impairments but greater difficulty in processing their errors (FA and SH), both at the automatic and more conscious level.

Regarding the results obtained in the Balloon Analogue Risk Task evaluating decision-making, corresponding to the second factor of the impulsivity model (Dawe and Loxton, 2004; Dawe et al., 2004), and especially feedback processing, both groups also presented the classical electrophysiological pattern associated with external sources. Findings did not reveal group differences, suggesting an absence of significant change for feedback processing according to binge drinking pattern. BD present similar ability to process an external feedback and to regulate their behavior than non-BD. The present study thus emphasizes a dissociation between preserved processing of external sources and altered detection of internal sources in PM.

Finally, it has to be underlined that this study was conducted by taking into account the potential influence of psychopathological comorbidities. The cross-sectional nature of our design does not allow to draw any causal conclusion about the relationship between binge drinking and PM impairments, but the strict group matching supports the proposal that the observed differences are not related to acute alcohol consumption, other-substances consumption, family history of alcohol-dependence, or psychopathological disorders. Nevertheless, future research should confirm these results obtained with BD and non-BD groups by comparing different binge drinking levels but also BD to control non-drinking participants. Moreover, future studies should also further investigate the electrophysiological correlates of late answers because, in this paradigm, SH was always followed by an indication specifying that the response was too slow. As this study used time-locked response analyses, it has to be mentioned that the processing of this indication could step in the analyses of SH.

5. Conclusion

This study was the first to jointly explore the cerebral correlates of both cognitive and motivational impulsivity systems and to target the impaired subcomponents of PM in binge drinking. Results indicate that the behavioral performance did not significantly differ between groups. However, the present study shows that PM, crucial for cognitive control in everyday life, is impaired in BD for inhibitory control, when internal cues guide behavior. In a more global view considering both systems, electrophysiological findings suggest that binge drinking is mainly related to cognitive level modifications. According to previous results, it could be hypothesized that the motivational system is centrally involved in the explanation of alcohol consumption (versus non consumption) whereas the cognitive system is central for the understanding of the alcohol consumption pattern (i.e. modification in binge drinking compared to alcohol drinkers without binge drinking habits). This impairment could lower their control on alcohol consumption and thus favor the increase of excessive drinking habits.

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Conflict of Interest Statement

None of the authors have potential conflicts of interest to be disclosed.

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