Cognitive, Affective, & Behavioral Neuroscience (2018) 18:1076–1088
https://doi.org/10.3758/s13415-018-0623-3

Electrophysiological correlates of emotional crossmodal processing in binge drinking

Séverine Lannoy[1] · Fabien D'Hondt[2,3] · Valérie Dormal[1] · Marine Blanco[1] · Mélanie Brion[1] · Joël Billieux[4] · Salvatore Campanella[5] · Pierre Maurage[1]

Published online: 9 August 2018

Abstract

Emotional crossmodal integration (i.e., multisensorial decoding of emotions) is a crucial process that ensures adaptive social behaviors and responses to the environment. Recent evidence suggests that in binge drinking—an excessive alcohol consumption pattern associated with psychological and cerebral deficits—crossmodal integration is preserved at the behavioral level. Although some studies have suggested brain modifications during affective processing in binge drinking, nothing is known about the cerebral correlates of crossmodal integration. In the current study, we asked 53 university students (17 binge drinkers, 17 moderate drinkers, 19 nondrinkers) to perform an emotional crossmodal task while their behavioral and neurophysiological responses were recorded. Participants had to identify happiness and anger in three conditions (unimodal, crossmodal congruent, crossmodal incongruent) and two modalities (face and/or voice). Binge drinkers did not significantly differ from moderate drinkers and nondrinkers at the behavioral level. However, widespread cerebral modifications were found at perceptual (N100) and mainly at decisional (P3b) stages in binge drinkers, indexed by slower brain processing and stronger activity. These cerebral modifications were mostly related to anger processing and crossmodal integration. This study highlights higher electrophysiological activity in the absence of behavioral deficits, which could index a potential compensation process in binge drinkers. In line with results found in severe alcohol-use disorders, these electrophysiological findings show modified anger processing, which might have a deleterious impact on social functioning. Moreover, this study suggests impaired crossmodal integration at early stages of alcohol-related disorders.

Keywords Binge drinking · Emotion · Cross-modality · Event-related potentials · Alcohol-use disorders

Electronic supplementary material The online version of this article (https://doi.org/10.3758/s13415-018-0623-3) contains supplementary material, which is available to authorized users.

✉ Pierre Maurage
pierre.maurage@uclouvain.be

[1] Laboratory for Experimental Psychopathology, Psychological Science Research Institute, Université Catholique de Louvain, Louvain-la-Neuve, Belgium

[2] Sciences Cognitives et Sciences Affectives (SCALab), Université de Lille, Lille, France

[3] CHU Lille, Clinique de Psychiatrie (CURE), Lille, France

[4] Addictive and Compulsive Behaviours Lab, Institute for Health and Behaviour, University of Luxembourg, Esch-sur-Alzette, Luxembourg

[5] Laboratoire de Psychologie Médicale et d'Addiction, Neuroscience Institute, Université Libre de Bruxelles, Bruxelles, Belgium

Introduction

Human beings are continuously confronted with a wide range of simultaneous inputs coming from different sensorial modalities, which have to be integrated into a single comprehensive representation (Driver & Spence, 2000). This phenomenon, called "crossmodal integration", is essential, because it allows for adequate understanding of the surrounding environment through complex associative processes, thus promoting adapted and efficient behavioral responses. Numerous studies have investigated crossmodal processing and its cerebral correlates in healthy (see Campanella & Belin, 2007, for a review) and psychopathological (see Maurage & Campanella, 2014, for a review) populations, confirming its crucial role in daily life and interpersonal relationships. Specifically, emotional crossmodal processing has been explored in severe alcohol-use disorders, showing altered

crossmodal integration at behavioral and brain levels (Maurage, Campanella, Philippot, Pham, & Joassin, 2007; Maurage, Joassin, et al., 2013; Maurage, Philippot, et al., 2008). To further extend this research line, studies have proposed exploring crossmodal processing in other alcohol-related disorders and centrally in binge drinking (Maurage & Campanella, 2014), an excessive and deleterious alcohol consumption pattern that is especially frequent in young people (Courtney & Polich, 2009) and is characterized by a specific alternation between episodes of strong intake and withdrawal (Stephens & Duka, 2008). More precisely, according to the National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2004), binge drinking is defined as a blood alcohol concentration level of 0.08 g/dl, reached after a consumption episode of at least four (for women) or five (for men) drinks within 2 hours. This definition has been adapted in most European countries (in which a standard alcohol dose contains 10 g of pure ethanol) from studies indicating that a blood alcohol concentration level of 0.08% is obtained after the consumption of at least six drinks (López-Caneda, Rodríguez Holguín, Corral, Doallo, & Cadaveira, 2014). Beyond this classification, some studies have proposed computing a binge drinking score based on consumption speed and drunkenness frequency to target the specific alternation found in binge drinking between intense intake and withdrawal (Townshend & Duka, 2002). Notably, binge drinking has been found to be associated with cognitive impairments that are comparable (although to a lesser extent) to those reported in severe alcohol-use disorders (Hartley, Elsabagh, & File, 2004) and can be viewed as a first step towards alcohol-related disorders (Bonomo, Bowes, Coffey, Carlin, & Patton, 2004). A wide range of cognitive abilities have been explored in binge drinking. Nonetheless, although some studies have focused on affective processes in unimodal ways (Maurage et al., 2009), these abilities have not been deeply examined; crossmodal integration could bring crucial findings to the understanding of affective processes. Consequently, we investigated the behavioral and brain correlates of emotional crossmodal processing in binge drinking to offer an in-depth view of the deficits associated with this consumption pattern.

Binge drinking was initially considered as a harmless consumption mode, but it is now established that this drinking pattern is related to various behavioral deficits in high-level cognitive processes, such as memory and executive functions (Goudriaan, Grekin, & Sher, 2011; Heffernan et al., 2010; Parada et al., 2012), as well as in attentional abilities (Hartley et al., 2004). Several studies also indicated brain dysfunctions at the electrophysiological and neuroimaging levels in the absence of behavioral deficits among binge drinkers (for reviews, see Hermens et al., 2013; Maurage, Petit, & Campanella, 2013; Petit et al., 2014). It has moreover been proposed that this preserved behavioral performance, coupled with increased cerebral recruitment, indicates the presence of brain compensation mechanisms (Crego et al., 2012; Lopez-Caneda et al., 2013). Alongside these cognitive consequences, there is a serious need to explore emotional abilities in binge drinking, because they probably play a key role in the development and maintenance of alcohol-related disorders (Brion et al., 2016; D'Hondt et al., 2014). Recently, to respond to this need and consider the importance of multimodal integration by means of ecological paradigms, researchers have investigated the crossmodal processing of emotions among binge drinkers who showed good efficiency at the behavioral level (Lannoy, Dormal, Brion, Billieux, & Maurage, 2017). Neuroscience studies assessing the processing of affective voices in binge drinking have, however, focused on cerebral modifications observable through slower event-related components associated with perceptual (P100) and executive/decisional (P3b, N200) processes (Maurage et al., 2009), as well as on the reorganization of brain activity showing reduced temporal and increased frontal activations (Maurage, Bestelmeyer, Rouger, Charest, & Belin, 2013). Because reliable evidence has shown the brain areas and specific processing related to multisensorial integration (Calvert et al., 2001), we proposed to go beyond the behavioral outcomes and explore, for the first time, the brain correlates of emotional crossmodal processing in binge drinking.

In the current work, we explored the event-related potentials (ERPs) associated with emotional and crossmodal processing in binge drinking. An emotion detection task was used that required the identification of happiness and anger in visual and auditory modalities displayed in unimodal or crossmodal conditions. This paradigm was in line with studies conducted in severe alcohol-use disorders (Maurage et al., 2007). Specifically, we explored the different steps associated with stimulus processing, from early perceptual (P100 for visual, N100 for auditory) and modality-related (N170 for visual, N200 for auditory) to later decisional (P3b) processes. Regarding electrophysiological explorations, a first analytic step compared emotional processing across sensorial modalities in binge drinkers and control participants, and a second analytic step focused on subtraction waveforms (Teder-Sälejärvi et al., 2002) to isolate the specific crossmodal-related activity compared with unimodal activities [i.e., crossmodal – (unimodal face + unimodal voice)] and observe whether binge drinking is characterized by a specific impairment of crossmodal integration, in accordance with what has been found in severe alcohol-use disorders (Maurage, Philippot et al., 2008). We expected to find, in agreement with the literature (Lannoy, Dormal et al., 2017; Maurage, Bestelmeyer et al., 2013; Maurage et al., 2012), no significant difference between groups at the behavioral level but hypothesized the presence of widespread cerebral modifications among binge drinkers from early perceptive to late decisional processing stages. More precisely, we postulated cerebral compensation indexed by an increased amplitude in ERP components in binge drinkers compared with control participants. Concerning the proposal that binge drinking and severe alcohol-use disorders belong to

the same continuum (Enoch, 2006), we also hypothesized specific difficulty in crossmodal integration among binge drinkers, characterized by electrophysiological modifications related to complex crossmodal integration.

Method

Participants

A preliminary screening questionnaire was sent to the student community from the Université catholique de Louvain (Belgium) to assess sociodemographic characteristics (age, gender, education level, and native language), psychological symptoms (e.g., anxiety, depression), and alcohol consumption (mean number of alcohol units per drinking occasion [an alcohol unit in Belgium containing 10 g of pure ethanol], mean number of drinking occasions per week, consumption speed in number of units per hour, mean number of alcohol units per week, and drunkenness frequency). To compare binge drinkers, moderate drinkers, and nondrinkers, we selected specific drinking patterns among the participants on the basis of both their binge drinking score (Townshend & Duka, 2002) and the number of alcohol doses that they consumed per occasion (López-Caneda et al., 2014). The binge drinking score was computed according to the following

Table 1 Demographic and psychological measures for binge drinkers (BDs), moderate drinkers (MDs), and nondrinkers (NDs)

Variable	BDs (*n* = 17)	MDs (*n* = 17)	NDs (n = 19)
Demographic measures			
Age	20.18 (1.55)	21.00 (2.72)	20.37 (2.81)
Gender ratio (female/male)	7/10	8/9	8/11
Belgian students (%)	100	88.24	84.21
Tobacco use (*n*)	2	0	0
Handedness (right-handed/left-handed)	16/1	16/1	17/2
Psychopathological measures			
Beck Depression Inventory	6.00 (2.85)	5.24 (4.04)	5.53 (4.21)
STAI state anxiety inventory	28.76 (7.08)	32.06 (8.69)	32.84 (8.46)
STAI trait anxiety inventory	54.18 (2.81)	51.59 (5.43)	50.16 (9.48)
Psychological measures			
Emotional regulation			
Acceptance	12.88 (2.50)	12.47 (3.39)	12.89 (3.49)
Positive refocusing	11.59 (2.72)	11.12 (2.67)	10.95 (2.76)
Refocus on planning	14.47 (3.39)	13.88 (2.91)	14.63 (3.32)
Positive reappraisal	12.35 (3.76)	12.24 (3.23)	13.32 (3.76)
Putting into perspective	11.35 (2.52)	11.47 (3.11)	12.68 (3.20)
Catastrophizing	7.41 (2.69)	6.65 (1.80)	7.42 (1.87)
Rumination	10.41 (3.16)	10.76 (2.99)	11.26 (2.73)
Self-blame	10.24 (2.14)	10.35 (2.32)	11.37 (2.95)
Blaming others	9.18 (2.30)	8.59 (1.73)	9.16 (2.59)
Emotional reactivity	46.00 (12.67)	42.06 (14.71)	51.63 (15.45)
Emotional intensity	21.59 (6.66)	19.59 (7.50)	24.47 (7.87)
Emotional sensitivity	15.29 (4.59)	12.59 (4.91)	17.37 (5.89)
Emotional persistence	9.12 (2.67)	9.88 (3.22)	9.79 (3.01)
Alcohol consumption measures			
Alcohol Use Disorders Identification Test	16.18 (4.42)	2.41 (4.35)	0
Binge drinking score	37.90 (27.43)	3.82 (4.03)	0
Total alcohol units per week	27.06 (12.01)	2.47 (5.50)	0
Number of occasions per week	2.94 (0.75)	0.41 (0.87)	0
Number of alcohol units per occasion	9.17 (3.14)	1.15 (2.05)	0
Consumption speed (units per hour)	3.82 (1.38)	0.77 (0.73)	0

Data are presented as mean (*SD*) except where otherwise indicated. STAI = State-Trait Anxiety Inventory.

formula: (4 × consumption speed) + number of drunkenness episodes + (0.2 × percentage of drunkenness episodes). The cutoff scores for participants' responses (Townshend & Duka, 2005) were adapted for Belgium's alcohol unit measurement, as reliably proposed in previous studies (Lannoy, D'Hondt, Dormal, Billieux, & Maurage, 2017; Lannoy, Dormal, et al., 2017) and reinforced by other alcohol-related measures (Table 1). For all participants, the number of drinking occasions per week also was limited to four to exclude the presence of severe alcohol-use disorders in our sample. Participants who agreed to take part in the experimental phase of the study were contacted if they fulfilled the following criteria: no severe alcohol-use disorders and no family history of alcohol-use disorders, no positive self-reported psychological or neurological disorders, no current medication that has an impact on vigilance, no major medical problems, corrected-to-normal visual abilities, normal auditory abilities, and total absence of past or current drug consumption (except for alcohol and tobacco). We based these exclusion criteria on self-reported items (i.e., binary "yes or no" questions, such as "Does a member of your family present current or past severe alcohol-use disorders?") in order to select the initial group of participants. We then used several validated questionnaires at the beginning of the experimental phase to confirm this preliminary recruitment by assessing participants' state-trait anxiety (State-Trait Anxiety Inventory; Spielberger et al., 1983), depressive symptoms (Beck Depression Inventory; Beck, Steer, & Brown, 1996), emotional reactivity (Emotion Reactivity Scale; Lannoy et al., 2014), emotional regulation (Cognitive Emotion Regulation Questionnaire; Jermann et al., 2006), and alcohol-related disorders (Alcohol Use Disorders Identification Test; Babor et al., 2001). Although participants who reported clinically significant psychological disorders were excluded in the first recruitment step, the results obtained with the Beck Depression Inventory led us to exclude six additional participants (3 binge drinkers, 2 moderate drinkers, and 1 teetotaler) who presented significant depressive symptoms (score >12; Beck et al., 1996). The final sample included 17 binge drinkers (BDs; binge drinking score ≥16, number of doses consumed per occasion ≥6, consumption speed ≥2, number of drinking occasions per week between 2 and 4); 17 moderate drinkers (MDs; binge drinking score between 1 and 12, number of doses consumed per occasion ≤3, consumption speed between 0.33 and 2, number of drinking occasions per week ≤3); and 19 nondrinkers (NDs; binge drinking score = 0; number of doses consumed per occasion = 0, consumption speed = 0, number of drinking occasions per week = 0). Two control groups, composed of moderate drinkers and teetotalers, were selected in this study. Notably, the final sample did not differ from the initially selected sample (n = 59, including the 6 excluded participants) for age, $t(57) = 0.80$, $p = 0.43$; gender, $\chi^2(1, N = 59) = 0.55$, $p = 0.46$; Alcohol Use Disorders Identification Test, $t(57) = 1.36$, $p = 0.18$; and binge drinking score, $t(57) = 0.77$, $p = 0.45$. All participants (BDs: 7 women, MDs: 8 women, NDs: 8 women) were fluent French speakers between ages 18 and 29 years (BDs: $M = 20.18$, $SD = 1.55$; MDs: $M = 21$, $SD = 2.72$; NDs: $M = 20.37$, $SD = 2.81$).

Task description and procedure

The experimental task was an emotional crossmodal task used in a previous study (Lannoy, Dormal, et al., 2017) in which participants had to detect the emotional valence (happiness or anger) of facial and vocal stimuli by pressing the response key with their dominant hand. Stimuli were presented in separate (unimodal) or simultaneous (crossmodal) conditions; the crossmodal trials displayed either identical (crossmodal congruent) or different (crossmodal incongruent) emotions. The task included 100 unimodal trials (i.e., 50 faces, 50 voices), 200 crossmodal congruent trials (i.e., 100 faces, 100 voices), and 200 crossmodal incongruent trials (i.e., 100 faces, 100 voices). The task was administrated in three blocks (i.e., face unimodal, voice unimodal, crossmodal). In the last crossmodal block, congruent and incongruent trials were randomly presented; the first part required the participant to make a decision by focusing on facial stimuli, and the second part (appearing after a break) required the participant to focus on vocal stimuli. In all conditions, a fixation cross (500 ms) was presented before the target stimulus, which consisted of a face, a voice, or both (500 ms). The target was followed by a blank screen (2,000 ms), and the participant had 2,500 ms to answer. The experiment was conducted in a quiet and dimly lit room; participants were placed 60 cm from a Dell E176FP monitor (resolution: 1,280 × 1,024 pixels). Before the experiment, the participants completed questionnaires that assessed psychopathological variables. We also ensured, by means of self-reported measures, that participants had abstained from alcohol in the 3 days preceding the experiment to avoid any influence of acute alcohol consumption. They then performed the emotional crossmodal task while their EEG was recorded. As in the previous study (Lannoy, Dormal, et al., 2017), the two first blocks were presented in a pseudorandomized order across participants (i.e., intra-unimodal randomization) and were always followed by the crossmodal block to end the experiment. Accuracy scores (percentage of correct responses) and reaction times (for correct responses) were used for behavioral data. At the end of the experiment, participants were debriefed and received compensation for their participation (20 €). The study

protocol and procedure were approved by the ethics committee of the Psychological Science Research Institute of the Université catholique de Louvain and the study was performed according to the Declaration of Helsinki.

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 (0–208 Hz bandwidth). EEG processing was performed in BrainVision Analyzer (version 2) with the following procedure: (1) band-pass filter between 0.1 and 30 Hz (Butterworth Zero Phase Filters, 12 dB/oct); (2) notch filter at 50 Hz; (3) independent component analysis for ocular artifacts (Jung et al., 2000) conducted with the semiautomatic procedure proposed by BrainVision Analyzer (consisting of a first automatic detection, and then confirmed and validated by the experimenter by checking eye movement patterns at prefrontal electrodes); (4) re-referencing to average of all signals; (5) construction of EEG segments beginning 200 ms before stimulus occurrence and ending 800 ms after stimulus onset, and baseline correction regarding the mean activity during the 200 ms prior to stimulus onset; and (6) algorithmic artifact rejection of voltage exceeding ± 100 μV and visual data inspection of segmented data (segments with artifacts were manually rejected, i.e., 15. 56%). Finally, individual participants' averages were calculated separately for correct responses in each condition. In accordance with previous studies (Maurage, Philippot, et al., 2008) and visual inspection of the data, each ERP component was quantified by measuring peak amplitude and peak latency at the mean activity from the central, left, and right electrodes in adapted time intervals: 100-130 ms at Oz-O1-O2 for P100; 80-150 ms at Cz-C3-C4 for N100; 150-200 ms at Oz-P7-P8 for N170; 190-320 ms at Oz-P7-P8 for N200; and 300-500 ms at Pz-P3-P4 for P3b. Importantly, the N200 component selected in this study is a specific ERP related to auditory processing, which peaks at occipito-temporal site (Joassin et al., 2004). Waveforms subtraction between crossmodal (congruent and incongruent) and unimodal conditions (i.e., crossmodal – [unimodal face + unimodal voice] for both emotions and both modalities) also were computed to explore the electrophysiological activity related to crossmodal integration. From these waveforms, a first visual inspection allowed targeting of specific components by selecting peak amplitude and peak latency from the mean activity at frontal (Fz, F3, F4), central (Cz, C3, C4), occipital (Oz, O1, O2), parieto-temporal (P7, P8), and parietal (Pz, P3, P4) sites (Maurage, Philippot, et al., 2008; Teder-Sälejärvi et al., 2002).

Statistical analyses

A first preliminary step consisted of group comparisons of demographic, psychological, and psychopathological variables, as well as alcohol consumption characteristics. Behavioral performance in the emotional crossmodal task was then evaluated via 3 × 2 × 2 × 3 repeated measures analyses of variance (ANOVAs) with Group (BDs, MDs, NDs) as the between-subjects factor and Emotion (Happiness, Anger), Modality (Face, Voice), and Condition (Unimodal, Crossmodal Congruent, Crossmodal Incongruent) as the within-subjects factors, computed separately for accuracy scores and reaction times. Electrophysiological activity was then assessed in two complementary steps. First, to explore specific emotional processing, we focused the analyses on emotions and computed them independently for amplitude and latency and each component of interest. For the specific visual (P100, N170) and decisional (P3b) components, 3 × 2 ANOVAs with Group (BDs, MDs, NDs) as the between-subjects factor and Emotion (Happiness, Anger) as the within-subjects factor were computed on visual trials and separately for unimodal, crossmodal congruent, and crossmodal incongruent conditions. For the specific auditory (N100, N200) and decisional (P3b) components, 3 × 2 ANOVAs with Group (BDs, MDs, NDs) as the between-subjects factor and Emotion (Happiness, Anger) as the within-subjects factor were computed on auditory trials for unimodal, crossmodal congruent, and crossmodal incongruent conditions. Second, to evaluate emotional crossmodal integration, we conducted analyses on the subtraction waves related to the specific crossmodal activity to compare all conditions of the emotional crossmodal task. Preliminary one-sample *t* tests were performed to evaluate whether the selected peaks significantly differed from 0. Significant activities were then investigated by using 3 × 2 × 2 × 2 ANOVAs with Group (BDs, MDs, NDs) as the between-subjects factor and Emotion (Happiness, Anger), Modality (Face, Voice), and Condition (Crossmodal Congruent, Crossmodal Incongruent) as the within-subjects factors. Finally, to support the absence of an acute alcohol effect beyond alcohol abstinence in the 3 days preceding the experiment, we performed correlations between electrophysiological activities that significantly differed between groups and total consumption in the week before the experiment. Because the main purpose of this study was to explore possible differences between groups regarding emotional processing, the

Results section globally focuses on the main effects and interactions involving groups. Significant group comparisons are reported below, and a Supplementary Materials section presents all other results, including nonsignificant group differences. For all statistical analyses, an alpha level of 0.05 was used.

Results

Demographic and psychopathological measures

Descriptive data are reported in Table 1. Groups did not significantly differ for age, $F(2, 50) = 0.53$, $p = 0.59$; gender, $\chi^2(2, N = 53) = 0.11$, $p = 0.74$; depressive symptoms, $F(2, 50) = 0.18$, $p = 0.84$; state anxiety, $F(2, 50) = 1.25$, $p = 0.30$; and trait anxiety, $F(2, 50) = 1.66$, $p = 0.20$. Moreover, groups did not differ in their ability to regulate emotional states: acceptance, $F(2, 50) = 0.10$, $p = 0.91$; positive refocusing, $F(2, 50) = 0.26$, $p = 0.77$; refocus on planning, $F(2, 50) = 0.26$, $p = 0.77$; positive reappraisal, $F(2, 50) = 0.50$, $p = 0.61$; putting into perspective, $F(2, 50) = 1.13$, $p = 0.33$; catastrophizing, $F(2, 50) = 0.74$, $p = 0.48$; rumination, $F(2, 50) = 0.38$, $p = 0.69$; self-blame, $F(2, 50) = 1.12$, $p = 0.33$; blaming others, $F(2, 50) = 0.38$, $p = 0.69$. Similarly, the total score of emotional reactivity, $F(2, 50) = 2.03$, $p = 0.14$, as well as the subfacets of emotional intensity, $F(2, 50) = 2$, $p = 0.15$, and emotional persistence, $F(2, 50) = 0.34$, $p = 0.72$, did not differ between groups. However, NDs had higher emotional sensitivity than did MDs, $t(34) = 2.63$, $p = 0.01$, but BDs did not differ from NDs, $t(34) = 1.17$, $p = 0.25$, and MDs, $t(32) = 1.66$, $p = 0.11$.

Behavioral analyses

Behavioral data are reported in Table 2, and results from the group comparisons are reported in the Supplementary Materials.

Table 2 Accuracy scores (percentage of correct answers) and reaction times (in milliseconds) for binge drinkers and control participants in each experimental condition (i.e., emotions, modalities, and conditions) of the crossmodal task

				Condition		
Emotion	Modality	Variable	Group	Unimodal	Crossmodal congruent	Crossmodal incongruent
Happiness	Face	AS	BDs	80.00 (12.21)	88.12 (7.43)	68.82 (24.38)
			MDs	75.41 (11.38)	82.24 (9.35)	64.35 (27.08)
			NDs	82.42 (15.74)	85.58 (10.14)	63.37 (28.55)
		RT	BDs	1075.39 (221.36)	993.14 (227.49)	1027.37 (231.72)
			MDs	967.52 (315.91)	935.04 (239.65)	1004.72 (292.67)
			NDs	958.90 (227.40)	949.86 (202.67)	1030.71 (239.02)
	Voice	AS	BDs	96.12 (5.45)	93.41 (9.97)	85.65 (20.91)
			MDs	92.47 (9.76)	90.71 (7.97)	81.06 (19.46)
			NDs	93.16 (10.72)	86.00 (13.43)	78.42 (24.57)
		RT	BDs	1046.63 (211.99)	982.12 (287.37)	975.09 (263.40)
			MDs	1026.44 (238.80)	917.18 (203.35)	927.77 (207.12)
			NDs	1022.77 (196.45)	953.74 (183.91)	1000.27 (199.04)
Anger	Face	AS	BDs	74.71 (15.07)	77.41 (15.31)	56.24 (23.80)
			MDs	72.00 (18.29)	80.00 (22.35)	65.18 (27.40)
			NDs	74.00 (10.46)	80.21 (12.16)	54.63 (20.72)
		RT	BDs	1101.46 (242.14)	1025.63 (228.31)	1090.29 (248.61)
			MDs	987.33 (345.77)	952.71 (222.03)	1005.17 (274.71)
			NDs	954.26 (230.15)	967.29 (182.34)	1017.58 (175.83)
	Voice	AS	BDs	94.88 (6.94)	89.65 (14.79)	83.41 (22.55)
			MDs	92.94 (9.75)	88.47 (18.84)	82.24 (21.79)
			NDs	94.74 (6.61)	91.79 (12.25)	88.42 (16.10)
		RT	BDs	1101.46 (242.14)	980.00 (241.16)	1006.09 (236.13)
			MDs	987.33 (345.77)	906.17 (204.52)	941.05 (215.63)
			NDs	954.26 (230.15)	917.41 (165.89)	966.01 (193.77)

Data are presented as mean (*SD*). AS = accuracy score; RT = reaction time; BDs = binge drinkers; MDs = moderate drinkers; NDs = nondrinkers.

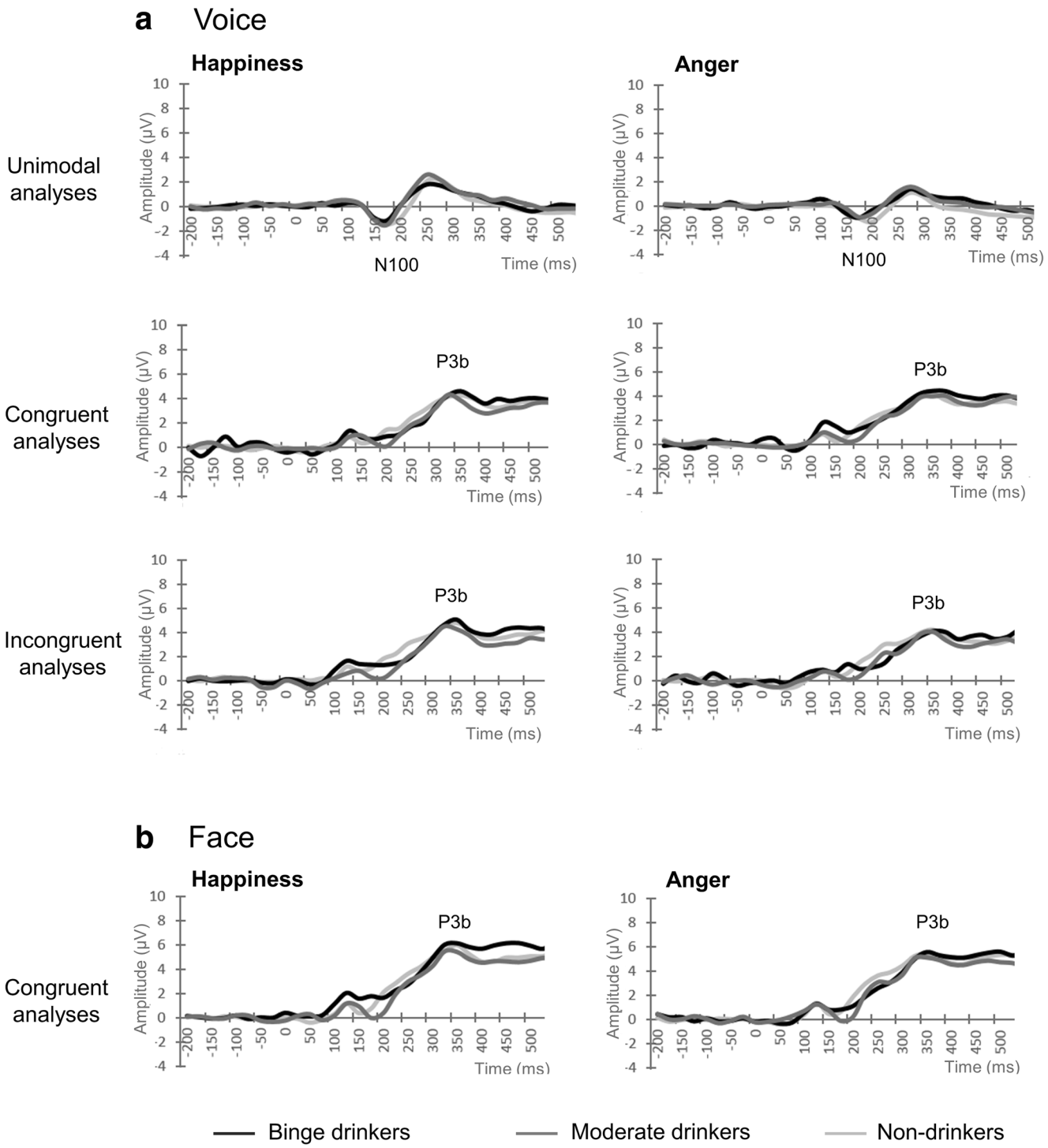

Fig. 1 Grand average event-related potential waveforms for the differences found between groups: binge drinkers (black line); moderate drinkers (dark gray line); and non-drinkers (light gray line). (**A**) Voice modality: (1) absence of differential processing between anger and happiness in binge drinkers, but N100 latency (ms) faster for anger in non-drinkers; (2) delayed P3b latency (ms) for the processing of both happiness and anger in binge drinkers; and (3) larger P3b amplitude (μV) for the processing of both happiness and anger in binge drinkers. (**B**) Face modality: larger P3b amplitude (μV) for happiness than for anger in binge drinkers in crossmodal congruent trials

Electrophysiological analyses

All analyses are reported in the Supplementary Materials; the current section only describes and depicts (Fig. 1) the activities that significantly differ between groups. The electrophysiological activities on which analyses have been performed (i.e., mean Oz-O1-O2 for P100, mean Cz-C3-C4 for N100, mean Oz-P7-P8 for N170 and N200, mean Pz-P3-P4

for P3b) are presented in Supplementary Figure 1 (visual processing) and Figure 2 (auditory processing) while the electrophysiological activities from frontal to occipital sites (i.e., Fpz, Fz, Cz, Pz, Oz) are illustrated in Supplementary Figure 3 (visual processing) and Figure 4 (auditory processing).

Unimodal analyses

Auditory processing

N100 Latency: A Group × Emotion interaction, $F(2, 50) = 3.31$, $p = 0.05$, $\eta_p^2 = 0.117$, was identified, showing longer latency for happiness than for anger in NDs, $t(18) = 3.18$, $p = 0.005$, but not in MDs, $t(16) = 0.68$, $p = 0.51$, and BDs, $t(16) = 0.05$, $p = 0.96$ (Fig. 1A).

Crossmodal analysis: congruent part

Visual processing

P3b Amplitude: A Group × Emotion interaction was found, $F(2, 50) = 3.28$, $p = 0.04$, $\eta_p^2 = 0.116$, showing larger amplitude for happiness than for anger in BDs, $t(16) = 2.20$, $p = 0.04$, but not in MDs, $t(16) = 0.78$, $p = 0.45$, and NDs, $t(18) = 0.31$, $p = 0.76$ (Fig. 1B).

Auditory processing

P3b Latency: A main effect of Group was observed, $F(2, 50) = 3.98$, $p = 0.02$, $\eta_p^2 = 0.137$, and indicated a delayed latency in BDs compared with MDs, $t(32) = 2.88$, $p = 0.007$, but not with NDs, $t(34) = 1.28$, $p = 0.21$ (Fig. 1A).

Crossmodal analysis: incongruent part

Auditory processing

P3b Amplitude: A main effect of Group was found, $F(2, 50) = 3.83$, $p = 0.03$, $\eta_p^2 = 0.133$, showing larger amplitude in BDs than in MDs, $t(32) = 2.34$, $p = 0.03$, and NDs, $t(32) = 2.08$, $p = 0.04$ (Fig. 1A).

Crossmodal integration

The subtraction waveforms (Fig. 2A) obtained to isolate the specific electrophysiological activity related to crossmodal integration revealed several relevant electrophysiological components. At frontal and central sites, a first negative peak between 100 and 160 ms was followed by a positive deflection peaking between 150 and 260 ms. At occipital and temporal sites, the first deflection was positive and appeared in the same time interval as the negative found in frontal and central

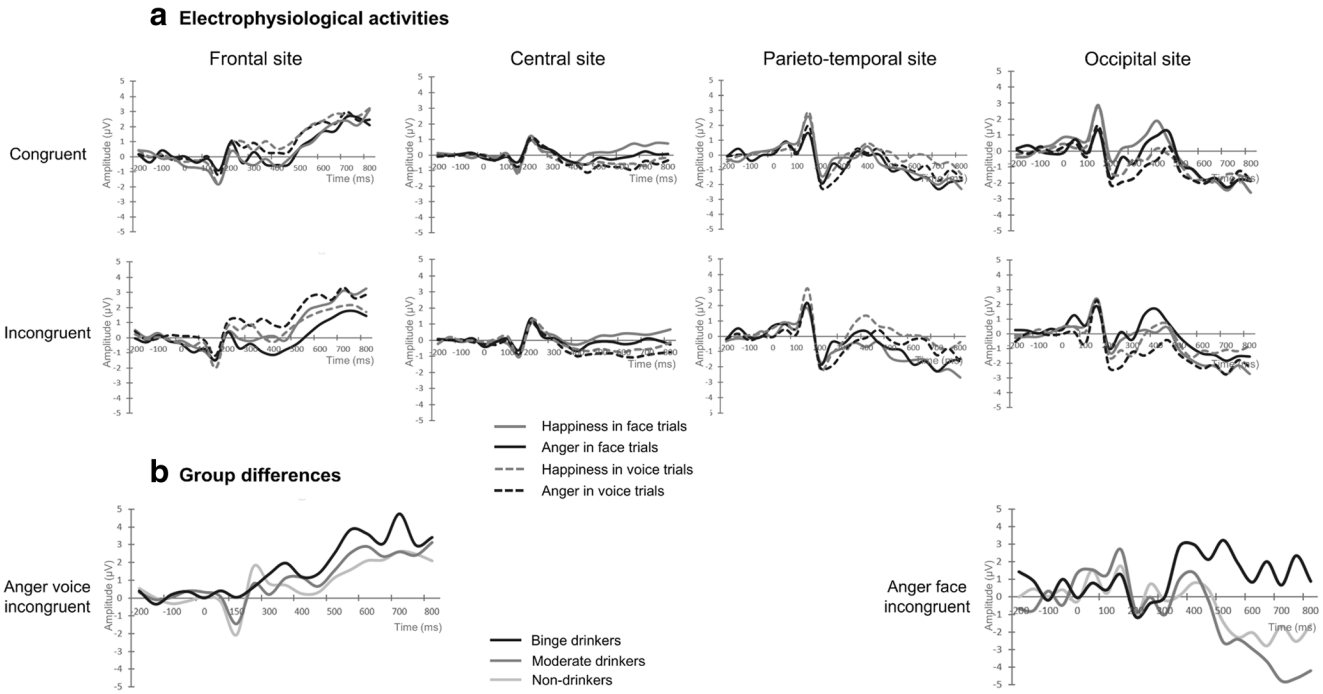

Fig. 2 Subtraction waveforms. (**A**) Electrophysiological activities at frontal, central, parieto-temporal, and occipital sites separately for congruent and incongruent subtractions (full lines represent waveforms in face modality and dotted lines represent waveforms in voice modality; happiness is in gray and anger in black). (**B**) Group differences (binge drinkers in black, moderate drinkers in dark gray, and nondrinkers in light gray) at frontal and occipital sites, depicting that binge drinkers have a delayed latency (ms) of the second positive component during the processing of anger in incongruent voice trials (frontal) and a higher amplitude (μV) of the third positive component during the processing of anger in incongruent face trials (occipital)

Table 3 Significant crossmodal integration-related components for congruent and incongruent subtraction waveforms

Electrophysiological components			Congruent	Happiness	Anger	Incongruent	Happiness	Anger
Region	Time (ms)	Type		*t* value	*t* value		*t* value	*t* value
Frontal	100-160	Negative	Face	9.05	6.53	Face	7.20	7.42
	150-260	Positive		8.97	8.68		9.75	8.36
Central	100-160	Negative		9.72	8.62		9.03	9.13
	150-260	Positive		11.80	9.20		11.31	10.08
Temporal	100-160	Positive		9.45	9.84		8.33	8.15
	160-230	Negative		8.17	8.14		10.06	8.09
Occipital	100-160	Positive		9.93	8.75		8.10	10.09
	160-230	Negative		6.88	9.18		9.94	9.13
	300-500	Positive		6.97	5.30		6.69	6.07
Frontal	100-160	Negative	Voice	8.88	9.97	Voice	7.64	8.70
	150-260	Positive		9.08	8.92		8.95	9.51
Central	100-160	Negative		12.76	8.76		9.85	10.30
	150-260	Positive		11.68	9.19		10.89	11.18
Temporal	100-160	Positive		10.21	9.65		9.10	9.70
	160-230	Negative		8.42	8.31		8.02	9.58
Occipital	100-160	Positive		9.29	8.61		8.70	7.95
	160-230	Negative		8.10	9.32		7.62	8.32
	300-500	Positive		5.66	4.47		5.16	5.19

All p values < 0.001

regions (100-160 ms); this activity was followed by a negative peak between 160 and 230 ms. Moreover, at the occipital site, a third positive deflection peaked between 300 and 500 ms. Finally, electrodes selected at the parietal site did not allow selection of a specific electrophysiological activity. These activities significantly differed from 0 (Table 3) and thus were compared between groups. All analyses involving group are reported in the Supplementary Materials; the following section describes only group differences.

Activities at frontal site

First negative component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Second positive component There was no main effect of Group and no interaction with Group for amplitude (Fig. 2B). Regarding latency, there was a Group × Condition × Emotion × Modality interaction (p = 0.007, η_p^2 = 0.179), showing a delayed latency in the processing of incongruent trials when anger was presented in voice modality in BDs compared with MDs, $t(32)$ = 2.17, p = 0.04, as well as in BDs compared with NDs, $t(34)$ = 2.25, p = 0.03, but no main effect of Group or any other interaction.

Activities at central site

First negative component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Second positive component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Activities at temporal site

First positive component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Second negative component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Activities at occipital site

First positive component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Second negative component For amplitude and latency, there was no main effect of Group and no interaction with Group.

Third positive component A Group × Modality interaction was found (p = 0.04, η_p^2 = 0.123), indicating that although MDs had a higher amplitude for face than for voice trials, $t(16)$ = 3.92, p = 0.001, BDs, $t(16)$ = 0.53, p = 0.61, and ND participants, $t(18)$ = 1.46, p = 0.16, did not show this difference. The Group × Modality interaction was qualified by a Group × Condition × Emotion × Modality interaction (p = 0.004, η_p^2 = 0.198), showing larger amplitude in the processing of incongruent trials when anger was presented in the face modality in BDs compared with MDs, $t(32)$ = 2.05, p = 0.04. There was no main effect of Group and no significant other interactions. For latency, there was a main effect of Group (p = 0.03, η_p^2 = 0.130), showing that BDs were faster than MDs, $t(32) = 2.48$, $p = 0.02$, but not NDs, $t(34) = 1.49$, $p = 0.15$. All interactions with Group were not significant.

Complementary analysis

Correlations supported the absence of an acute alcohol effect by showing no significant relationship between electrophysiological activities that significantly differed between groups and total alcohol consumption the week before the experiment (see Supplementary Materials for more details).

Discussion

Crossmodal integration is a critical process in humans and has repeatedly been identified as impaired in psychiatric disorders, including severe alcohol-use disorders (Maurage & Campanella, 2014). Although the crossmodal processing of emotions appeared to be preserved in BDs at the behavioral level (Lannoy, Dormal, et al., 2017), in the present electrophysiological study, we further investigated its cerebral correlates.

Consistent with our hypotheses, several differences were identified between BDs and controls at early and later stages of emotional processing (N100 and P3b), as well as in the specific activities related to crossmodal integration. Interestingly, BDs displayed a higher amplitude than did control participants, in line with the "compensation hypothesis". This proposal has been made in neuroimaging research (Campanella et al., 2013; Maurage, Bestelmeyer, et al., 2013; Schweinsburg et al., 2010) and has recently been reinforced by electrophysiological explorations (Crego et al., 2012; Lannoy, D'Hondt, et al., 2017; Lopez-Caneda et al., 2013; Smith, Mattick, & Sufani, 2015), which underlined that BDs need enhanced attentional and executive resources, as well as increased error detection activations, to correctly perform cognitive tasks. The present results thus support the assumption of brain compensation and extend previous findings described for cognitive abilities in emotional processing and crossmodal integration.

Specifically, at the early perceptual level, a group difference was found for unimodal auditory processing between BDs and NDs. Findings indicated no difference between anger and happiness for N100 latency in BDs, whereas NDs exhibited faster processing of anger than happiness. Lower resource recruitment and accelerated time to process anger has been associated with an adaptive behavioral response, as this allows faster motor reactions (Marsh, Ambady, & Kleck, 2005; Maurage, Philippot, et al., 2008); although observed only in nondrinking participants, this adjusting mechanism appears to be absent in BDs. Moreover, anger processing has been widely shown to be disrupted in severe alcohol-use disorders at behavioral and brain levels (Maurage, Campanella, et al., 2008; Park et al., 2015), and its consequences for social functioning and relapse risks also are well documented (Kornreich et al., 2002; Maurage, Campanella, et al., 2008). In BDs, early slowing down to detect anger voices can hide emotional difficulties in recognizing anger in others, as it has previously been shown in binge drinking (Maurage, Bestelmeyer, et al., 2013), which may play an important role in social conflicts (Attwood & Munafò, 2014) and more globally in interpersonal relationships.

At the later decisional level, group differences were observed in crossmodal conditions, both for congruent and incongruent trials. Regarding emotional visual processing, BDs had a higher P3b amplitude for happiness than for anger in congruent trials, whereas NDs and MDs did not show differential processing of these emotions at the decisional stage. Consistent with the previously discussed point, this result in BDs seems adaptive. It thus can be hypothesized that this differential processing of anger and happiness appears in BDs only in crossmodal congruent conditions, which are known to facilitate the identification of emotional content (Calvert et al., 2001), as well as at the later stage of the cognitive stream, whereas this processing is observed at early stages for unimodal trials in controls. However, differential P3 processing between BDs and controls has previously been reported during cognitive tasks and may index impaired neural attentional and inhibitory processes (Crego et al., 2009). Concerning emotional auditory processing, BDs were slower than controls in congruent trials, as indicated by a Group effect. This difference is particularly present in comparison to MDs and could index a reduced facilitation effect. The facilitation effect has been described as the marker of efficient crossmodal integration (Calvert et al., 2001; Li et al., 2015) and is indexed by faster processing of crossmodal stimuli compared with unimodal stimuli. This effect has been described at behavioral and brain levels and appeared to be clearly disrupted in patients with severe alcohol-use disorders. At the behavioral level, patients displayed no significant reaction time difference between unimodal and crossmodal

conditions, whereas a classic crossmodal facilitation effect was clearly observed in controls (Maurage et al., 2007; Maurage, Joassin, et al., 2013). At the cerebral level, impaired crossmodal integration was observed at the later decisional stage (P3b), consistent with the current results. However, results in patients did not show this deficit for latency but rather through a larger P3b amplitude for crossmodal than unimodal conditions (Maurage, Philippot, et al., 2008), which might index the increase in this impairment in those with severe alcohol-use disorders compared with that in BDs. Indeed, in the current study, no difference was shown at the behavioral level, which supports the persistence of a facilitation effect in binge drinking, although it seems that, at the electrophysiological level, BDs have a reduced ability to take advantage of crossmodal information. Moreover, this slower P3b processing is consistent with results of previous binge drinking studies that used an emotional valence detection task with auditory stimuli (i.e., a neutral word pronounced with prosodies of happiness and anger) presented in a unimodal way (Maurage et al., 2009). Furthermore, during incongruent trials, BDs showed larger electrophysiological activity for the processing of both happiness and anger than did MDs and NDs. According to previous studies (Crego et al., 2012; López-Caneda et al., 2012), this observation of enhanced brain recruitment in incongruent trials might reflect a compensatory mechanism for processing that requires more executive resources. In addition, in line with previous studies indicating that slower P300 could be a marker of decisional and executive deficits in binge drinking (Ehlers et al., 2007), these findings suggest a specific impairment for crossmodal processing at this decisional stage.

As a whole, regarding emotional processing, electrophysiological analyses on classic ERP components underline a possible difficulty in identifying anger, which could lead to maladaptive responses and deleterious social functioning. At the crossmodal level, findings reveal less efficient processing in binge drinking, with a reduced advantage offered by crossmodal congruent stimuli and a stronger incongruence effect. These impairments might be related to difficulties in daily life situations and social interactions, as crossmodal processing represents an ecological emotional evaluation.

Finally, concerning crossmodal integration per se, the observed deflections are in accordance with those described by Teder-Sälejärvi et al. (2002). Regarding group differences, these results confirm both delayed latency and enhanced amplitude for the processing of anger in incongruent trials among BDs, the first being detected on a second positive component peaking at the frontal site and the second on a third positive component at the occipital site. These analyses thus account for the presence of cerebral modifications in binge drinking, showing a need for higher resources and more time to process crossmodal stimuli. In line with the first analyses, the comparisons based on subtraction waveforms also indicated that cerebral changes during crossmodal processing in BDs seem related to later stages. According to previous studies (Joassin et al., 2004; Miller, Stein, & Rowland, 2017), crossmodal integration is first related to complex visuo-auditory interactions at perceptual stages, followed by the building of an integrated crossmodal representation at later stages, together with facilitatory or inhibitory processes. Nevertheless, although results appear to be consistent with the literature and throughout our different analysis steps, findings also reveal that BDs process faster than MDs do on the last component of the occipital site, although no difference was found with NDs. This effect is unexpected and constitutes a limitation for the current interpretation.

Altogether, the findings of the current work clearly emphasize modified cerebral functioning in binge drinking. The processing of anger stimuli and incongruent trials appears to be especially disturbed in BDs, whereas an absence of the facilitation effect might be present during congruent crossmodal trials. Because this study is the first to explore the electrophysiological correlates of emotional crossmodal processing in binge drinking, the current results should be reinforced in future studies. More specifically, participants' recruitment was based on a first binary screening questionnaire followed by validated self-reported scales, leading to the exclusion of clinical and subclinical comorbidities (anxiety, depression, other addictive disorders). Because the presence of such comorbid conditions is frequent in binge drinking, our selection criteria (although leading to a more specific and controlled measure of binge drinking consequences) might have ended up in a sample not fully representative of the general BDs' population. Moreover, although we excluded an acute alcohol effect by asking participants to abstain from alcohol in the 3 days before the experiment, which was confirmed by the absence of a relationship between total alcohol consumption in the week preceding the experiment and ERP data, subsequent studies should further investigate this effect with objective measures (e.g., breathalyzers). Eventually, the findings obtained should be replicated and confirmed in studies with larger samples (i.e., to increase the statistical power), as well as more specific research questions (e.g., focusing on congruent crossmodal integration).

Beyond these limitations, the current study offers first insights into this research field and has several implications. First, it underlines that BDs have difficulties in processing emotional stimuli, and these disturbances appear to be in line with those previously found among patients with severe alcohol-use disorders. The cerebral modifications suggest that BDs are still able to compensate for their difficulties, because they correctly perform at the behavioral level. Nevertheless, difficulties can persist with the perpetuation of excessive alcohol consumption and constitute critical factors in the development of alcohol-related disorders (Oscar-Berman et al., 2014). This proposal is reinforced by the fact that behavioral

impairments are observed in BDs with more complex emotional paradigms (Maurage, Bestelmeyer, et al., 2013). Second, this study offers some insights regarding the origin of the crossmodal deficit in alcohol-related disorders (Maurage & Campanella, 2014) by showing that such brain dysfunctions are already present, although to a lesser extent, in binge drinking. Finally, this study confirms the reliability of electrophysiology for exploring the different stages related to specific processing and highlights possible deficits that are not observable with classic behavioral methods.

Acknowledgments This research was supported by the Belgian Fund for Scientific Research (FRS-FNRS) and the "Fondation pour la Recherche en Alcoologie" (FRA). These funds did not exert any editorial influence or censorship on any part of this article.

Compliance with ethical standards

Conflict of interest The authors declare no conflicts of interest pertaining to the data or analyses presented.

References

Attwood, A. S., & Munafò, M. R. (2014). Effects of acute alcohol consumption and processing of emotion in faces: Implications for understanding alcohol-related aggression. *Journal of Psychopharmacology*, *28*(8), 719–732.

Babor, T., Higgins-Biddle, J., Saunders, J., & Monteiro, M. (2001). *The alcohol use disorders identification test: Guidelines for use in primary care*. Geneva, Switzerland: World Health Organization.

Beck, A. T., Steer, R.A., & Brown, G. K. (1996). *Beck depression inventory manual*, 2nd ed. Psychological Corporation: San Antonio, TX

Bonomo, Y. A., Bowes, G., Coffey, C., Carlin, J. B., & Patton, G. C. (2004). Teenage drinking and the onset of alcohol dependence: A cohort study over seven years. *Addiction*, *99*(12), 1520–1528.

Brion, M., D'Hondt, F., Davidoff, D. A., & Maurage, P. (2016). Beyond cognition: Understanding affective impairments in Korsakoff syndrome. *Emotion Review*, *8*(4), 376–384.

Calvert, G. A., Hansen, P. C., Iversen, S. D., & Brammer, M. J. (2001). Detection of audio-visual integration sites in humans by application of electrophysiological criteria to the BOLD effect. *NeuroImage*, *14*(2), 427–438.

Campanella, S., & Belin, P. (2007). Integrating face and voice in person perception. *Trends in Cognitive Sciences*, *11*(12), 535–543.

Campanella, S., Peigneux, P., Petit, G., Lallemand, F., Saeremans, M., Noël, X., … Verbanck, P. (2013). Increased cortical activity in binge drinkers during working memory task: A preliminary assessment through a functional magnetic resonance imaging study. *PLoS ONE, 8*(4), e62260.

Courtney, K. E., & Polich, J. (2009). Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin*, *135*(1), 142–156.

Crego, A., Cadaveira, F., Parada, M., Corral, M., Caamaño-Isorna, F., & Rodríguez Holguín, S. (2012). Increased amplitude of P3 event-related potential in young binge drinkers. *Alcohol*, *46*(5), 415–425.

Crego, A., Holguín, S. R., Parada, M., Mota, N., Corral, M., & Cadaveira, F. (2009). Binge drinking affects attentional and visual working memory processing in young university students. *Alcoholism: Clinical and Experimental Research, 33*, 1870–1879.

D'Hondt, F., Campanella, S., Kornreich, C., Philippot, P., & Maurage, P. (2014). Below and beyond the recognition of emotional facial expressions in alcohol dependence: From basic perception to social cognition. *Neuropsychiatric Disease and Treatment*, 2177.

Driver, J., & Spence, C. (2000). Multisensory perception: Beyond modularity and convergence. *Current Biology*, *10*(20), R731–R735.

Ehlers, C. L., Phillips, E., Finnerman, G., Gilder, D., Lau, P., & Criado, J. (2007). P3 components and adolescent binge drinking in Southwest California Indians. *Neurotoxicology and Teratology*, *29*(1), 153–163.

Enoch, M.-A. (2006). Genetic and environmental influences on the development of alcoholism: Resilience vs. risk. *Annals of the New York Academy of Sciences*, *1094*(1), 193–201.

Goudriaan, A. E., Grekin, E. R., & Sher, K. J. (2011). Decision making and response inhibition as predictors of heavy alcohol use: A prospective study: Neurocognitive predictors of heavy alcohol use. *Alcoholism: Clinical and Experimental Research*, *35*(6), 1050–1057.

Hartley, D. E., Elsabagh, S., & File, S. E. (2004). Binge drinking and sex: Effects on mood and cognitive function in healthy young volunteers. *Pharmacology Biochemistry and Behavior*, *78*(3), 611–619.

Heffernan, T., Clark, R., Bartholomew, J., Ling, J., & Stephens, S. (2010). Does binge drinking in teenagers affect their everyday prospective memory? *Drug and Alcohol Dependence*, *109*(1–3), 73–78.

Hermens, D. F., Lagopoulos, J., Tobias-Webb, J., De Regt, T., Dore, G., Juckes, L., … Hickie, I. B. (2013). Pathways to alcohol-induced brain impairment in young people: A review. *Cortex, 49*(1), 3–17.

Jermann, F., Van der Linden, M., d'Acremont, M., & Zermatten, A. (2006). Cognitive Emotion Regulation Questionnaire (CERQ). *European Journal of Psychological Assessment*, *22*(2), 126–131.

Joassin, F., Maurage, P., Bruyer, R., Crommelinck, M., & Campanella, S. (2004). When audition alters vision: An event-related potential study of the crossmodal interactions between faces and voices. *Neuroscience Letter, 369*, 132–137.

Jung, T-P., Makeig, S., Humphries, C., Lee, T-W., Mckeown, M. J., Iragui, V., … Sejnowski, T. J. (2000). Removing electroencephalographic artifacts by blind source separation. *Psychophysiology, 37,* 163–778.

Kornreich, C., Philippot, P., Foisy, M-L., Blairy, S., Raynaud, E., … Verbanck, P. (2002). Impaired emotional facial expression recognition is associated with interpersonal problems in alcoholism. *Alcohol and Alcoholism, 37*(4), 394–400.

Lannoy, S., D'Hondt, F., Dormal, V., Billieux, J., & Maurage, P. (2017). Electrophysiological correlates of performance monitoring in binge drinking: Impaired error-related but preserved feedback processing. *Clinical Neurophysiology*, *128*, 2110-2121.

Lannoy, S., Dormal, V., Brion, M., Billieux, J., & Maurage, P. (2017). Preserved crossmodal integration of emotional signals in binge drinking. *Frontiers in Psychology*, *8*.

Lannoy, S., Heeren, A., Rochat, L., Rossignol, M., Van der Linden, M., & Billieux, J. (2014). Is there an all-embracing construct of emotion reactivity? Adaptation and validation of the emotion reactivity scale among a French-speaking community sample. *Comprehensive Psychiatry*, *55*(8), 1960–1967.

Li, Y., Long, J., Huang, B., Yu, T., Wu, W., Liu, Y., … Sun, P. (2015). Crossmodal integration enhances neural representation of task-relevant features in audiovisual face perception. *Cerebral Cortex, 25*(2), 384–395.

Lopez-Caneda, E., Cadaveira, F., Crego, A., Doallo, S., Corral, M., Gomez-Suarez, A., & Rodriguez Holguin, S. (2013). Effects of a persistent binge drinking pattern of alcohol consumption in young people: A follow-up study using event-related potentials. *Alcohol and Alcoholism*, *48*(4), 464–471.

López-Caneda, E., Cadaveira, F., Crego, A., Gómez-Suárez, A., Corral, M., Parada, M., … Rodríguez Holguín, S. (2012). Hyperactivation of right inferior frontal cortex in young binge drinkers during

response inhibition: A follow-up study: Response inhibition in young binge drinkers. *Addiction, 107*(10), 1796–1808.

López-Caneda, E., Rodríguez Holguín, S., Corral, M., Doallo, S., & Cadaveira, F. (2014). Evolution of the binge drinking pattern in college students: Neurophysiological correlates. *Alcohol*, *48*(5), 407–418.

Marsh, A. A., Ambady, N., & Kleck, R. E. (2005). The effects of fear and anger facial expressions on approach- and avoidance-related behaviors. *Emotion*, *5*(1), 119–124.

Maurage, P., Bestelmeyer, P. E. G., Rouger, J., Charest, I., & Belin, P. (2013). Binge drinking influences the cerebral processing of vocal affective bursts in young adults. *NeuroImage: Clinical*, *3*, 218–225.

Maurage, P., & Campanella, S. (2014). Towards a crossmodal exploration of cognitive deficits in psychopathology. *Psychologica Belgica*, *54*(3), 282–297.

Maurage, P., Campanella, S., Philippot, P., Pham, T. H., & Joassin, F. (2007). The crossmodal facilitation effect is disrupted in alcoholism: A study with emotional stimuli. *Alcohol and Alcoholism*, *42*(6), 552–559.

Maurage, P., Campanella, S., Philippot, P., Vermeulen, N., Constant, E., Luminet, O., & de Timary, P. (2008). Electrophysiological correlates of the disrupted processing of anger in alcoholism. *International Journal of Psychophysiology*, *70*(1), 50–62.

Maurage, P., Joassin, F., Pesenti, M., Grandin, C., Heeren, A., Philippot, P., & de Timary, P. (2013). The neural network sustaining crossmodal integration is impaired in alcohol-dependence: An fMRI study. *Cortex*, *49*(6), 1610–1626.

Maurage, P., Joassin, F., Speth, A., Modave, J., Philippot, P., & Campanella, S. (2012). Cerebral effects of binge drinking: Respective influences of global alcohol intake and consumption pattern. *Clinical Neurophysiology*, *123*(5), 892–901.

Maurage, P., Pesenti, M., Philippot, P., Joassin, F., & Campanella, S. (2009). Latent deleterious effects of binge drinking over a short period of time revealed only by electrophysiological measures. *Journal of Psychiatry & Neuroscience: JPN*, *34*(2), 111.

Maurage, P., Petit, G., & Campanella, S. (2013). Pathways to alcohol-induced brain impairment in young people: A review by Hermens et al., 2013. *Cortex*, *49*(4), 1155–1159.

Maurage, P., Philippot, P., Joassin, F., Pauwels, L., Pham, T., Prieto, E. A., … Campanella, S. (2008). The auditory-visual integration of anger is impaired in alcoholism: An event-related potentials study. *Journal of Psychiatry & Neuroscience: JPN, 33*(2), 111.

Miller, R. L., Stein, B. E., & Rowland, B. A. (2017). Multisensory integration uses a real-time unisensory–multisensory transform. *The Journal of Neuroscience, 37*(20), 5183–5194.

National Institute of Alcohol Abuse and Alcoholism (NIAAA). (2004, Winter). NIAAA council approves definition of binge drinking. *NIAAA Newsletter, 2004*(3), 3.

Oscar-Berman, M., Valmas, M. M., Sawyer, K. S., Ruiz, S. M., Luhar, R. B., & Gravitz, Z. R. (2014). Profiles of impaired, spared, and recovered neuropsychologic processes in alcoholism. In *Handbook of Clinical Neurology* (Vol. 125, pp. 183–210). Elsevier.

Parada, M., Corral, M., Mota, N., Crego, A., Rodríguez Holguín, S., & Cadaveira, F. (2012). Executive functioning and alcohol binge drinking in university students. *Addictive Behaviors*, *37*(2), 167–172.

Park, M.-S., Kim, S.-H., Sohn, S., Kim, G.-J., Kim, Y.-K., & Sohn, J.-H. (2015). Brain activation during processing of angry facial expressions in patients with alcohol dependency. *Journal of Physiological Anthropology*, *34*(1), 6.

Petit, G., Maurage, P., Kornreich, C., Verbanck, P., & Campanella, S. (2014). Binge drinking in adolescents: A review of neurophysiological and neuroimaging research. *Alcohol and Alcoholism*, *49*(2), 198–206.

Schweinsburg, A. D., McQueeny, T., Nagel, B. J., Eyler, L. T., & Tapert, S. F. (2010). A preliminary study of functional magnetic resonance imaging response during verbal encoding among adolescent binge drinkers. *Alcohol*, *44*(1), 111–117.

Smith, J. L., Mattick, R. P., & Sufani, C. (2015). Female but not male young heavy drinkers display altered performance monitoring. *Psychiatry Research: Neuroimaging*, *233*(3), 424–435.

Spielberger, D. C., Gorsuch, R. L., Lushene, R., Vagg, P. R., Jacobs, G. A. (1983). *Manual for the state-trait anxiety inventory*. Consulting Psychology Press, Palo Alto.

Stephens, D. N., & Duka, T. (2008). Cognitive and emotional consequences of binge drinking: Role of amygdala and prefrontal cortex. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *363*(1507), 3169–3179.

Teder-Sälejärvi, W. A., McDonald, J. J., Di Russo, F., & Hillyard, S. A. (2002). An analysis of audio-visual crossmodal integration by means of event-related potential (ERP) recordings. *Cognitive Brain Research 14*, 106-114

Townshend, J. M., & Duka, T. (2002). Patterns of alcohol drinking in a population of young social drinkers: A comparison of questionnaire and diary measures. *Alcohol & Alcoholism*, 37(2), 187-192.

Townshend, J. M., & Duka, T. (2005). Binge drinking, cognitive performance and mood in a population of young social drinkers. *Alcoholism: Clinical & Experimental Research*, *29*(3), 317–325.