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Binge drinking influences the cerebral processing of vocal affective bursts in young adults

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ABSTRACT

Binge drinking is now considered a central public health issue and is associated with emotional and interpersonal problems, but the neural implications of these deficits remain unexplored. The present study aimed at offering the first insights into the effects of binge drinking on the neural processing of vocal affect. On the basis of an alcohol-consumption screening phase (204 students), 24 young adults (12 binge drinkers and 12 matched controls, mean age: 23.8 years) were selected and performed an emotional categorisation task on morphed vocal stimuli (drawn from a morphed fear–anger continuum) during fMRI scanning. In comparison to controls, binge drinkers presented (1) worse behavioural performance in emotional affect categorisation; (2) reduced activation of bilateral superior temporal gyrus; and (3) increased activation of right middle frontal gyrus. These results constitute the first evidence of altered cerebral processing of emotional stimuli in binge drinking and confirm that binge drinking leads to marked cerebral changes, which has important implications for research and clinical practice.

1. Introduction

Binge drinking refers to repeated alternation between massive alcohol intakes and abstinence periods, and is becoming increasingly prominent among young adults (Johnston et al., 2012). Its deleterious consequences have been described at medical, cognitive and social levels (Laghi et al., 2012), but the influence of binge drinking on brain functioning has received only limited attention. However, as it is largely established that long-term excessive alcohol consumption leads to marked cerebral impairments (Bühler and Mann, 2011a; Duka et al., 2004, 2011) and as binge drinking might constitute a first step towards alcohol-dependence (Sanhueza et al., 2011; Tucker et al., 2003), young binge drinkers may already present cerebral changes. This proposal is further reinforced by two arguments: First, binge drinking is characterized by repeated alternations between intense alcohol consumption and withdrawal periods which are harmful for brain functioning (Maldonado-Devincci et al., 2010; Obernier et al., 2002; Overstreet et al., 2002). Second, the adolescent brain is in an intense neuronal remodelling phase and is thus highly sensitive to the effects of alcohol (Bava and Tapert, 2010; Blakemore, 2012).

Nevertheless, very few functional neuroimaging studies have explored the cerebral consequences of binge drinking habits in humans. Several studies have investigated the cerebral effects of alcohol-dependence in young individuals (De Bellis et al., 2005; Schweinsburg et al., 2005) or the anatomical consequences of binge drinking (McQueeny et al., 2009; Squeglia et al., 2012a) but without exploring the related functional changes. Moreover, electrophysiological studies suggested altered brain functioning in binge drinking but without precisely identifying the brain regions involved (Crego et al., 2009; López-Caneda et al., 2012; Maurage et al., 2009a, 2012). Actually, only five studies have specifically explored the functional correlates of binge drinking using fMRI (Campanella et al., 2013; Schweinsburg et al., 2010, 2011; Squeglia et al., 2011; Xiao et al., 2013). These studies showed that binge drinking in adolescence is associated with decreased activity in occipital, hippocampal and prefrontal areas, but with increased activity in amygdala, insula, parietal and superior frontal regions during memory and decision making tasks. While contributing to a better understanding of cerebral changes in binge drinking, these preliminary studies have not answered several crucial questions:

(1.) It is unclear whether the cerebral changes observed are specific to the high-level tasks used earlier or would also be observed when other cognitive abilities are required. Specifically, as large-scale

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emotional impairments have been shown in alcohol-dependence (Maurage et al., 2008a, 2009b) and as affective deficits have been suggested in binge drinking (Stephens and Duka, 2008), the brain deficits may also be present during the processing of emotions.

(2.) Most of these studies focused on adolescent binge drinkers, but this habit is far more frequent among young adults (Naimi et al., 2003) and the generalisation of earlier results to the global binge drinking population remains unknown. As brain remodelling is more limited among young adults, binge drinking might indeed have less cerebral effects in this population.

(3.) Binge drinkers with current marijuana use (Schweinsburg et al., 2010) or other drug abuse (Campanella et al., 2013; Schweinsburg et al., 2011) have been included in previous studies, and subclinical comorbidities have not always been controlled for, leading to group differences in depression and anxiety (Squeglia et al., 2011). Given that marijuana, subclinical depression and anxiety affect brain activity (Cavanagh and Geisler, 2006; Jager and Ramsey, 2008), the cerebral changes reported might partly be due to these comorbidities.

Here, we used an auditory affective two-alternative forced choice task among young adult binge drinkers without comorbidities and additional drug abuse to avoid the aforementioned shortcomings. Our main hypothesis is that the functional brain changes due to binge drinking, thus far observed during cognitive tasks, are also present in emotional tasks. We specifically hypothesized a reduced performance during auditory emotional processing in binge drinking, associated with a reduced activation of the brain regions classically involved in the processing of vocally expressed emotions. Specifically, earlier studies conducted among healthy participants have shown that the processing of human affective bursts and prosody mainly relies not only on superior temporal gyri (Beaucousin et al., 2007; Ethofer et al., 2009a, 2012; Grandjean et al., 2005), but also on the right middle temporal gyrus (Ethofer et al., 2006; Imaizumi et al., 1997; Mitchell, 2007) and inferior frontal gyri (Ethofer et al., 2009b; Wildgruber et al., 2005). Moreover, as our population consisted of young adults without psychopathological comorbidities, the present study will also test whether the deficit observed in previous studies on adolescent binge drinkers can be generalised to the core population of binge drinkers, and whether this deficit persists when comorbidities are controlled for.

2. Material and methods

2.1. Participants

A general screening phase was first conducted among the under- and postgraduate community of the University of Glasgow (United Kingdom). 204 students filled in a questionnaire assessing psychological measures and alcohol-drug consumption characteristics. On the basis of this evaluation, 24 students were selected, fulfilling the following conditions: no positive personal or family history of alcohol-dependence, absence of past or current other drug or psychotropic medication consumption, absence of present nicotine dependence, no major medical problems, no central nervous system disorder (including epilepsy and history of brain trauma), no auditory impairment, low depression (i.e. score lower than 8 at the Beck Depression Inventory, BDI (Beck and Steer, 1987)) and anxiety scores (i.e. scores lower than 45 and 52 at the State and Trait Anxiety Inventory, STAI A–B (Spielberger et al., 1983), respectively), and right-handedness.

Three variables were used to determine the groups: mean number of alcohol doses per drinking occasion (i.e. per day during which alcoholic drinks are consumed), mean number of drinking occasions per week and consumption speed in doses per hour. One dose corresponds to 10 g of pure ethanol. According to their alcohol consumption during the last six months, subjects were distributed among two groups of 12 participants (see Table 1 for descriptive data): controls (CR; doses per occasion <2; occasions per week <1; consumption speed <1) and binge drinkers (BD; doses per occasion >5; occasions per week >3; consumption speed >2). One-way analyses of variance (ANOVAs) were performed to check if the participants were correctly distributed across groups. These analyses showed that groups did not significantly differ for age at first alcohol consumption [$F(1,22) = 1.03$, NS], but binge drinkers presented as expected higher number of drinking occasions per week [$F(1,22) = 94.78$, $p < .001$], doses per occasion [$F(1,22) = 72.67$, $p < .001$], doses per week [$F(1,22) = 35,26$, $p < .001$] and drunkenness episodes [$F(1,22) = 12.69$, $p < .01$] than controls, as well as a faster consumption speed [$F(1,22) = 10.05$, $p < .01$]. Groups were matched for age (age range: 19–32 years in each group), gender (7 males per group) and education. Education level was assessed according to the number of years of education completed since starting primary school. Participants were asked to abstain from any alcohol consumption for at least three days before the scanning session. Before starting the fMRI study, a brief hearing test was performed to ensure that all participants were of normal hearing, and they were assessed for psychological control measures to evaluate subclinical depression (BDI), anxiety (STAI A–B) and current alcohol craving (as assessed by the Obsessive–Compulsive Drinking Scale, OCDS (Anton et al., 1995)). Participants were provided with full details regarding the aims of the study and the procedure to be followed. After receiving this information, all participants gave their informed consent. The study was approved by the local ethics committee and carried out according to the Declaration of Helsinki. Participants were reimbursed £12 for their time.

2.2. Stimuli and tasks

Participants performed a two-alternative forced choice task regarding one of two emotion categories of morphed affective bursts. Original recordings were taken from the Montreal Affective Voices Battery (Belin et al., 2008) in which actors were instructed to produce emotional interjections using the vowel /a/. We chose four identities (two female), each expressing two emotions (anger and fear). The use of auditory stimuli expressing negative emotions is justified by earlier studies showing a strong deficit for the processing of these stimuli in alcohol-dependence and binge drinking (Maurage et al., 2008b, 2009a). Stimuli were normalised in energy (root mean square) before and after morphing. Angry to fearful

Table 1
Results for demographic, psychopathological and alcohol consumption measures for controls (CR) and binge drinkers (BD): mean (S.D.).

	CR (N = 12)	BD (N = 12)
Age[NS]	23.4 (4.21)	24.2 (4.49)
Gender ratio (female/male)	5/7	5/7
Educational level[NS]	17.2 (1.53)	15.9 (1.56)
BDI[NS]	1.75 (1.48)	2.83 (1.85)
STAI-A[NS]	30.33 (7.26)	32.42 (7.99)
STAI-B[NS]	36.75 (6.78)	39.17 (12.46)
OCDS[NS]	5.4 (2.18)	6.9 (3.72)
Age at first alcohol consumption[NS]	15.2 (4.02)	13.6 (3.6)
Age when starting binge drinking habits	/	19.04 (2.55)
Duration of binge drinking habits (in months)	/	63.1 (41.92)
Consumption speed[ab*]	.86 (.68)	2.51 (1.06)
Number of doses per week[b**]	.5 (1.17)	31.7 (18.19)
Mean number of occasions per week[b**]	.33 (.65)	4.1 (1.16)
Mean number of doses per occasion[b**]	1.37 (1.13)	7.5 (3.01)
Number of drunkenness episodes[b*]	.17 (0.57)	8.83 (8.41)

BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory (A = state anxiety; B = trait anxiety); OCDS = Obsessive–Compulsive Drinking Scale.
NS = non-significant.
[a] In doses per hour.
[b] During the last six months.
* $p < 0.01$.
** $p < 0.001$.

continua were created separately for each identity, in seven steps that corresponded to 5/95%, 20/80%, 35/65%, 50/50%, 65/35%, 80/20% and 95/5% fear/anger. The duration of the bursts within each continuum was kept constant (range: .6–9 s). We used STRAIGHT software (Kawahara and Matsui, 2003) for stimulus manipulation and Psychtoolbox3 (Brainard, 1997) for stimulus presentation and response recording. Both programmes are based on MatlabR2009b (Mathworks, Inc.). Acoustic analyses of the stimuli are published elsewhere (Bestelmeyer et al., 2010).

A continuous carry-over design (Aguirre, 2007) was employed to control for the effects of one stimulus upon the next using a first-order serially balanced sequence of stimuli known as type-1-index-1 (Nonyane and Theobald, 2007). In this sequence each stimulus is preceded and followed by every other stimulus (seven morph steps and one silence, i.e. a period without auditory stimulation) with an equal number of times, totalling 65 stimuli. The sequence was repeated six times in each of three runs. Each run started with two silences, and each sequence of 65 stimuli was followed by nine TRs of silences. The total number of events per run was thus 446 (each corresponding to one fMRI volume), and the total number of trials for each morph step was 144.

Stimuli were presented binaurally using the electrostatic NNL headphone system (NordicNeuroLab, Inc.) at 80 dB SPL(C). Participants were asked to perform a two-alternative forced choice task in which each affective voice had to be categorised as angry or fearful by means of two button presses mounted on an MRI compatible response box (NordicNeuroLab, Inc.). Subjects had to react as fast as possible and keep their eyes closed. Response to each stimulus and reaction times were recorded. Only correct answers were considered for behavioural analysis. As the 50/50% morph step equally comprises fear and anger, there is no correct response for this level, and it has not been included in the behavioural analyses. Before the experiment, each subject underwent a short training session to practise the task.

2.3. Imaging procedure and fMRI data analysis

Scans were acquired in a 3.0 Tesla Siemens Tim Trio scanner using a 12-channel head coil. Whole brain T1-weighted anatomical scans were performed using fast gradient echo known as T1 'Magnetization Prepared Rapid Gradient Echo' (MPRAGE) consisting of 192 axial slices of 1 mm thickness with an inplane resolution of 1 × 1 × 1 (FOV = 256) and a matrix of 256 × 256 performed at the end of the experiment. T2-weighted functional scans were acquired using an interleaved ascending sequence consisting of 32 slices of 3 mm thickness (3 mm gap) with an inplane resolution of 3 × 3 × 3 (FOV = 1260) and an acquisition matrix of 70 × 70. The three runs (TR = 2 s, TE = 30 ms) consisted of 446 volumes each. The presentation of a stimulus coincided with the beginning of the TR. Vocal stimuli ranged in duration between 600 and 900 ms. A voice localizer scan (TR = 2 s, TE = 30 ms, 310 volumes) was performed before the experimental scans, allowing reliable identification of the temporal voice areas using the vocal versus non-vocal contrast (Belin et al., 2000). This voice localizer tested whether groups differed concerning basic cerebral activations related to human voice processing.

All MRI data were analysed using SPM8 (Wellcome Department of Cognitive Neurology, University College London). Pre-processing of functional scans consisted of corrections for head motion (trilinear interpolation) and scans were realigned to the first volume. Functional runs were then coregistered to their corresponding individual anatomical scans. Functional (3 mm isotropic voxels) and anatomical (1 mm isotropic voxels) data were transformed to Montreal Neurological Institute (MNI) space after segmentation of the anatomical scans. Normalised data were spatially smoothed by applying a Gaussian kernel of 8 mm full width at half maximum (FWHM). Condition-related changes in regional brain activity were estimated for each participant by computing the contrasts between the mean hemodynamic responses evoked by each morph step relative to the silent baseline periods between runs. Significant cerebral activations were then examined by means of a full-factorial model with group as between-subjects factor and morph step as within-subjects factor. The statistical threshold was set to $p < .05$ FWE-corrected at cluster level for multiple comparisons with a cluster size of at least 10 contiguous voxels. Random effects analyses with one-sample t-tests were also used to explore the global activations found in each group and each emotion type, with statistical threshold set to $p < .05$ FWE-corrected for multiple comparisons using cluster size and extending to at least 20 voxels. Beta-values of the peak activity recorded in the superior temporal and middle frontal gyri were extracted using rfxplot toolbox (Gläscher, 2009) in order to perform Pearson's correlations with behavioural data.

3. Results

3.1. Demographic and psychopathological measures

ANOVAs showed that groups did not significantly differ for age [$F(1,22) = .18$, NS], gender, educational level [$F(1,22) = 3.92$, NS], depression [$F(1,22) = 2.50$, NS], state anxiety [$F(1,22) = .44$, NS], trait anxiety [$F(1,22) = .35$, NS] and alcohol craving [$F(1,22) = 1.57$, NS], confirming the correct matching of groups. These results are described in Table 1.

3.2. Behavioural data

These results are shown in Fig. 1. 6 × 2 ANOVAs with morph step (5–20–35–65–80–95% of fear) as within-factor and group (CR, BD) as between-factor were conducted separately for reaction times and behavioural responses, with post-hoc paired-samples t-tests:

- Reaction times: we found no significant group effect [$F(1,22) = .39$, NS] and no group X morph step interaction [$F(5,110) = 1.23$, NS], but obtained a significant main effect of morph step [$F(1,22) = 94.78$, $p < .001$] which was due to shorter reaction times to the less ambiguous morphs.
- Behavioural responses: we found no group X morph step interaction [$F(5,110) = .73$, NS] but a significant group effect [$F(1,22) = 9.62$, $p < .01$] was identified: BD presented lower correct response rates than CR, independently of the morph step. We also obtained a signif-

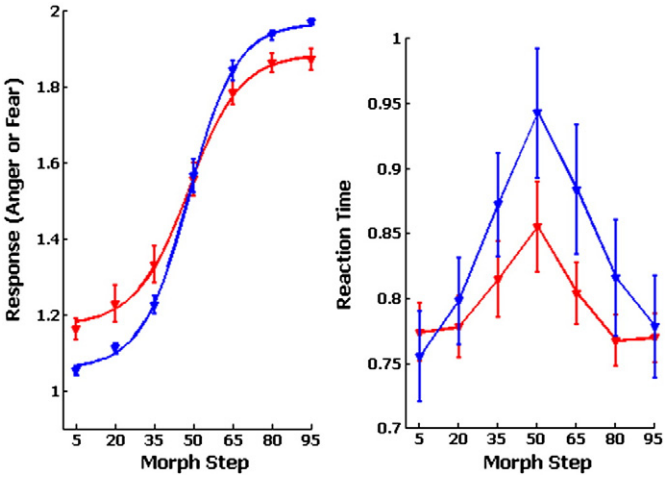

Fig. 1. Behavioural results for each morph step (numbers represent the percentage of fear in the affective bursts) among controls (in blue) and binge drinkers (in red). The left part shows the mean response (from 1 "anger" to 2 "fear") and the right part depicts the mean reaction time (in seconds). Error bars represent standard error of the mean (S.E.M.).

icant main effect of morph step [$F(5,110) = 24.94$, $p < .001$], which was driven by higher correct response rates for the less ambiguous morphs.

3.3. fMRI data

The voice localizer showed the classical activations in areas along bilateral superior temporal sulcus/superior temporal gyrus (Belin et al., 2000) for both groups with no significant group differences. The 7 × 2 full-factorial model with morph step as within-factor and group (CR, BD) as between-factor showed two main effects:

- Main effect of morph step (Table 2, part a): Both anger (i.e. 5–20–35% morph steps) and fear (i.e. 65–80–95% morph steps) bursts activated right cerebellum, left inferior frontal gyrus, left supplementary motor area and bilateral superior temporal gyri. Moreover, activations were found in bilateral thalami for anger stimuli and in left insula, right amygdala and right medial frontal gyrus for fear stimuli. The main effect of morph step showed that: (1) anger stimuli led to significantly higher activations than fear ones in left inferior frontal gyrus and supplementary motor area; and (2) fear stimuli led to higher activations than anger in bilateral insula and right medial frontal gyrus.
- Main effect of group (Table 2, part b): Both groups presented significant activations in right cerebellum, right inferior and medial frontal gyri, bilateral superior temporal gyri and left thalamus. Moreover, activations were found in left inferior parietal lobule for binge drinkers. The main effect of group showed that: (1) control participants presented higher activations than binge drinkers in bilateral superior temporal gyri; and (2) binge drinkers presented higher activations than controls in right middle frontal gyrus. These results are illustrated in Fig. 2.
- No significant activations were found for the interaction between group and morph step.

3.4. Correlational analyses

Pearson's correlations were performed to explore the links between:

(1.) Behavioural results and cerebral activations: A significant positive correlation was found between behavioural accuracy (i.e. percentage of correct responses) and peak voxel activity in the superior temporal gyri ($r = 0.435$, $p < .05$), higher activation in the superior temporal gyri thus being related to better performance. Moreover, a significant negative correlation was found between behavioural reaction times and peak voxel activity in the middle

Table 2
Brain regions showing significant activation related to the main effect of emotion (anger versus fear stimuli, part A) and to the main effect of group [control participants (CR) versus binge drinkers (BD), part B].

Contrast	Brain area	x	y	z	BA	L/R	k	t-Statistic	p-Value
(A) Main effect of emotion type									
Anger	Cerebellum	9	−64	−14	/	R	148	8.72	<.0001
	Inferior frontal gyrus	−48	5	40	9	L	53	5.73	<.001
	Superior temporal gyrus	−51	−19	7	22	L	1079	20.76	<.0001
	Superior temporal gyrus	54	−16	4	22	R	885	17.34	<.0001
	Supplementary motor area	−6	5	56	32	L	69	8.94	<.0001
	Thalamus	−12	−16	7	/	L	223	8.08	<.0001
	Thalamus	12	−13	7	/	R	26	5.9	<.05
Fear	Amygdala	21	−6	−13	/	R	84	6.15	<.05
	Cerebellum	9	−59	−10	30	R	192	9.52	<.0001
	Inferior frontal gyrus	−48	5	34	9	L	29	6.21	<.05
	Insula	−39	−1	−3	13	L	296	8.16	<.05
	Medial frontal gyrus	51	2	44	6	R	20	6.72	<.05
	Superior temporal gyrus	−53	−21	7	22	L	1046	19.41	<.0001
	Superior temporal gyrus	63	−25	4	22	R	901	16.11	<.0001
	Supplementary motor area	−9	18	46	32	L	41	8.37	<.001
Anger > fear	Inferior frontal gyrus	−45	17	10	44	L	26	5.95	<.05
	Supplementary motor area	9	23	49	32	R	27	6.13	<.05
Fear > anger	Insula	33	22	7	13	R	36	5.32	<.05
	Insula	−33	20	4	13	L	41	5.18	<.05
	Medial frontal gyrus	9	52	4	10	R	359	5.71	<.05
(B) Main effect of group									
CR	Cerebellum	18	−52	−20	/	R	112	8.04	<.0001
	Inferior frontal gyrus	42	38	16	46	R	67	6.77	<.001
	Medial frontal gyrus	51	2	46	6	R	26	7.56	<.05
	Superior temporal gyrus	−51	−30	1	22	L	879	15.81	<.0001
	Superior temporal gyrus	54	−19	1	22	R	822	15.14	<.0001
	Thalamus	−12	−16	7	/	L	79	6.52	<.0001
BD	Cerebellum	24	−49	−20	/	R	32	5.98	<.05
	Inferior frontal gyrus	42	7	36	9	R	51	5.77	<.001
	Inferior parietal lobule	−36	−37	39	40	L	41	5.4	<.001
	Medial frontal gyrus	9	8	49	6	R	74	8	<.001
	Superior temporal gyrus	−54	−21	4	22	L	755	14.06	<.0001
	Superior temporal gyrus	54	−16	4	22	R	443	9.63	<.0001
	Thalamus	−18	−13	17	/	L	93	5.95	<.0001
CR > BD	Superior temporal gyrus	−57	−31	4	22	L	41	5.49	<.05
	Superior temporal gyrus	60	−22	−2	22	R	124	6.32	<.0001
BD > CR	Middle frontal gyrus	42	38	25	46	R	101	4.95	<.05

x, y and z are stereotaxic coordinates of peak-height voxels.
BA = Brodmann's area, L = left hemisphere, R = right hemisphere, k = cluster size.
Threshold set at $p < .05$ FWE corrected with a minimum cluster size of 20 contiguous voxels.
Presented data result from the contrast between the mean hemodynamic responses evoked by each experimental condition and those evoked by the silent baseline periods between runs (e.g. "anger" = mean cerebral activations during anger stimuli presentation minus mean cerebral activations during silent baseline/"CR" = mean cerebral activations shown by controls during the task minus mean cerebral activations shown by controls during silent baseline).

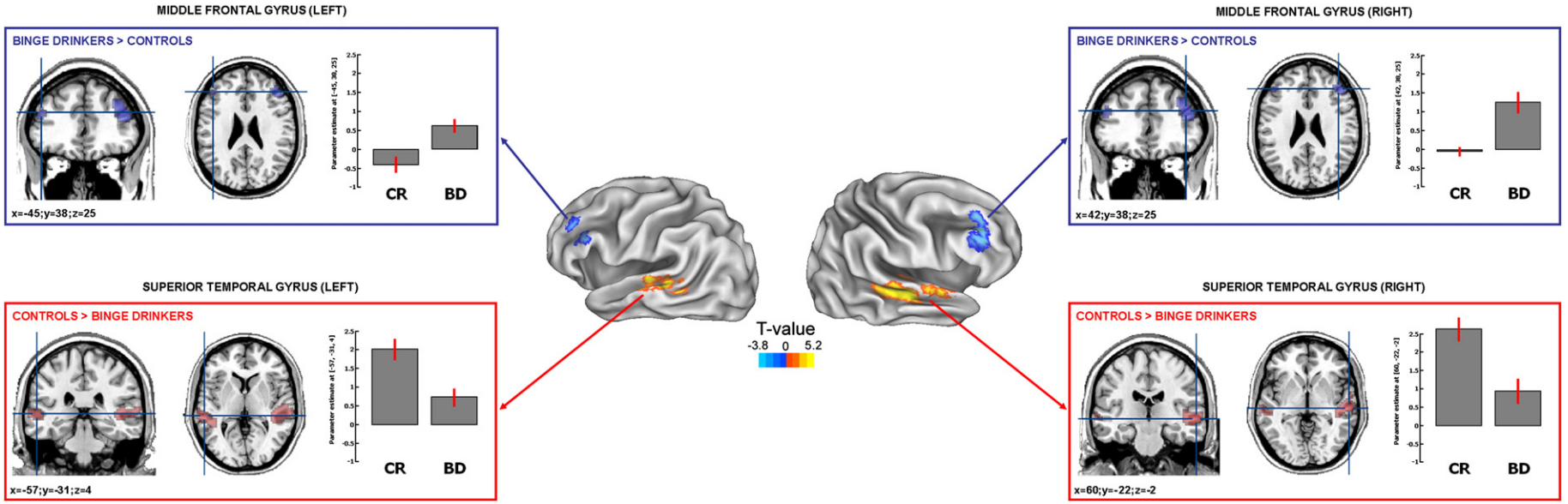

Fig. 2. Neuroimaging results for the group comparison (across all morph steps) between controls (CR) and binge drinkers (BD). Activations are illustrated on inflated cortex and SPM template for $p < .001$ (uncorrected) with an extent threshold of 40 voxels. Contrast of controls > binge drinkers is illustrated in red while the contrast of binge drinkers > controls is shown in blue. Bar graphs represent parameter estimates with error bars (S.E.M.). All activations (except left frontal) survive FWE-correction at the cluster level (see Table 2b).

frontal gyri ($r = -0.387$, $p < .05$), higher activation in the middle frontal gyri thus being related to shorter reaction times.

(2.) Superior temporal and middle frontal gyrus activations: a significant negative correlation was found between the peak voxel activity in the superior temporal and middle frontal gyri ($r = -0.338$, $p < .05$) showing that a reduced activation of the superior temporal gyri is linked with increased activation in the middle frontal gyri.

4. Discussion

This study was the first to explore the cerebral correlates of emotional processing in binge drinking, and the results clearly suggest that binge drinkers present impaired behavioural and cerebral processing of emotional bursts. At the behavioural level, binge drinkers presented altered processing of negative prosody, which constitutes the primary observation of impaired emotional decoding in this population. At the neuroimaging level, the classical activation pattern related to emotional bursts processing was totally replicated: In both groups, both emotions activated the bilateral superior temporal gyri (the key region for human voice processing (Beaucousin et al., 2007; Belin et al., 2000; Ethofer et al., 2009a, 2012; Grandjean et al., 2005)) and the left inferior frontal gyrus (involved in the conscious categorization of emotional sounds (Ethofer et al., 2009b; Wildgruber et al., 2005)). Moreover, in line with earlier results, fear led to increased activations in the amygdala, insula and medial frontal gyrus (Mothes-Lasch et al., 2011), while anger led to increased activations in the inferior frontal gyrus and supplementary motor area (Sander et al., 2005). More centrally, two main group differences were found: (1) Binge drinkers had reduced activations in bilateral superior temporal gyri, which is involved in the processing of affective bursts (Beaucousin et al., 2007; Ethofer et al., 2009a, 2012; Grandjean et al., 2005). This result corroborates behavioural ones and constitutes the first description of the cerebral correlates of impaired prosody processing in binge drinking; and (2) Binge drinkers had increased activations in the right middle frontal gyrus. This region is involved in the processing of the social characteristics of human voice (Szameitat et al., 2010) and is strongly connected with the superior temporal gyrus (Ethofer et al., 2012), but is not directly involved in the decoding of affective bursts.

These increased frontal activations among binge drinkers reflect the enhanced involvement of alternative areas usually not activated during affective bursts processing, and could index a compensatory activity aiming at counterbalancing the reduced activations in the superior temporal gyri. Earlier studies already showed increased frontal and parietal activations among populations with alcohol-related problems to compensate for impaired temporal activations during memory tasks (Campanella et al., 2013; Schweinsburg et al., 2010). The present results reinforce this "compensation hypothesis" suggesting that binge drinking leads to a double functional change: reduced activation of the areas typically activated during the task and increased activation of alternative regions to compensate for this deficit. This suggestion is further reinforced by the correlational analyses, as they showed (1) that higher activity in the middle frontal gyri is associated with improved performance (i.e. faster reaction times), confirming that increasing the activity of this area can lead to faster emotional processing and (2) centrally, that the activations of the superior temporal gyri and middle frontal gyri are negatively correlated, which support the proposal that the increased middle frontal gyrus activations might compensate altered superior temporal gyrus functioning. Our results also support the "continuum hypothesis" (Enoch, 2006) suggesting that binge drinking and alcohol-dependence could constitute two successive steps of a same pathology, leading to analogous impairments. Indeed, as alcohol-dependence is associated with marked emotion decoding deficits (Maurage et al., 2008a, 2009b) and marked grey matter loss in the superior temporal gyri (Demirakca et al., 2011), the similarities between our results in binge drinking and earlier ones in alcohol-dependence are in line with the proposal of a continuum in the brain deficits between these two populations. This "continuum hypothesis" should nevertheless be specifically tested in studies offering a direct comparison between binge drinkers and alcohol-dependent individuals. Moreover, the cerebral correlates of the transition from binge drinking to alcohol-dependence should also be explored, as it could be postulated that binge drinkers are still able to activate alternative brain areas to compensate for impaired activations, while during the evolution towards alcohol-dependence, this compensation might disappear due to the generalisation of brain impairments, particularly in frontal areas (Bühler and Mann, 2011b; Taki et al., 2006).

Centrally, our results confirmed our main hypothesis by showing for the first time that cerebral changes in binge drinking are present not only during cognitive processes but also during affective ones, and that binge drinking is deleterious not only for the adolescent brain but also among young adults. Importantly, the strict control of comorbidities ensures that these cerebral changes observed are related to alcohol consumption per se and not to other drug dependence or psychopathological states. It should however be noted that, while we postulate (in line with earlier longitudinal studies (Maurage et al., 2009a; Norman et al., 2011; Squeglia et al., 2009)) that these cerebral changes are the direct consequence of alcohol consumption, the reverse causation cannot be totally excluded as several brain modifications could precede and influence the development of binge drinking habits (Squeglia et al., 2012b). The absence of group effect concerning current alcohol craving also suggests that the results are not due to stronger alcohol-related thoughts (which might have impaired the performance) among binge drinkers. In view of the limited sample size and variety of emotional bursts, future studies on larger populations are needed to confirm these results, notably to explore the specificity of these deficits for emotional processing (by including control non-emotional stimuli) and the persistence of these brain changes when binge drinking habits end. Moreover, while the participants were repeatedly asked to avoid any alcohol consumption during the three days preceding scanning, the absence of acute alcohol consumption measure at testing day does not allow to totally make sure that no participant had consumed alcohol in the days preceding scanning. Despite these limits, this study clearly shows that binge drinking is associated with impaired emotional processing, as indexed by reduced performance in affective bursts categorization and decreased activations in superior temporal gyri. Moreover, the increased activations in an alternative voice processing network among binge drinkers support the "compensation hypothesis" (Campanella et al., 2013; Schweinsburg et al., 2010, 2011) and show that binge drinking leads to a reorganisation of brain functioning, combining reduced and increased activations.

In conclusion, the present results offer the first insights concerning the cerebral correlates of emotional impairments in binge drinking and could constitute a first step towards the development of the affective neurosciences of binge drinking, potentially bearing crucial fundamental and clinical implications. At the experimental level, this could shed new light on the causes and development of alcohol-dependence by underlining the involvement of emotional impairments in the early stages of this pathology. At the therapeutic level, understanding emotional impairments in binge drinking could assist in the development of prophylactic interventions such as focusing on the rehabilitation of emotional and interpersonal skills. Our results highlight that binge drinking, despite constituting a widespread alcohol consumption habit among young people in Western countries, is associated with deleterious effects at behavioural and cerebral levels. We contribute to a growing body of literature emphasising the urgent need for more education among binge drinkers and for a reconsideration of public health policies among adolescents and young adults.

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