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Latent deleterious effects of binge drinking over a short period of time revealed only by electrophysiological measures

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Background: Episodic excessive alcohol consumption (i.e., binge drinking) is now considered to be a major public health problem, but whereas short- and long-term harmful consequences of this behaviour are clearly established at medical, social and cognitive levels, the cerebral correlates of these impairments are still unknown. Our study explores the midterm cerebral effects of binge-drinking behaviours among young adults. **Methods:** We selected 2 groups of first-year university students with no history of drinking habits, paired for psychological and behavioural measures on the basis of their expected alcohol consumption during the forthcoming academic year. The binge drinker group expected to have high personal alcohol consumption, whereas the control group expected low consumption. We used a test–retest paradigm within a 9-month period (session 1 in September 2005, session 2 in May 2006). At each testing session, we recorded auditory event-related potentials while the participants performed an emotional valence judgment task. **Results:** There were no differences between the groups in behavioural or electrophysiological measures at baseline. After 9 months, the binge drinkers had significantly delayed latencies for all event-related potential components (P1, N2, P3b) of emotional auditory processing compared with the control group ($p < 0.006$), with no behavioural differences. **Limitations:** As the present study explored the electrophysiological correlates of binge drinking with an emotional task only, the results will have to be extended to other cognitive processes using various experimental tasks. **Conclusion:** We report the first direct evidence that short-term binge drinking can produce marked cerebral dysfunction undetectable by behavioural measures alone. The observed latency abnormalities, similar to those observed in long-term alcoholism, constitute an electrophysiological marker of slowed cerebral activity associated with binge drinking.

Contexte : La consommation excessive épisodique d'alcool (alcoolisation paroxystique intermittente) est désormais considérée comme un problème de santé publique majeur. Or, si les conséquences néfastes de ce comportement à brève et à longue échéance sont clairement établies du point de vue médical, social et cognitif, les corrélats cérébraux de ces effets sont encore inconnus. Notre étude se penche sur les conséquences de ce comportement sur le cerveau des jeunes adultes à moyen terme. **Méthodes :** Nous avons sélectionné 2 groupes d'étudiants universitaires de première année n'ayant pas d'antécédents de consommation régulière d'alcool et nous les avons appariés en fonction de variables psychologiques et comportementales sur la base de leur consommation prévue d'alcool au cours de l'année universitaire qui allait débuter. Les participants du groupe susceptible de s'adonner à l'alcoolisation paroxystique intermittente s'attendaient à consommer personnellement beaucoup d'alcool, tandis que ceux du groupe témoin s'attendaient à en consommer peu. Nous avons utilisé un paradigme test–retest échelonné sur une période de 9 mois (première séance, septembre 2005; deuxième séance, mai 2006). Lors de chaque séance d'évaluation, nous avons enregistré les potentiels évoqués, tandis que les participants effectuaient une tâche liée au jugement de la valence émotionnelle. **Résultats :** Il n'y avait aucune différence entre les groupes pour ce qui est des paramètres comportementaux ou électrophysiologiques au départ. Après 9 mois, les adeptes de l'alcoolisation paroxystique intermittente présentaient des latences significativement prolongées pour toutes les composantes des potentiels évoqués (P1, N2, P3b) dans le traitement auditif des stimuli émotionnels, comparativement au groupe témoin ($p < 0{,}006$), sans différences sur le

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plan du comportement. **Limites :** Étant donné que la présente étude portait uniquement sur les corrélats électrophysiologiques de l'alcoolisation paroxystique intermittente lors d'une tâche émotionnelle, les résultats devront être appliqués à d'autres processus cognitifs au moyen de tâches expérimentales diverses. **Conclusion :** Nous rapportons la première preuve directe selon laquelle ce comportement peut produire à court terme une dysfonction cérébrale marquée, qui ne sera pas décelable au moyen d'échelles comportementales uniquement. Les anomalies de latence observées, semblables à celles que l'on note dans l'alcoolisme chronique, sont des marqueurs électrophysiologiques du ralentissement de l'activité cérébrale associé à l'alcoolisation paroxystique intermittente.

Introduction

Adolescents and young adults are at high risk of initiating alcohol use, which can lead to the development of later alcohol use disorders[1] and is considered to be a major public health problem.[2] Epidemiological studies show that binge drinking, the repeated excessive consumption of alcohol over a short period of time, affects about 40% of 18- to 24-year-olds.[3] Although a single massive intake of alcohol may have dramatic acute consequences (e.g., increased risk of motor vehicle collisions, alcoholic coma, uninhibited sexual or violent behaviour[4,5]), repeated alcohol intake over a long period leads to negative long-term medical (e.g., cardiovascular disorders, gastrointestinal diseases, malignant neoplasms[4]) and social (e.g., poor academic results, problems of social integration[6]) effects. The neurotoxicity induced by long-term chronic alcoholism is also well established.[7] Animal studies show that the adolescent brain, still immature and in a critical period of remodelling and development, is particularly sensitive to alcohol.[8] For instance, the hippocampus is more sensitive to the acute effects of ethanol and its neurotoxic effects during adolescence.[9] In adults with alcohol addiction, alterations in brain metabolites are observed in the frontal, cerebellar and thalamic regions, consistent with regional axonal damage (as inferred from *N*-acetylaspartate concentrations) and with changes in glial and general cell membrane metabolism.[10] Moreover, voxel-based morphometry of grey matter revealed a susbtantial decrease in density in the precentral gyrus, middle frontal gyrus, insular cortex, dorsal hippocampus, anterior thalamus and cerebellum; there was also reduced density of white matter in the periventricular area, pons and cerebellar pedunculi.[11] These structural brain connectivity alterations have clear deleterious effects on cognition. Indeed, various cognitive abilities, such as the allocation of attentional resources,[12–14] visuo-spatial processing, short-term memory storage abilities[15] and executive functions[16] have been reported to be altered by long-term chronic alcohol abuse.[17]

With ambiguous messages about some positive medicinal effects of moderate alcohol consumption (e.g., reduced risk of heart attack[18,19] or cognitive decline[20]), the detrimental effects of short-term alcohol consumption are not clear-cut. Young drinkers are often confused about what constitutes "moderate consumption" and what damage they may actually be doing to their health in general and to their brains in particular, especially because metabolic alterations and behavioural deficits have so far only been described after several years of binge drinking.[10,13,16] Although the consequences of alcohol intake among young adults have been widely explored in studies focusing on behavioural and electrophysiological impairments owing to a positive family history of alcoholism,[21,22] acute alcohol consumption[23,24] or heavy drinking,[25,26] the chronic midterm cerebral consequences of excessive alcohol consumption have not yet been explored in a controlled test–retest paradigm.

Because binge drinking is characterized by repeated periods of alcohol intoxication and abstinence leading to multiple withdrawals that are particularly deleterious for brain function,[27,28] we hypothesized that cognitive impairments due to short-term binge drinking remain latent and that these impairments could be revealed by appropriate measures. Therefore, we used event-related potentials (ERPs), which allow us to monitor the electrical activity of the brain with high temporal resolution and detect even minor neurocognitive restrictions that are undetectable at the behavioural level.[29] On the basis of the self-expected alcohol consumption, we selected 2 groups of 18 participants (controls and binge drinkers) and used a test–retest paradigm with a 9-month period separating the 2 testing sessions, corresponding to the beginning and the end of the participants' first academic year. We recorded ERPs while the participants performed an emotional valence judgment task on auditory stimuli expressing negative or positive emotions. This task has been shown to be highly impaired in chronic alcoholic individuals who have a reduced amplitude and a delayed latency for the P3b, a long-lasting positive component appearing at parietal sites between 300 and 800 ms after stimulus onset and indexing the decisional and executive processing,[30] but also earlier components associated with perceptual processes (i.e., P1, N2 and P2 waves).[31] We thus expected functional impairments in binge drinkers on the same ERP components as those affected in chronic alcoholic individuals.

Methods

Participants

We screened a total of 462 students beginning their first academic year in the Faculty of Psychology, Catholic University of Louvain, Belgium. The screening was anonymous and comprised 2 parts:

- psychological measures evaluating anxiety (State-Trait Anxiety Inventory, A and B[32]), depression (Beck Depression Inventory[33]), interpersonal problems (quantity and quality of social interactions, integration in the family and relationship background[34]) and alexithymia;[35] and
- a 75-item questionnaire, adapted from a binge-drinking habits questionnaire,[36] evaluating previous and future alcohol–drug consumption, family history of alcohol

consumption, social integration and medical problems.

To be included in our study, the students had to meet the following selection criteria: no positive family history of alcohol dependence; very low past alcohol consumption (mean consumption during 6 months preceding the study 1.61, standard deviation [SD] 1.84 units per week, where a unit corresponds with 10 g of pure ethanol); total absence of past binge-drinking habits; total absence of past or current drug consumption (including tobacco and any medication); no major medical problems; no central nervous system disease (including epilepsy); no auditory impairment; no moderate or high depression–anxiety scores; and no history of psychiatric disorder. The final selection criterion was the expected alcohol consumption during the academic year. We individually paired each member of the binge drinker group with a control participant for age, sex, education and psychological measures (state and trait anxiety, depression, interpersonal problems and alexithymia). We evaluated alcohol consumption based on individual self-estimated reports. All participants abstained from alcohol for at least 3 days before each testing session.

We provided participants with full details regarding the aims of our study and the procedure before they gave their informed consent. The ethical committee of the Faculty of Psychology of the University of Louvain approved our study.

Stimuli

We administered an auditory task based on emotional valence detection (negative or positive) at each testing session. The participants had to decide which emotion was evoked by the stimulus by pressing 1 of 2 buttons with their right forefinger. The stimuli were audio recordings selected from a standardized battery[37] consisting of the enunciation of a semantically neutral word ("paper") with 2 emotional prosodies (anger or happiness) and 4 voices (2 female, 2 male) for each prosody. We manually edited all recordings to equilibrate pitch and duration. We presented each auditory stimulus with an intensity of 70 dB.

Experimental design and procedure

The first testing session took place in September 2005; the second testing session occurred at the end of the same academic year in May 2006. During the ERP recording session, the participants sat in a dark room on a chair with their heads restrained in a chin rest; the auditory stimuli were presented via binaural headphones. The study consisted of 8 blocks of 32 stimuli (16 for each emotion); the order of the blocks varied across participants. A trial consisted of the following series of events: stimulus presented for 700 ms, an 800-ms time period during which the answer could still be given, and an interstimulus interval of random duration between 0 and 500 msec. The total duration of a trial thus varied from 1500 to 2000 ms, and the participants had 1500 ms to answer. We recorded response latencies and accuracy. The instructions emphasized the need to respond as quickly as possible while keeping errors to a minimum.

Recording ERPs

We obtained electroencephalograms (EEGs) using 32 electrodes mounted in an electrode Quick-Cap. Electrode positions included the standard 10–20 system locations and intermediate positions. Recordings were taken with a linked mastoid physical reference but rereferenced using a common average. We used battery-operated amplifiers (Advanced Neuro Technology) with a gain of 30 000 and a band-pass of 0.01–100 Hz to amplify the EEGs. The impedance of all electrodes was always kept below 5 kW. We manually eliminated trials contaminated by electrooculogram artifacts (mean of 11%) off-line. Epochs were created starting 200 ms before stimulus onset and lasting for 1500 ms. Data were filtered using a 30 Hz low-pass filter. To compute different averages of ERP target stimuli for each participant, we coded 2 parameters for each stimulus: the condition type (anger or happiness) and the response type (correct or incorrect).

Statistical analysis

We considered only correct responses for the analysis of response latencies and ERPs. For each participant and each component of interest (P1, N2 and P3b), we obtained individual peak amplitudes and maximum peak latencies from several electrodes separately for the ERPs to each type of stimulus: Oz, O1, O2, T5 and T6 for the early primary perceptive processing of auditory information (P1);[38] T5 and T6 for the specific perceptive processing of human voices (N2);[39] Pz, P3 and P4 for the decisional processes associated with the closure of the cognitive activities (P3b).[40] We tested these values using repeated-measures analysis of variance (ANOVA, Greenhouse–Geisser correction applied when appropriate) and paired sample *t* tests.

Finally, we performed complementary analyses in the binge-drinker group to test the following effects.

- Potential sex effect: Whereas groups were perfectly matched for sex, excluding a general influence of sex on the observed group differences, sex could influence the drinking pattern and/or the intensity of the latency impairment. We thus formed 2 subgroups in the binge drinker group according to sex, and we performed independent sample *t* tests to assess the potential sex influence on the quantity of alcohol consumed at Session 2 and the latency delays (as reported in Table 1) observed among binge drinkers for P1, N2 and P3b at Session 2.
- Potential acute binge-drinking effect: Although all participants abstained from alcohol for at least 3 days before each testing session, the results in the binge drinker group could have been influenced by the last alcohol consumption. In other words, performance could vary as a function of the number of days elapsed since the last alcohol consumption (mean 8.34, SD 2.78) or of the number of doses consumed during this last occasion (mean 10.7, SD 3.91). To test this potential influence, we performed Pearson correlations between these last alcohol consumption characteristics and every behavioural–electrophysiological result.
- The hypothesis of a link between latency impairments

observed among binge drinkers for successive ERP components (see "Difference" line in Table 1 for the mean and SD of the delays for P1, N2 and P3b): Indeed, earlier studies[41] in alcoholism postulated a continuum between the deficits described for the ERP components associated with perceptual, attentional and decisional stages. The greater the latency delay for perceptual stages (P100), the greater the delay for subsequent attention and decisional stages. To test this, we performed Pearson correlations between the delays observed for the 3 ERP components (P1, N2 and P3b).

- The influence of binge-drinking intensity on the latency impairment observed at Session 2 among binge drinkers: Although a general latency delay is observed in the binge-drinker group, it is not clear whether this deficit varies according to the amount of alcohol intake (i.e., whether this deficit is proportional to the intensity of binge drinking habits). To test this hypothesis, we performed Pearson correlations in the binge drinker group between the mean alcohol consumption at Session 2 (in doses per week) and the latency delay observed for P1, N2 and P3b (see Table 1 for the mean and SD of these delays).

Results

Participants

Of the 462 students we screened, 50 met our inclusion criteria. Of these, 25 participants expected to have a personal alcohol consumption greater than 20 units per week and were assigned to the binge-drinker group; the other 25 expected to have an alcohol consumption less than 3 units per week and were assigned to the control group. Seven participants in the binge-drinking group did not complete the study: 2 abandoned their studies, 2 had an alcohol consumption less than 10 units per week, and 3 smoked tobacco or cannabis. We excluded the 7 paired controls from the study, which left 2 groups of 18 participants for inclusion in our analysis. The mean age of participants was 18.16 (SD 0.86) years, and there were 7 men and 11 women in each group.

Alcohol consumption and psychological measures

The 2 groups did not differ in their alcohol consumption at the first session ($t_{17} = 0.43$, $p = 0.67$), but at the second session binge drinkers had a higher total alcohol consumption ($t_{17} = 7.01$, $p < 0.001$), number of alcohol consumption occasions per week ($t_{17} = 9.62$, $p < 0.001$) and number of drinks per occasion ($t_{17} = 11.84$, $p < 0.001$) than participants in the control group. There were no group differences in depression ($t_{17} = 1.93$, $p = 0.07$), trait ($t_{17} = 0.92$, $p = 0.37$) or state ($t_{17} = 2.02$, $p = 0.06$) anxiety, interpersonal problems ($t_{17} = 1.31$, $p = 0.21$) or alexithymia ($t_{17} = 0.73$, $p = 0.47$) at the second session, which suggests that the experimental results were not influenced by these psychological factors. These data are summarized in Table 2.

Behavioural results

A 2×2 ANOVA with session (first, second) as a within-subject factor and group (binge drinkers, controls) as a between-subject factor revealed a main effect of session, with longer latencies at the first session than the second (mean 789, SD 87.5 ms v. mean 765.4, SD 95.7 ms, respectively; $F_{1,34} = 8.94$, $p = 0.005$), but there was no main effect of group ($F_{1,34} = 0.16$, $p = 0.68$) and no interaction ($F_{1,34} = 0.023$, $p = 0.88$). There was no main effect or interaction for accuracy (session $F_{1,34} = 0.56$, $p = 0.46$; group $F_{1,34} = 2.19$, $p = 0.15$; interaction $F_{1,34} = 0.17$, $p = 0.68$).

Electrophysiological results

For each electrophysiological component of interest (P1, N2 and P3b), we computed a 3-way repeated-measure ANOVA with session (first, second) and localization (Oz, O1, O2, T5 and T6 for P1; T5 and T6 for N2; Pz, P3 and P4 for the P3b) as within-subject factors and group (binge drinkers, controls) as a between-subject factor separately for latencies and amplitudes (Table 1). These results are illustrated in Figure 1. For latencies, there was no main effect of group (except on P1,

Table 1: Latencies and amplitudes of each electrophysiological component as a function of group and session

	ERP component; mean (SD)					
	P1		N2		P3b	
Group; session	Latency, msec	Amplitude, mv	Latency, msec	Amplitude, mv	Latency, msec	Amplitude, mv
Binge drinkers						
Session 1	124 (12.5)	2.87 (1.06)	216 (17.8)	–2.49 (1.54)	407 (78.6)	3.83 (1.72)
Session 2	133 (21.6)	2.96 (1.19)	230 (17.9)	–2.24 (1.27)	481 (89.3)	3.82 (1.93)
Difference*	8.94 (18.5)	0.09 (1.56)	13.83 (27.38)	0.005 (0.77)	73.07 (89.07)	–0.006 (1.55)
Controls						
Session 1	126 (12.6)	2.47 (1.33)	222 (24.2)	–2.41 (1.75)	422 (71.3)	4.62 (3.04)
Session 2	110 (11.4)	2.51 (1.48)	201 (18.2)	–2.07 (1.23)	411 (98.4)	4.07 (1.86)
Difference*	–15.87 (16.22)	0.05 (1.54)	–20.61 (21.59)	–0.67 (1.39)	–11.88 (84.23)	–0.55 (1.53)

ERP = event-related potential; SD = standard deviation.
*Results represent the mean variation of latency–amplitude values between sessions 1 and 2 (session 2 – session 1). The positive differences for latencies among binge drinkers illustrate latency delays at session 2 in this group (with the reverse pattern among controls).

but this effect was qualified by a group by session interaction) or session. We observed significant interactions between group and session for each electrophysiological component. The 2 groups did not differ at the first session, but at the second session binge drinkers had a significantly delayed latency for the ERP components functionally associated with the P1, N2 and P3b components. For the P1 component ($F_{1,34} = 18.24$, $p < 0.001$), the binge drinkers had longer P1 latencies than controls at session 2 ($t_{17} = 4.11$, $p < 0.001$) but not at session 1. For the N2 component ($F_{1,34} = 18.24$, $p < 0.001$), the binge drinkers had longer N2 latencies than controls at session 2 ($t_{17} = 2.96$, $p = 0.009$) but not at session 1. For the P3b component ($F_{1,34} = 8.65$, $p = 0.006$), the binge drinkers had longer P3b latencies than controls at session 2 ($t_{17} = 2.43$, $p = 0.023$) but not at session 1. This was not modulated by the topography of the electrodes (all $p > 0.3$). Finally, there were no main effects or interactions for the amplitudes of the various components.

Complementary analyses

Our complementary analyses on the binge drinker group showed the following.

- There was no sex influence on the quantity of alcohol consumed at session 2 ($t_{16} = 1.31$, $p = 0.21$) or on the latency delays at session 2 for P1 ($t_{16} = 0.76$, $p = 0.46$), N2 ($t_{16} = 0.48$, $p = 0.63$) and P3b ($t_{16} = 1.19$, $p = 0.25$). These results suggest that sex did not influence alcohol consumption pattern nor the intensity of the latency impairment among binge drinkers.

Table 2: Group characteristics for binge drinkers and controls

	Group; mean (SD)*	
Characteristic	Binge drinkers	Controls
Sex, women:men	11:7	11:7
Age, yr†	18.17 (0.38)	18.21 (0.31)
Alcohol consumption, units per wk		
Session 1†	1.99 (1.85)	1.41 (2.88)
Session 2‡	35.17 (19.72)	1.11 (2.93)
Duration of binge drinking habits at session 2, mo	8.75 (0.11)	NA
No. of alcohol units§ per binge drinking occasion at session 2	12.52 (4.48)	NA
No. of binge drinking occasions per wk at session 2	2.33 (1.02)	NA
BDI†	3.17 (1.94)	5.17 (0.45)
STAI A†	36.28 (9.22)	40.00 (11.47)
STAI B†	38.17 (5.77)	43.72 (9.87)
IIP†	1.05 (0.45)	1.29 (0.51)
TAS-20†	49.56 (7.22)	46.78 (12.69)

BDI = Beck Depression Inventory;[27] IIP = Inventory of Interpersonal Problems;[28] NA = not applicable; SD = standard deviation; STAI = State and Trait Anxiety Inventory;[26] TAS-20 = 20-item Toronto Alexithymia Scale[29]
*Unless otherwise indicated.
†No significant differences observed between binge drinkers and controls.
‡$p < 0.001$.
§One unit represents 10 g of alcohol.

- There was no significant correlation between last alcohol consumption characteristics and behavioural or electrophysiological results at session 1 or 2 ($p > 0.05$ for every correlation).
- We observed significant positive correlations between the delays for the 3 ERP components (P1–N2 $r = 0.537$, $p = 0.021$; P1–P3b $r = 0.731$, $p = 0.001$; N2–P3b $r = 0.622$, $p = 0.007$). The more important the latency impairment at the perceptual level (P1), the more important it will be at subsequent levels (N2 and P3b), which confirms among binge drinkers the observation made in earlier studies among alcoholic patients.
- We observed significant positive correlations between mean alcohol intake and each ERP component (P1 $r = 0.531$, $p = 0.022$; N2 $r = 0.593$, $p = 0.009$; P3b $r = 0.619$, $p = 0.007$), suggesting that the latency impairment is proportionate to the intensity of binge drinking habits.

Discussion

Knowledge about the cerebral effects of repeated excessive consumption of alcohol over short periods of time in adolescents and young adults is tremendously lacking. Our study compared young adult binge drinkers with perfectly matched paired controls before they started their drinking habits. Whereas the 2 groups did not differ on any psychological, behavioural or electrophysiological measures at the first session, after only 9 months the binge drinkers had a significantly delayed latency in the electrophysiological components indexing perceptive as well as decisional processes. It is noteworthy that this electrophysiological deficit was observed in the absence of any psychological or behavioural differences. Indeed, the only significant behavioural effect was a main effect of session (namely shorter reaction times during session 2 as compared with session 1) owing to greater familiarity with stimuli in session 2, which led to the classic test–retest effect. These ERP impairments concerned the latency of ERP components functionally associated with the early primary perceptual processing of information (P1), the specific perceptual processing of human voices (N2) and the decisional process associated with the closure of cognitive activities before starting the motor response (P3b). The deficits started with the early perceptual processes and lasted until the later decisional processes (as shown in Fig. 1). Importantly, complementary analyses suggested that the extent of these latency delays is proportionate to the severity of binge drinking behaviours. These latency abnormalities constitute the electrophysiological marker of a dysfunctional and slowed cerebral activity during the cognitive processing of complex stimuli. Various degenerative cerebral diseases and brain damage are known to cause abnormalities restricted to the latencies of ERP components. For instance, brain infarction slightly delays the P3b latency, without affecting its amplitude or scalp distribution.[42] Anatomically, this P3b latency variability is mainly related to white matter connectivity,[43] also known to be altered by alcohol consumption.[11] This fits perfectly with the fact that only the latencies were affected in our study.

Behavioural deficits have already been described in patients

with alcohol abuse but only after several years of binge drinking. Our results show that cerebral dysfunctions appear early and, importantly, before any detectable behavioural impairments. The use of modern electrophysiological and neuroimaging techniques, therefore, appears fundamental to correctly evaluate the actual level of impairment produced by

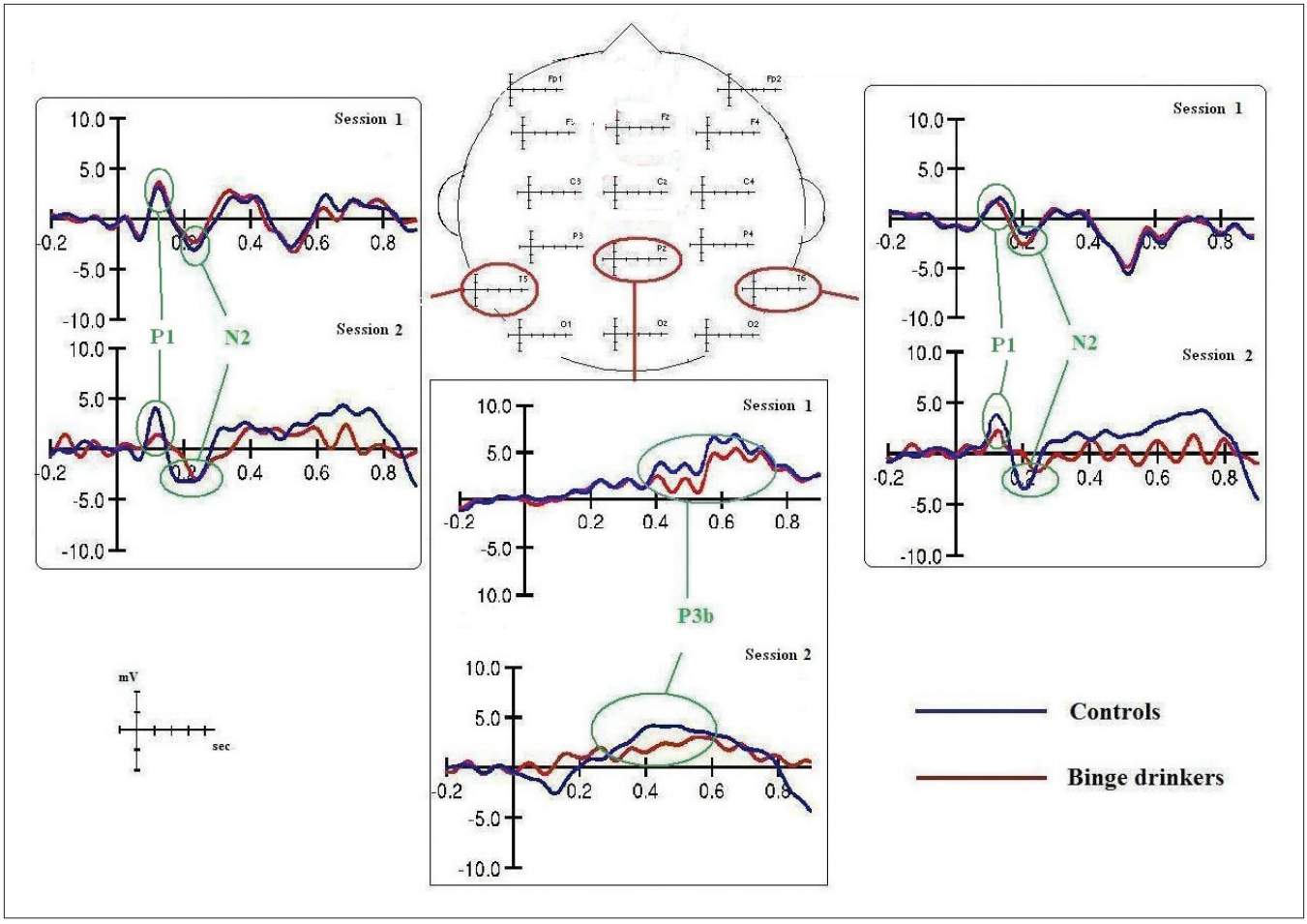

Fig. 1: Delayed latencies observed only at session 2 for binge drinkers on the P1 and N2 components, recorded at occipito-temporal sites (T5, T6), and the P3b component, recorded at parietal sites (Pz). These components overlapped at session 1. We observed the slowed cerebral processing for binge drinkers at session 2 at occipito-temporal and parietal sites.

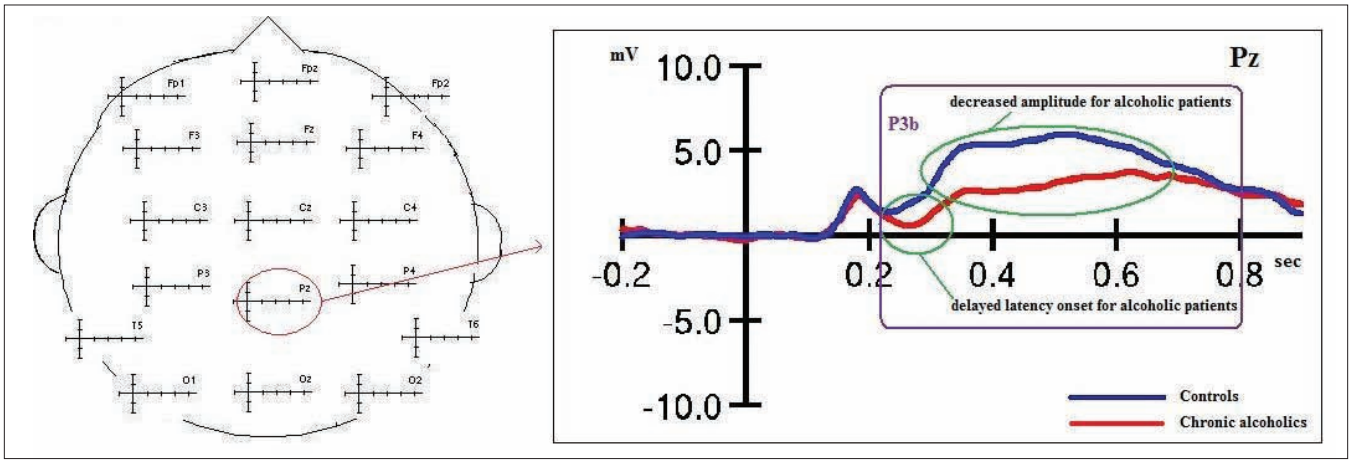

Fig. 2: Delayed and reduced P3b component recorded at parietal site (Pz) in adult chronic alcohol users in an emotional auditory task (adapted from Maurage et al.[44]).

alcohol consumption. The presence of an ERP latency abnormality, which reflects a neurophysiologically delayed transmission between neural sources, is necessarily linked to minor neurocognitive restrictions that may not affect psychometric measures. The cognitive effects of binge drinking, which had so far been evaluated only on the basis of behavioural measures, could thus have been largely underestimated.

Finally, our results show that the ERP delays observed in binge drinkers are similar, even if less marked, to the neurophysiological deficits observed in chronic alcoholic individuals (as illustrated in Fig. 2).[44,45] This supports the idea that binge drinking and chronic alcoholism may represent 2 stages of the same phenomenon.[46] There is indeed a clear parallelism between the observed deficits. First, the perceptual deficit (namely for the P1 and N2 components) is absent in several other psychiatric disorders, including depression, anxiety or antisocial personality disorders. Second, the deficits in the binge drinkers were observed in the same complex emotional valence judgment task as that impaired in chronic alcoholic individuals.[47] Third, as shown in the complementary analyses, the correlations between the deficits observed in successive ERP components, which had been described in earlier studies among alcoholic individuals,[41] are also shown here among binge drinkers, confirming the parallelism in the deficits presented by these 2 populations. Our results show that even a short period of binge drinking is sufficient to lead to abnormal delayed ERP latencies, possibly representing a first step before extending to ERP amplitude values, as in chronic alcoholism. Our observations raise again the major health issue as to whether binge drinking is or is not an open door to chronic alcoholism. At the very least, our results highlight the need to study the patterns and correlates of binge drinking trajectories from early adolescence into adulthood. More broadly, showing that frequent binge drinking (i.e., more than 10 doses at least 2 times a week) rapidly leads to cerebral dysfunctions should lead public health officials to tackle this problem, notably by developing information programs and brief motivational interventions among college student drinkers.[48]

Limitations

Although based on a sound test–retest paradigm with a high control of potential bias, our results need to be generalized and extended. First, we only used stimuli from 1 sensory modality, namely audition. Future studies should thus generalize these results to other modalities, particularly vision. Moreover, our task was based on emotional judgments only, therefore we cannot rule out the possibility that the observed deficit was specific to emotional processing. Complementary work is thus needed to confirm that these alterations will also be observed using nonemotional stimuli. Finally, if electrophysiological data recorded here gave very useful insight into the deficits described at a temporal level (namely, a delay in the ERP components), nothing is known about the cerebral areas responsible for the alterations observed. The localization of the brain structures affected by binge drinking is thus a major priority for future studies.

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Contributors: Drs. Maurage and Campanella designed the study. Dr. Maurage acquired the data, which all authors analyzed. Drs. Maurage, Pesenti and Campanella wrote the article, which Dr. Philippot reviewed. All authors gave final approval for publication.

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Correction

Genotype over diagnosis in amygdala responsiveness: affective processing in social anxiety disorder

In the print version of the article by T. Furmark, S. Henningsson, L. Appel, F. Ahs, C. Linnman, A. Pissiota, V. Faria, L. Oreland, M. Bani, E. Merlo Pich, E. Eriksson and M. Fredrikson (*JPN* 2009;34[1]:30–40), the title was incorrectly printed as "Genotype over-diagnosis in amydala responsiveness: affective processing in social anxiety disorder." This affected only the print version; for correct citations, please refer to the online version, available at cma.ca/jpn.

We apologize for this error.