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Escola de Psicologia

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Mestrado Integrado em Psicologia

Trabalho realizado sob orientação do

Doutor Eduardo López Caneda

e do

Doutor Alberto Crego

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Declaração

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Os Efeitos da Ressaca no Controlo Inibitório em Jovens *Binge Drinkers*: Um Estudo de Potenciais Evocados Relacionados com Eventos

Resumo

O Binge drinking (BD), é um padrão comum entre os jovens que envolve o consumo excessivo durante um curto período, seguido por abstinência. A ressaca, relacionada com o BD, é caracterizada por sintomas aversivos que seguem o consumo excessivo. Alguns estudos têm demonstrado que a ressaca pode afetar o rendimento cognitivo. No entanto, nenhum estudo explorou as consequências comportamentais e electrofisológicas da ressaca no dia seguinte a um episódio BD habitual. Com o propósito de compreender como o controlo inibitório é afetado pela ressaca, medidas comportamentais e a atividade cerebral foram registadas enquanto sete estudantes universitários BDs realizaram uma tarefa Go/NoGo antes e depois de uma noite BD habitual. Os tempos de reação; percentagem de respostas correctas e de inibições corretas; a amplitude e latência das componentes P2, N2, P3 e da componente positiva tardia (LPC) foram analisados. Os resultados não revelaram efeitos da ressaca a nível comportamental. No entanto, observaram-se menores amplitudes da P2 e maiores amplitudes da N2 a seguir ao episódio BD quando comparado com o dia normal sem consumo de álcool. Isto sugere que, durante a ressaca os mecanismos atencionais e os processos de monitorização de conflitos parecem estar comprometidos.

Palavras-chave: binge drinking, ressaca, control inibitório, electroencefalografia, estudantes universitários

The Effects of Hangover on Inhibitory Control in Young Binge Drinkers: An Event-Related Potentials Study

Abstract

Binge Drinking (BD) is a pattern widespread among youngsters that involves heavy drinking over a short period followed by abstinence. Hangover, related to BD, is characterized by aversive symptoms, which follow the excessive drinking. Some studies have demonstrated that hangover may affect cognitive performance. However, no study has explored the behavioural and electrophysiological consequences of hangover on the day after a regular BD episode. Aiming to understand how inhibitory control may be affected by hangover, behavioural measurements and brain activity were recorded while seven college student BDs performed a Go/NoGo task before and after a typical BD night. The reaction times; percentages of correct responses and correct inhibitions; the amplitude and latency of the P2, N2, P3 components and of the Late Positive Component (LPC) were analysed. The results revealed no hangover effects at a behavioural level. However, lower P2 amplitudes and greater N2 amplitudes were observed the day after a BD episode when compared to the normal day without alcohol consumption. This suggests that during the hangover state the attentional resources and the conflict monitoring processes seem to be impaired.

Keywords: binge drinking, hangover, inhibitory control, electroencephalography, college students

The Effects of Hangover on Inhibitory Control in Young Binge Drinkers: An Event-related Potentials Study

During adolescence and at the time of entering university, young people seek new sensations and they are more likely to engage in high-risk behaviors, such as drug abuse (Spear, 2000; Zeigler et al., 2005). The drug which is most widely used by youngsters and the overall population is alcohol (World Health Organization, 2014).

Recent reports from European and American national health agencies have shown that heavy alcohol use is frequently associated with major social and health consequences such as motor-vehicle accidents, poor academic performance, use of other drugs and risky sexual behavior (Eurobarometer, 2010; National Institute of Alcohol Abuse and Alcoholism, 2015). Likewise, alcohol consumption has particularly high economic costs – reaching up to 2-5% of the gross domestic product in the European Union (Anderson & Baumberg, 2006) - and largely contributes to the number of deaths in both the United States and Europe, where alcohol is directly or indirectly involved in over 30% of all deaths (WHO, 2011).

The consumption is higher in Europe, where 86% of the citizens aged between 15 to 24 reported having drunk alcohol in the last 30 days (Eurobarometer, 2010). In Portugal, alcohol is also the most common intoxicant used by the population in general, including youngsters and young adults (15-24 years old), who demonstrated a consumption rate of 38% in the last month (Balsa, Vital, & Urbano, 2018).

Excessive drinking can lead to a specific pattern known as binge drinking (BD), which is rather common among youngsters and more significantly among college students (NIAAA, 2015; Eurobarometer, 2010). This pattern is characterised by an excessive intake of alcohol over a brief period of time, namely four (or more) drinks in two hours for women and five (or more) drinks in the same time span for men, until the blood alcohol concentration (BAC) reaches 0.08g/dL or above (NIAAA, 2004). To be considered BD, these episodes of heavy alcohol consumption need to occur at least once in two weeks or in a month and be followed by a period of abstinence (Courtney & Polich, 2009).

Epidemiological data have shown that in Europe there is a prevalence rate of 33% of BD among people between 15 and 24 years old (Eurobarometer, 2010). As for Portugal, the BD prevalence rate is higher in the population with ages between 15 and 24, where it is around 30% (Balsa et al., 2014). Furthermore, a more recent report showed that 6% of young people (15-24 years old) had one or more BD episodes per month in the last 12 months (Balsa et al., 2018)

Alcohol in the Adolescent and Young Brain

Adolescence and youth are critical periods of development, because the brain undergoes major structural and functional changes (Bava & Tapert, 2010). It is when synaptic pruning, myelination and modifications in the neurotransmitters occur and when the neuromaturation of prefrontal cortex and other high-order association areas are taking place (Bava & Tapert, 2010; Lenroot & Giedd, 2006). Altogether, these changes in brain morphology lead to deep changes in cognitive, emotional and behavioural levels (Bava & Tapert, 2010). Regarding the cognitive level, this period is characterized by the development and refinement of high-order functions, such as attention, working memory and inhibitory control, which are mainly linked to the maturation of the frontal areas (De Luca & Leventer, 2008).

Thus, alcohol consumption during this neurodevelopmental window is particularly worrying, given that excessive drinking might disrupt the maturational processes of these still-in-development regions and consequently have a negative impact on behavioural, cognitive and psychosocial levels (Squeglia, Jacobus, & Tapert, 2009). This assumption seems to have found support in animal studies, from which it has been observed that adolescent rats with a heavy alcohol consumption suffer more brain damage than adult rats with the same pattern of alcohol use, namely in the prefrontal cortex and in the limbic system (Crews, Braun, Hoplight, Switzer, & Knapp, 2000; Pascual, Pla, Miñarro, & Guerri, 2013).

Effects of binge drinking in adolescent and young brain

The changes in brain development caused by early alcohol consumption can lead to future cognitive problems, which may manifest through functional consequences over the time (Jacobus & Tapert, 2013). Many studies in humans have reported several implications associated with BD, such as structural and functional brain impairments and poor cognitive performance (López-Caneda et al., 2014; Petit, Maurage, Korneich, Verbanck, & Campanella, 2013). More specifically, this pattern has been associated with low performance in tasks related to attention, memory, and executive functioning (Courtney & Polich, 200; López-Caneda et al., 2014c). Regarding the executive functions, the maintenance of this pattern seems to impair working memory, decision making, and inhibitory control (Courtney & Polich, 2009; López-Caneda et al., 2014c).

Among the different cognitive functions affected by the BD pattern, the one that deserves special attention is the inhibitory control (López-Caneda, Holguín, Cavadeira, Corral, & Doallo, 2014a). This cognitive process is a key function of human behaviour, that enables the suppression and the control of

inappropriate or impulsive responses and actions (Diamond, 2013). Thus, this executive function allows people to adapt their behaviour according to the demands of the surrounding environment and to their personal goals. Accordingly, the impairment of this human ability may contribute to the maintenance of this pattern, since it may compromise the individuals' ability to prevent or stop alcohol consumption (Carbia, Corral, Doallo, & Caamaño-Isorna, 2018).

ERP studies

The event-related potentials (ERPs) is an electroencephalographic (EEG) technique with high temporal resolution that has been broadly used to explore not only the chronic effects of longstanding alcohol use, but also the effects of BD on the electrical brain responses linked to cognitive processes (Hill & O'Brien, 2015; Petit et al., 2013). Studies using this technique have frequently observed that young BDs, in comparison with controls, display abnormal amplitude and/or latency values in several components associated with different cognitive functions (Petit et al., 2013).

Some ERPs studies showed that, despite the absence of significant effects at the behavioural level, BDs presented larger amplitudes than age-matched controls in components related to attention (P3 and N2), working memory (P3) and inhibitory control processes (NoGo-P3) (Crego et al., 2009, 2012; López-Caneda et al., 2012, 2013; López-Caneda, Rodríguez-Holguín, Corral, Doallo, & Cadaveira, 2014b; Smith, Iredale, & Mattick, 2016; Smith & Mattick, 2013; Watson, Newton-Mora, & Pirkle, 2016). These results were interpreted as a compensatory neurofunctional mechanism that allowed BDs to perform the task adequately. These findings are in line with functional Magnetic Resonance Imaging (fMRI) studies, which reported that young BDs exhibited greater brain response linked to different cognitive processes in comparison with healthy controls, despite not showing behavioural impairments (Campanella et al., 2013; Schweinsburg, McQueeny, Nagel, Eyler, & Tapert, 2010; Xiao et al., 2013). In sum, the BD pattern has been related to abnormalities in brain functioning. Specifically, BDs seem to recruit additional neural resources to maintain a similar cognitive performance than matched controls without this pattern of consumption.

Inhibitory Control

ERP studies have frequently used Go/NoGo paradigms to explore the neural correlates of response inhibition, that is, the behavioural dimension of inhibitory control (Diamond, 2013). During this task,

subjects need to respond to some trials (Go stimuli) and to inhibit their response to others (NoGo stimuli), evaluating thus their response execution and response inhibition. There are two major ERP components typically recorded throughout this task, the N2 and the P3. The NoGo-N2 is a negative deflection that peaks around 200-300ms after the stimulus presentation and its maximum amplitude occurs at fronto-central scalp locations. This component is followed by the NoGo-P3, a positive wave with a fronto-central distribution on the scalp that peaks between 300-500ms post-stimulus. These components seem to represent different sub-processes of response inhibition. While the NoGo-N2 has been linked to conflict monitoring processes (Donkers & Boxtel, 2004), the NoGo-P3 has been related to motor inhibition itself (Smith, Johnstone, & Barry, 2008). Consequently, the presence of abnormalities in either or both components may reflect an impairment on the inhibitory control processes (Euser & Franken, 2012).

The ERP studies concerning the effects of social and binge drinking on inhibitory control have found anomalies in both the latency (Petit, Kornreich, Noël, Verbanck, & Campanella, 2012) and, more frequently, the amplitude (López-Caneda et al., 2012; López-Caneda et al., 2014b; Smith et al., 2016; Smith & Mattick, 2013) of the NoGo-P3 component. In a follow-up study, López-Caneda et al. (2012), found larger NoGo-P3 amplitudes associated with a hyperactivation of the right inferior prefrontal cortex in young BDs. These results were interpreted as a compensation mechanism that allowed BDs to properly inhibit their response (López-Caneda et al., 2012). Similarly, Smith and Mattick (2013), using a stop-signal task, found that young BDs showed a larger Stop-P3 amplitude (an analogous component to NoGo-P3) during correct inhibitions as compared to failed inhibitions, which was suggestive of a greater cognitive effort in BDs for inhibiting their response properly (Smith & Mattick, 2013).

Petit et al. (2012) investigated young social drinkers during a visual Go/NoGo task and found delayed latencies of NoGo-P3. This delay on the latencies was associated with poor inhibitory performance (i.e., more commission errors) in these subjects (Petit et al., 2012).

Nevertheless, in a recent study, during a visual Go/NoGo task no significant differences on the ERP components were observed between BDs and controls (López-Caneda et al., 2017). However, in this study the BDs exhibited lower oscillatory responses in delta and theta frequency ranges in both Go and NoGo conditions (López-Caneda et al., 2017). These results suggested that the BDs, like alcohol dependent subjects, seem to present disruptions in neural oscillations associated with the motor inhibition and execution (López-Caneda et al., 2017).

Hangover

Besides the detrimental neurocognitive effects linked to the maintenance of BD, there are also important consequences immediately after a BD episode. One of these major consequences is directly related to the hangover experience (Wiese, Shlipak, & Browner, 2000).

The concept of hangover, closely related to BD, can be described as a series of unpleasant physical and mental symptoms, namely headache, nausea, thirst, drowsiness, sleepiness, dry mouth, weakness, concentration problems and reduced alertness, which follow the intake of a great quantity of alcohol (Penning, van Nuland, Fliervoet, Olivier, & Verster, 2010). All of these symptoms are especially significant when BAC reaches 0 g/dL and may persist within 24 hours (Prat, Adan, & Sánchez-Turet, 2009). Furthermore, their intensity can vary depending on the person or the occasion and their severity and frequency can be influenced both by the quantity of alcohol and the concentrations of congeners in the alcoholic beverages (Penning et al., 2010; Wiese et al., 2000). The congeners are complex organic molecules, which are present mainly in the darker drinks, such as wine, whiskey or tequila (Prat et al, 2009; Stephens, Ling, Heffernan, Heather, & Jones, 2008). These alcoholic beverages seem to cause more severe hangover symptoms than the clear drinks, that is, vodka or gin (Champan, 1970).

The phenomenon of hangover is commonly experienced by college students, as reflected in the fact that around 56% of them report having experienced at least one hangover episode during the previous month (Penning, McKinney, & Verster, 2012).

Although the causes of hangover remain unclear, there are several theories trying to explain its emergence and severity. According to them, hangover symptoms may result from direct or indirect alcohol effects on the body or the combination of both (Finnigan, Schulze, Smallwood, & Helander, 2005; Prat et al, 2009). Thereby, hangover may be caused by alcohol withdrawal, the toxic effects of the congeners contained in the alcoholic beverages or by the effects of alcohol metabolites, like acetaldehyde (AcH) (Swift & Davidson, 1998; Wiese et al., 2000). Dehydration, cytokine changes, hormonal abnormalities, alterations in blood sugar levels, and sleep or biological rhythm disturbances have also been proposed to explain the alcohol hangover symptoms (Swift & Davidson, 1998; Wiese et al., 2000).

The role of AcH, one of the main metabolic products of ethanol, has been largely discussed (Swift & Davidson, 1998). AcH itself and its metabolism have a significant role in diverse effects of alcohol (Eriksson, 2001). Raised concentrations of AcH lead to a characteristic set of symptoms such as increased

heart and respiratory rate, lowered blood pressure, sensation of dry mouth or throat, sweating, subjective feeling of heat, blushing, nausea, headache, vomiting and euphoria (Erikson, 2001; Swift & Davidson, 1998). Apart from euphoria, all of these reactions are analogous to the symptoms experienced during alcohol hangover. Therefore, and despite AcH is absent when BAC reaches 0%, researchers have proposed that this metabolite may cause hangover (McGregor, 2007; Yokoyama et al., 2005).

Though the economic and social impact of hangover are indeterminate and difficult to quantify, it is important to consider them and they deserve attention (Stephens et al., 2008). In this sense, beyond the fact that hangover can lead to interpersonal conflicts, enhance accident risk, and cause productivity loss, its negative effects have also been related to absenteeism and poor performance in academic and work contexts. (Foster & Vaughan, 2005; Prat et al., 2009; Wiese et al., 2000).

Hangover studies

While there are numerous studies on the acute effects of excessive alcohol consumption in humans, the consequences during the hangover period have been largely neglected (Ling, Stephens, & Heffernan, 2010; Verster, 2008). The studies that have been conducted in this field adopted three different approaches: retrospective, experimental and naturalistic.

Most of the retrospective studies, where subjects are assessed a few days after alcohol consumption, did not find significant impairments in neurocognitive performance (Prat, Adan, Pérez-Pàmies, & Sànchez-Turet., 2008). Nevertheless, some of them revealed a negative association between the quantity of alcohol consumed reported by the participants and the scores obtained in scales related to cognitive performance (Prat et al., 2008).

In the experimental studies, hangover is induced in the laboratory context and the subjects' performance is assessed when BAC decreases or reaches zero (Stephens et al., 2008). Most of these studies analyzed the effects of hangover on attention (Collins & Chiles, 1980; Chait & Perry, 1994; Finning, Hammersley, & Cooper, 1998; Howland et al., 2010; Lemon, Chesher, Fox, Greely, & Nabke, 1993). However, only two of them (25 %) found impairments during hangover on this neurocognitive function (Howland et al., 2010; Roehrs et al., 1991). In the study of Howland et al. (2010), college students with a BD pattern also showed deficits on visuospatial and motor functions, besides the impairments on attention. The study of Verster, van Duin, Volkerts, Schreuder, and Marinus (2003) revealed that hangover may lead to impairments on memory, more specifically on delayed recall. Similarly, Kim, Yoon, Lee, Choi, and Go

(2003) also found long-term memory impairments during hangover; however, given that BAC was not zero, it is unclear whether this was an acute intoxication effect or not (Kim et al., 2003).

Finally, in the naturalistic studies, subjects are assessed after an intoxication in a habitual context when BAC reaches zero. The results of these studies pointed to impairments on the neurocognitive performance. More specifically, when college students were assessed in the morning after a night of habitual alcohol intake, impairments on long-term memory, attention and psychomotor performance were found (McKinney & Coyle, 2004; McKinney, Coyle, & Verster, 2012). Anderson and Dawson (1999) also showed decrements in attention, namely in divided and focused attention, after 12h-16h of alcohol consumption in a naturalistic context (Anderson & Dawson, 1999). However, in another study detrimental effects linked to hangover were not found during vigilance, attention and short-memory tasks (Finnigan et al., 2005).

Regarding the hangover effects at a neurophysiological level, to the best of our knowledge there are only two laboratory studies that have examined the neural response during the hangover state. In these studies, the participants were assessed in three moments: sober, intoxicated and under hangover (Stock, Hoffmann & Beste, 2016; Wolff, Gussek, Stock & Beste, 2016). One of them used a task switching paradigm to explore the effects on cognitive flexibility and they found no differences between the sober and the hangover appointments in the amplitude and latency of the ERP components analyzed (P1, N1 and N2). These results suggested that the cognitive flexibility processes are not affected during the hangover (Wolff et al., 2016). The other one evaluated the sub-processes involved in the decision to select a response by a moving dots paradigm (Stock et al., 2016). The participants revealed shorter N2 latencies and larger N2 amplitudes in hangover state than when they were sober. According to the authors, this suggests that there is a greater conflict in response selection during hangover than during a sober state (Stock et al., 2016).

Altogether, these studies seem to indicate that alcohol hangover may involve major impairments on different cognitive processes, namely on long-term memory, attention, psychomotor performance and in response selection by the increased conflict monitoring. However, to the best of our knowledge, no study has been conducted with the aim of assessing the electrophysiological consequences of alcohol hangover after a typical night of alcohol consumption despite the important implications that might result from this research. Consequently, the present study seeks to fill in the existing gap by assessing the electrophysiological response linked to inhibitory control processes of young BDs before and after a BD episode.

Aims

The aim of this study was to examine for the first time the brain activity of young BDs the day after a typical BD episode. Specifically, we aimed to evaluate the electrophysiological response associated with inhibitory control processes in college students with a BD pattern by means of ERPs before and after a typical alcohol consumption. For this purpose, we analyzed the latency and amplitude of the main components traditionally linked to inhibitory control, namely N2 and P3 along with other two components commonly linked to attentional (P2) and working memory (late positive component, LPC) processes. Additionally, another goal was to examine the effects of hangover at the behavioural level measuring the reaction times as well as the percentage of correct responses and the percentage of correct inhibitions during task performance.

It was hypothesized that, in the day after a BD session (i.e., during the hangover state) subjects would show poorer task performance in comparison with a normal day without alcohol consumption, as reflected by longer reaction times and lower percentages of correct responses and/or correct inhibitions. In addition, concerning brain activity, it is expected that BDs would exhibit electrophysiological anomalies in the hangover condition, particularly during response inhibition, showing lower amplitudes and higher latencies in the ERP components.

Method

Participants

For the participants' recruitment, the experiment was available on the experiments' platform of Psychology School (EPsi) of University of Minho (UM). The psychology students that were interested in participating could register through this platform. The experiment had a value of 1.6 credits, which were attributed to the participants at the end of the semester. Students from other courses of UM and from University Católica of Braga were contacted by the researchers to participate in the experiment. A total of 39 Portuguese college students performed a clinical interview aimed at obtaining information regarding alcohol and drugs consumption, personality characteristics and medical history. Although 17 subjects met the inclusion criteria, only seven (five females) of these participants aged between 19 and 24 (M = 20.7; SD = 1.8) years were included in the posterior analysis of the present master thesis.

The exclusionary criteria were as follows: loss of consciousness lasting more than 20 minutes; non-

corrected sensory deficits; personal history of psychopathological disorders (according to DSM-5); family history of alcoholism or substance abuse in first degree relatives; personal history of alcohol use disorder according to DSM-5; Alcohol Use Disorder Identification Test (AUDIT) scores equal to or greater than 20; consumption of medication with psychoactive effects during the week prior to the evaluation; and use of illegal drugs (except cannabis). Considering the above criteria, 16 participants were excluded. Additionally, four subjects were excluded because did not met the age range (18-25 years) and two participants gave up the study. Taking into account the NIAAA's BD definition (NIAAA, 2004), the participants who drunk five or more alcoholic drinks (four or more for women) on the same occasion at least once a month during the last year, were classified as BDs.

Instruments

Alcohol Use Disorder Identification Test (AUDIT; Saunders, Aasland, Babor, De la Fuente, & Grant, 1993; Portuguese version: Gomes, 2014). The AUDIT is a self-reported measure that is used to identify current harmful and hazardous drinking. In the present study, this instrument allowed to determine whether the participants fulfilled the BD criteria (question 3: "How often do you have six or more drinks on the same occasion?"; one of the following answers was required: "At least once a month"; "At least once a week"; or "Daily or almost daily").

Clinical History Interview. The clinical interview consisted of a set of questions to explore the medical history of the participant and of their close relatives (first, second and third-degree).

Drugs Use Disorder Identification Test Extended (DUDIT-E; Berman, Palmstierna, Källmén, & Bergman, 2007). This test assesses the frequency and the positive and negative aspects of illicit drug use, and the treatment readiness. In the present study this instrument allowed to determine whether the students consume psychoactive drugs or other substances than alcohol.

Penn Alcohol Craving Scale (PACS; Flannery, Volpicelli & Pettinati, 1999; Portuguese version: Pombo, Ismail, & Cardoso, 2008). It is a self-report measure with five items, rated from 0 to 6, which assesses the frequency, intensity, and duration of craving, the resistance to drinking as well as the overall craving for alcohol, during the previous week. The higher score, the higher alcohol craving.

Edinburgh Handedness Inventory (Oldfield, 1971; Portuguese version: Espírito-Santo, Pires, Garcia, Daniel, Silva & Fazio, 2017). It is an inventory with 10 items which evaluates handedness.

Barratt Impulsivity Scale-11 (BIS-11; Patton, Stanford e Barratt, 1995; Portuguese version: Cruz & Barbosa, 2012). It is a self-reported questionnaire composed of 30 items, rated from 1 (never or rarely) to

4 (often/always), designed to assess the personality/behavioral construct of impulsiveness.

Symptom Checklist-90-Revised (SCL-90-R; Franke, 1995; Portuguese version: Laloni, 2001). It is a self-reported measure composed of 90 items. The participants had to rate on a five-point scale, ranging from 0 (not at all) to 4 (extremely), how much they were concerned about each symptom in the last week. This questionnaire assesses nine symptoms of psychopathology and provides three global distress indices.

Breathalyzer test. This test measures the breath alcohol level. In this study it was used to verify that the blood alcohol concentration (BAC) value was 0.0% at the assessment moment.

EEG Session Checklist. This instrument is constituted by a number of questions (e.g., number of sleeping hours, time since last meal, last day they drank alcohol, etc.) that allows to verify if the previous requirements for conducting the EEG recording are fulfil.

Alcohol Hangover Severity Scale (AHSS; Penning, McKinney, Bus, Olivier, Slot, & Verster, 2013). It is a scale composed of 12 items designed to assess the overall hangover severity. The participants had to rate on a ten-point scale, ranging from 0 (absent) to 10 (extreme), the severity of each symptom that they were experienced.

AlcoDroid Application. It is a mobile application in which the participants had to register when they drank and which alcoholic beverages they drank. This instrument allowed to determine whether the subject reached a BAC value of 0.08 g/dL or above, and to estimate when the BAC value returns to zero.

Procedure

All participants provided written informed consent prior to assessment. The experiment was divided into three moments. Firstly, the participants were evaluated through a clinical interview. Then, if they met the inclusion criteria, they were assessed twice: (1) on a typical day without alcohol consumption; and (2) during a hangover state, namely after a night with regular BD. During each assessment, EEG was recorded while the participants performed three different tasks aimed at evaluating their cognitive performance. Each one of these two sessions had a duration of 90 minutes.

The moment in which participants performed the first assessment was counterbalanced to avoid the training effect. Thus, half of the sample did the first assessment on a typical day without alcohol consumption and the other half were assessed during the hangover state.

Before the assessments, it was sent to the participants a text message with the criteria that they had to fulfil to perform the experiment. When the assessment was performed on a typical non-BD day, they could

not have consumed alcohol on the previous day nor had a BD episode on the last three days. Moreover, they could not smoke, drink tea or coffee in the three hours prior to the test or consume cannabis during that day. When the assessment was performed during the hangover state, participants could not consume other substances beyond alcohol and needed to sleep between six and nine hours before the data collection.

Before the EEG recording, it was collected a saliva sample of 3 ml with the aim of conducting biochemical analysis (e.g., pro-inflammatory cytokines) not reported in this study.

During the EEG recording, the BD subjects sat on chair in an experimental room of the EPsi and performed three tasks: A alcohol novelty oddball task, to evaluate attentional bias to alcohol-related cues; a continuous performance task (CPT), in order to assess working memory; and a Go/NoGo task, with the aim of evaluating inhibitory control. In the present study, only data obtained by the Go/NoGo task are reported. The order of the three tasks was randomized for all participants and it was ensured that this order was different in the first and second assessment for each participant. After the first two tasks, the brain activity during the resting state was recorded (three minutes with eyes open and three minutes with eyes closed).

The study was approved by the Ethic Subcommission of Social and Human Sciences of the UM and the procedure was undertaken in accordance with the Code of Ethical Principles for Medical Research Involving Humans Subjects outlined in the Declaration of Helsinki (Brazil, 2013).

Go/NoGo Task

During this task, the participants were instructed to fixate on a small cross located centrally on a LCD monitor, which was located 100cm from the participant. Squares or circles were presented at a visual angle of 3 X 3° for 50 ms over the cross, with a 1000-1400ms inter-stimulus interval (onset-onset). The number of stimuli was ranged between 140 and 160. The participants had to press a button with their preferred-hand in response to the Go stimuli (green circle and blue square) and not to respond to the NoGo stimuli (blue circle and green square). Stimuli were presented equiprobably in a randomized order.

EEG recording

The electroencephalogram (EEG) was recorded using the ActiveTwo Biosemi electrode system (Biosemi, Inc.) from 64 electrodes organized according the 10-10 system (American Clinical Neurophysiology Society, 2006) digitized at 512 Hz. Vertical and horizontal electro-oculogram activity was

recorded to control for eye movements and blinks. An additional two electrodes were placed on the mastoids bilaterally to provide the signal reference. Electrode impedances were kept below 20 kΩ and the EEG signal was filtered on-line with a 0.01–100 Hz band pass filter.

Data analysis

Demographic and drinking characteristics analysis

A Paired-samples t-test was used to explore the differences between the two moments in the PACS score and in the hours of sleep before the EEG assessment. Pearson correlations were also performed to explore the association between the PACS score obtained during the hangover state and both AHSS score and number of drinks consumed during the BD night.

Behavioural analysis

Only responses occurring between 100–1000 ms after the onset of a Go stimulus were considered as correct responses. No-responses to NoGo stimuli were scored as correct inhibitions. Reaction time (RT) and percentage of correct responses and correct inhibitions were analyzed by paired-samples t-tests.

ERP analysis

For the ERP analysis, data were processed with BrainVision Analyser software (Version 2.1). The EEG signal was corrected for vertical and horizontal ocular artifacts by Independent Component Analysis (ICA). It was then digitally filtered off-line with a 0.1–30 Hz band-pass filter and segmented into epochs of 1000 ms (from −100 to 900 ms). Baseline correction was applied; epochs exceeding ±80 μV at any scalp electrode was rejected and EEG epochs corresponding to incorrect responses (omissions or false alarms) were excluded. The number of retained trials was similar across conditions (Go and NoGo) for the two moments ($p = .214$): 63.14±14.43 (Go – Normal day); 63.00±12.34 (NoGo - Normal day); 53.14±10.76 (Go – Hangover state); and 62.86±16.20 (NoGo – Hangover state).

The EEG epochs corresponding to Go and NoGo trials were independently averaged. The ERPs were analyzed by an automatic peak detection procedure, subsequently reviewed, and manually corrected at each electrode. The components of both conditions were identified in the averaged waveforms elicited by Go and NoGo stimuli as the largest peak between 150 and 250 ms (P2), 200 and 325 ms (N2), 300 and 450 ms (P3), and between 500 and 700 ms (LPC) after the stimuli onset. Amplitude (mV) and Latency (ms) values of the four components were obtained from specific electrodes: FC3, FCz, FC4, C3, Cz, and C4 for the P2 component; F3, Fz, F4, FC3, FCz, and FC4 for the N2 component; CP3, CPz, CP4, P3, Pz, and

P4 for the P3 component; and F3, Fz, F4, FC3, FCz, and FC4 for the LPC component. A repeated-measures analysis of variance (ANOVA) with three within-subject factors (Moment: normal day and hangover state; Condition: Go and NoGo; and Electrode), was used to analyze each component separately (alpha level ≤.05). Whenever appropriate, degrees of freedom were corrected by the conservative Greenhousee-Geisser estimate. All post hoc paired comparisons were performed with the Bonferroni adjustment for multiple comparisons, also with an alpha level ≤ .05.

Results

Demographic and drinking characteristics

The demographic and drinking variables are presented in the table 1.

Table 1

Demograhic and drinking characteristics of the participants

	Participants
N (females)	7(5)
Age	20.71±1.79
Handedness (right/left)	6/1
Regular tobacco smokers	4
Regular use of cannabis	0
Age of onset of regular drinking	16.29±1.38
Number of times consuming 6 or more drinks in a day per month	2±0
Number of drinks in a standard drinking episode	12.14±9.82
Number of drinks consumed per hour (speed of alcohol consumption)	2.28±.48
Number of drinks consumed in the BD night before the assessment	10.57±3.10
Sleep hours before the assessment on normal day	7.43±.79*
Sleep hours before the assessment on hangover state	6.71±1.11*
PACS score normal day	1.71±1.70*
PACS score hangover state	4.00±2.82*
AHSS score	53.85±19.49
BIS-11 total score	68.57±5.74
Global Symptom Index (GSI) score (SCL-90-R)	19.43±24.26
Total AUDIT score	10.86±4.14

* $p < .05$

There was a significant difference in the sleep hours before the assessment on a normal day and

during hangover state; [t(6)= 2.50, p= .047]. These results revealed that the participants slept more hours before the EEG assessment on a normal day (M= 7.43; SD= .79) than before the assessment during hangover state (M= 6.71; SD= 1.11).

Regarding the PACS score, the results revealed significant differences between the scores obtained on the normal day and the scores obtained during the hangover state [t(6)= -3.06, p= .022)]. The participants had greater PACS scores during the hangover state (M= 4.00; SD= 2.82) than during the normal day (M= 1.71; SD= 1.70).

Results of the Pearson correlation indicated that there was a significant positive association between the PACS score obtained on the hangover state and the AHSS score, [r(7)= .97, p < .001], suggesting that the greater the PACS score on the hangover state the greater the AHSS score. However, there was no association between the PACS score obtained on the hangover state and the number of drinks consumed during the BD night before the assessment [r(7)= -.04, p= .936].

Behavioural results

Behavioural data are summarized in Table 2. There were no significant differences between the two moments (normal day and hangover state) for any of the behavioural variables analyzed (i.e., reaction time, percentage of correct responses and percentage of correct inhibitions).

Table 2

Behavioural data at the normal day and hangover state (mean±SD)

Behavioural performance	Normal day	Hangover state
Response time (ms)	511.45±52.40	488.50±47.21
% Correct response	90.03±7.55	88.10±12.26
% Correct inhibitions	87.20±10.38	88.33±12.24

ERP results

The grand averages of ERPs for each moment in both conditions are shown in Figure 1 and Figure 2.

Latencies

Analysis of the latencies did not show significant main effects or interactions for any of the ERP components studied.

Amplitudes

P2 amplitudes. Analysis of P2 amplitude showed significant effects for the Moment factor [$F(1)= 9.56$, $p= .031$], with larger amplitudes during the normal day than during the hangover state, and also for the Electrode factor [$F(1,90)= 5.71$, $p= .020$], with the largest amplitude at FCz (M= 5.22; SD= 1.10).

N2 Amplitudes. Analysis of N2 amplitude revealed a significant effect for the Moment factor [$F(1)= 9.29$, $p= .016$], with larger amplitudes during the hangover state than the normal day, and a significant Condition x Electrode interaction [$F(2,12)= 6.82$, $p= .009$]. Post hoc Bonferroni test were used to clarify this tendency; however no significant differences in the amplitude were observed.

P3 amplitudes. There were no significant main effects or interactions for the P3 component.

LPC amplitudes. Analysis of LPC amplitude only showed a significant effect for the Electrode factor [$F(5)= 4.95$, $p= .022$], with the largest amplitude at FCz (M= 6.79; SD= 1.63).

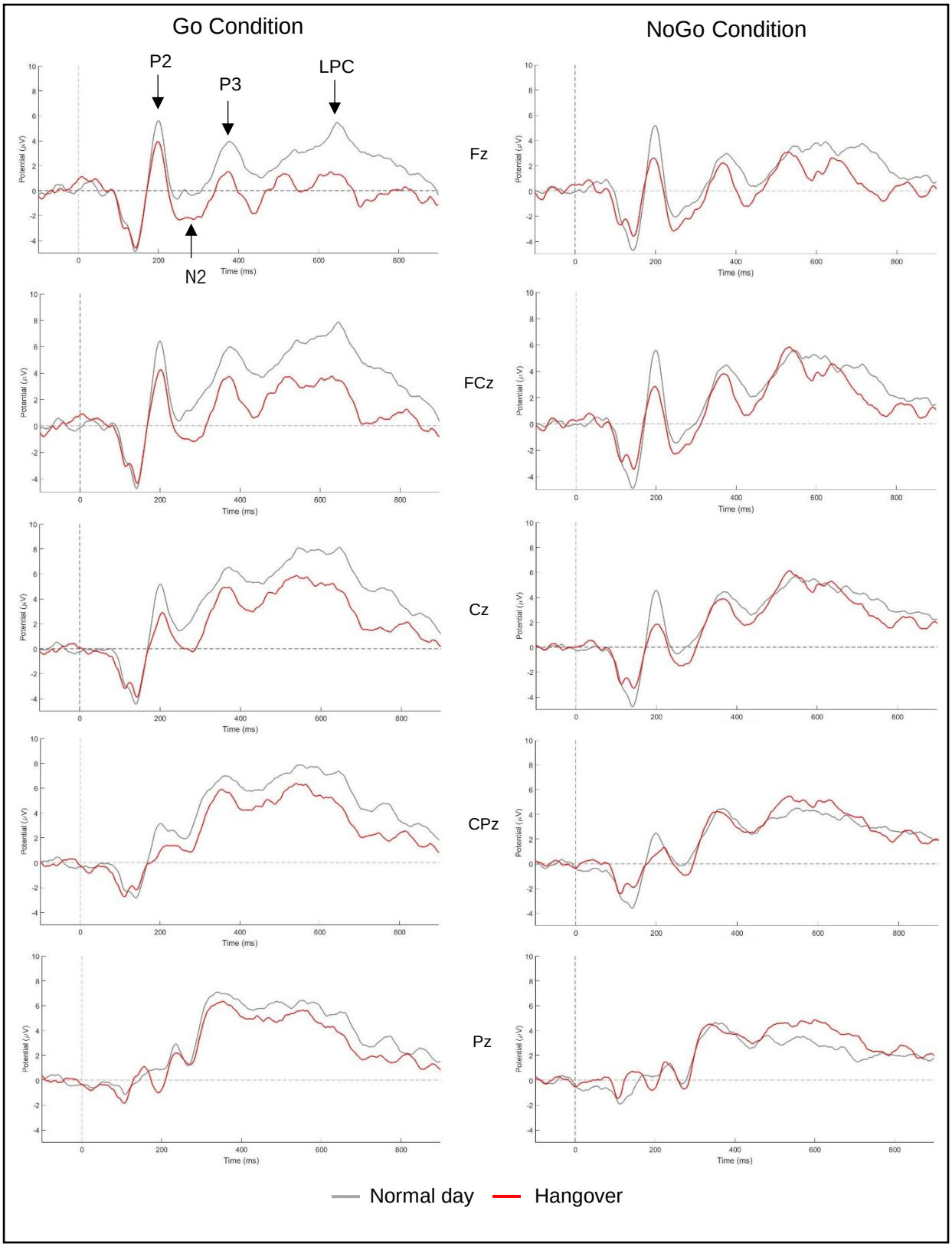

Figure 1. Grand averages of event-related potentials on normal day (grey line) and during hangover state (red line) for each condition (Go and NoGo). Averages are presented for Fz, FCz, Cz, CPz e Pz electrodes.

Discussion

To the best of our knowledge, this is the first study to explore the potential neurofunctional impairments linked to inhibitory control in college students the day after a BD session. In the present study, the participants performed a Go/NoGo task twice, once on a normal day without alcohol consumption and again the day after a typical BD episode, while the brain activity was recorded by EEG. It was expected that during the hangover state, the participants would perform worse on the task, exhibiting longer reaction times and lower percentages of correct response and/or correct inhibitions. Regarding the brain activity, we expected that on the day after a BD episode, it would be observed lower amplitudes and higher latencies in the components under study (P2, N2, P3, and LPC), particularly during response inhibition.

Results showed that there were no significant differences between the two moments of assessment for any of the behavioural variables analysed (i.e., reaction time, percentage of correct responses and percentage of correct inhibitions). Contrary to what was hypothesized, these results suggest that the participants' performance was not affected during the hangover state. However, although it is not statistically relevant, the reaction times were shorter during the hangover state than during the normal day. These results are in line with those found in studies assessing the cognitive performance of BDs in which they were faster than subjects without this pattern of consumption (Scaife & Duka, 2009; Townshend & Duka, 2005). The lower reaction times in BDs were associated with a greater motor impulsivity (Scaife & Duka, 2009). Accordingly, in the present study the reduced reaction times during hangover may suggest that after a typical BD episode the participants became more impulsive, leading to more spontaneous and fast choices (Scaife & Duka, 2009). However, these results are not congruent with those that were found in some studies which assessed the hangover effects on cognitive performance. In such studies, longer reaction times were found during the hangover than during the session after no alcohol consumption (Howland et al., 2010; Mckinney & Coyle, 2004; Mckinney et al, 2012). Consequently, further research assessing the hangover effects on the behavioural performance linked to inhibitory control is necessary to clarify these mixed results.

At the neurofunctional level, we observed significant differences between the two moments of assessment in the amplitude of P2 and N2 components. With regard to the P2 component, as we expected, BDs exhibited lower amplitudes during the day after a BD episode in comparison with the day without alcohol consumption. The P2 component has been commonly associated with attentional resources

allocation processes (Hillyard & Mangun, 1986; Sugimoto & Katayama, 2013). Specifically, previous research suggests that enhanced attention results in increased P2 amplitudes (Luck & Hillyard, 1994; Banz & Davalos, 2017). Consequently, the decreased amplitudes during hangover may reflect that young BDs have less attentional resources under the residual alcohol effects. Accordingly, several studies have associated the P2 amplitude attenuation with the effects of alcohol, namely with their acute effects and excessive alcohol use (Jääskeläinen, Näätanen, & Sillanaukee, 1996; Koskinen et al, 2011). The study of Koskinen et al. (2011) revealed a negative association between the self-reported alcohol use and the P2 amplitude, suggesting that a greater alcohol consumption leads to decreased P2 amplitudes. The P2 amplitudes also seems to decrease with alcohol intake. In this sense, a recent study by Hernández et al., (2014) found that the administration of a moderate alcohol amount leads to a reduction in P2 amplitudes, suggesting that alcohol contributes to lower levels of alertness and attentiveness (Hernández, García-Martínez, & Monteón, 2014). Additionally, studies exploring the effects of hangover on neurocognitive performance have found impairments on the attentional processes (Howland et al., 2010; McKinney & Coyle, 2004; McKinney et al., 2012; Roehrs et al., 1991). Altogether, the present results seem to point to an attenuation in the P2 amplitude as a result of the day after a heavy drinking episode, reflecting a decrement in the attentional resources that are allocated during task performance.

Concerning the N2 component, contrary to our predictions, the amplitudes were higher during the hangover state than during the normal day. The N2 component in a Go/NoGo task has been linked to conflict monitoring processes (Donkers & Boxtel, 2004). More specifically, it seems to represent the conflict that occurs when there is a competition between the execution and the inhibition of a response (Nieuwenhuis, Yeung, Van den Wildenberg, & Ridderinkhof, 2003). Therefore, the degree of conflict seems to modulate the N2 amplitude, being the enlarged amplitudes associated with a greater conflict (Folstein & van Petten, 2008). Bearing this in mind, the increased N2 amplitudes during the day after a BD episode represents a greater conflict related to response selection, i.e., whether the stimulus requires a response or not. This suggests that the residual effects of a BD episode may impair the conflict monitoring mechanisms. Supporting our results, Stock et al. (2016) also observed greater N2 amplitudes in the hangover condition than in the sober condition during a moving dots paradigm. According to the authors, the enlarged N2 amplitudes during the hangover state might reflect a greater conflict in selecting a response (Stock et al., 2016).

Despite an apparent difference between the two moments in P3 and LPC amplitudes in the grand

averages (Figure 1 and Figure 2), these differences were not statistically significant. This absence of significant differences may be due to the small sample size of the present study. Consequently, further research is warranted to clarify these trends in the P3 and LPC amplitudes.

Regarding the scores obtained in the questionnaires used before the EEG assessments, we observed that the craving for alcohol reported by participants was greater during the hangover state than during the normal day. Furthermore, the results revealed a positive association between the PACS score obtained in the hangover state and the AHSS score. This suggests that the greater the severity of hangover symptoms experienced by the participants, the greater their craving for alcohol. According to the literature, hangover may lead to an increase or a decrease in the future alcohol consumption (Piasecki, Robertson, & Epler, 2010). On the one hand, hangover can be seen as a negative consequence of alcohol intake that may lead to avoiding or stopping future alcohol use. On the other hand, the unpleasant hangover symptoms may lead to alcohol consumption to relieve their discomfort (Piasecki et al., 2010). Our findings seem to be in accordance with the second standpoint, since the participants reported to have greater craving or desire to drink alcohol when they experienced more severe hangover symptoms.

Despite the innovation of this study, it is important to note that there are some limitations which need to be taken into consideration when interpreting the results. Firstly, the reduced size of the sample may compromise the reliability of the results and it could also explain the absence of relevant hangover effects at a behavioural and functional levels. Furthermore, there were more female (n=5) than male (n=2) participants, which made it difficult to explore the gender differences. Likewise, albeit the difference was minimal, the number of participants that began to be assessed during the normal day (n=4) was not the same as the one of those who began to be assessed during the hangover state (n=3), thereby the training effect might be present. Another limitation of this study is the variation, among the participants, of the time between the moment in which they woke up and the moment of the EEG assessment during the hangover state. While some subjects were assessed shortly after they had woken up, others were assessed after having done other activities (e.g., having lunch and/or having classes) or in the evening. This may explain the variability of the hangover severity reported by the participants, inferred by the AHSS scores. Thus, in future studies, it would be advisable to maintain the time constant between the moment in which the participants stop drinking and the assessment moment in order to have a similar level of hangover for all the participants at the assessment moments. According to the existing literature, hangover usually begins within six to eight hours after the alcohol consumption (Prat et al., 2009). Thus, participants would ideally

be assessed about eight hours after they had stopped drinking.

Therefore, with an increase in the size of the sample, the individual differences as well as the training effect would be attenuated and significant differences could appear. Consequently, the results would be more reliable and more easily generalized to the population under study. Additionally, further research exploring the electrophysiological consequences of alcohol hangover is necessary to support or refute the results found in the present study. Apart from the aspects mentioned above, in future studies it would be interesting to have a mobile app specifically designed to record the daily alcohol consumption. This would aid in confirm whether they have at least one BD episode per month and would allow exploring the behavioural and functional consequences associated with their pattern of consumption. Recent research has compared the retrospective measures with real-time measures. The results revealed that when the information is retrospectively recorded the daily alcohol consumption of the subjects appear to be underestimated (Dulin, Alvarado, Fitterling, & Gonzalez, 2017; Monk, Heim, Qureshi, & Price, 2015). A study comparing the recording of daily drinking by retrospective measures with by a mobile app replicated these previous findings (Poulton, Pana, Bruns, Sinnottb, & Hestera, 2017). Accordingly, the use of a mobile app, once it is quick and easy to use, makes it easier to record the number of drinks ingested, providing more detailed information about the alcohol consumption habits (Poulton, et al., 2017).

In conclusion, the present study is the first to assess the brain activity of college students before and after a typical BD episode. Despite having found no hangover effects at a behavioural level, electrophysiological abnormalities emerged the day after a heavy alcohol drinking episode. Specifically, reduced P2 amplitudes were observed during the hangover state in comparison with a normal day without alcohol consumption, suggesting that a single episode of BD may significantly compromise the allocation of attentional resources needed to perform the task in the following day. Furthermore, enlarged N2 amplitudes appeared the day after a BD session, suggesting that the processes involved in conflict monitoring processes may be compromise. In other words, in addition to the reduced attentional capacity, college students under a hangover state seem to display difficulties in deciding whether a response need to be inhibited or not. However, due to the limitations exposed above, the present findings must be carefully interpreted and further studies are warranted to confirm or refute these results.

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