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Stress and binge drinking: A toxic combination for the teenage brain

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ABSTRACT

Young adult university students frequently binge on alcohol and have high stress levels. Based on findings in rodents, we predicted that heavy current alcohol use and elevated stress and depression scores would be associated with deficits on high interference memory tasks, while early onset, prolonged binge patterns would lead to broader cognitive deficits on tests of associative encoding and executive functions. We developed the Concentration Memory Task, a novel computerized version of the Concentration card game with a high degree of interference. We found that young adults with elevated stress, depression, and alcohol consumption scores were impaired in the Concentration Memory Task. We also analyzed data from a previous study, and found that higher alcohol consumption scores were associated with impaired performance on another high interference memory task, based on Kirwan and Stark's Mnemonic Similarity Test. On the other hand, adolescent onset of binge drinking predicted poorer performance on broader range of memory tests, including a more systematic test of spatial recognition memory, and an associative learning task. Our results are broadly consistent with findings in rodents that acute alcohol and stress exposure suppress neurogenesis in the adult hippocampus, which in turn impairs performance in high interference memory tasks, while adolescent onset binge drinking causes more extensive brain damage and cognitive deficits.

1. Introduction

The vast majority of psychological studies are conducted on university undergraduates (Henrich et al., 2010). While this limits the generality of such findings (Henrich et al., 2010), in other respects undergraduates are assumed, by many, to be an ideal participant pool: a homogeneous group of high-functioning, and physically and mentally healthy young adults. However, these assumptions may be called into question, considering the high levels of binge drinking, chronic stress and depression in the undergraduate population. In our own studies involving hundreds of undergraduates over the past 10 years, we find that 25–30% score in the mild to severe range on the Beck Depression Inventory II, and engage in regular binge drinking (where a binge is defined by the National Institute on Alcohol Abuse and Alcoholism to be 4 drinks per 2 h for a female and 5 drinks per 2 h for a male); similar binge levels have been reported in the literature for this population (e.g. Wechsler et al., 1995). Worldwide, rates of binge drinking and dangerous alcohol consumption behaviour are on the rise in adolescents (Naimi et al., 2003; WHO, 2007, 2011). Given the ongoing brain development that occurs in adolescence to early adulthood (Giedd et al., 1999), it is important to establish the long-term consequences of exposure to binge drinking and stress during this period.

While the neurotoxic effects of chronic, long-term stress, depression and alcohol on the human brain are well established, acute effects have been less studied. Multiple episodes of major depressive disorder and prolonged alcohol abuse both lead to hippocampal/medial temporal lobe volume loss (e.g. Agartz et al., 1999; Videbech and Ravnkilde, 2004; Campbell et al., 2004); long-term alcohol exposure also affects other brain regions including the prefrontal cortex and fronto-striatal reward circuits (Durazzo et al., 2011; Taki et al., 2006). The effects of prolonged heavy drinking are even more pronounced in the adolescent brain (Squeglia et al., 2015). Although less is known about the acute effects on the human brain, there is evidence that periodic binge drinking in adolescence may also cause brain volume loss (Wilson et al., 2015).

In animal models, the acute effects of stress and alcohol exposure have been studied more extensively. In adult rodents, several days of binge alcohol or stress exposure reduces hippocampal neurogenesis (Nixon and Crews, 2002; McEwen, 2001). Adolescent animals are especially vulnerable to the effects of binge alcohol exposure on the inhibition of neurogenesis (Crews et al., 2006); they also exhibit more widespread brain damage than adult-exposed animals, in regions including the temporal and frontal lobes (Crews et al., 2007; Vetreno et al., 2015). Based on these findings, we would expect to see parallel effects of acute stress and binge drinking in the human adolescent brain.

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Unfortunately, we lack a means of assaying neurogenesis non-invasively in humans. In rodents, the effects of neurogenesis knockdown versus broader hippocampal pathology can be distinguished behaviourally. Knockdown of neurogenesis results in selective impairments on a wide range of high interference memory tasks, whether the interference arises from overlapping stimuli, time delay between learning and retrieval, context effects, or reversal of previously learned responses (Becker, 2005; Luu et al., 2012; Winocur et al., 2006; Kalm et al., 2013; Vukovic et al., 2013; Garthe et al., 2009; Creer et al., 2010; Clelland et al., 2009; Wojtowicz et al., 2008; Shors et al., 2001; Drew et al., 2010; Winocur et al., 2012). In contrast, broader hippocampal pathology leads to more general associative encoding deficits (Winocur and Moscovitch, 1990; Winocur, 1990; Brasted et al., 2003; Langston et al., 2010; Honey et al., 1998; Chinnakkaruppan et al., 2014).

Consistent with findings from rodent studies, in humans with no current or previous psychiatric diagnosis, elevated depression and stress scores are associated with selective impairments on high interference memory tasks including the CANTAB delayed match to sample at long delays (Becker et al., 2009), Kirwan and Stark's (2007) Mnemonic Similarity Task and variants (MST, formerly called the Behavioural Pattern Separation Task) (Déry et al., 2013; Shelton and Kirwan, 2013; Déry et al., 2015), and recognition memory across a 2-week delay (Déry et al., 2015). Conversely, exercise is an established up-regulator of neurogenesis in animal models (Praag et al., 1999) and of neurogenesis biomarkers in rodents and humans (Pereira et al., 2007); exercise causes improved human performance on an MST-like task (Déry et al., 2013). Thus, data from humans and animal models consistently point to a selective role for hippocampal neurogenesis in mitigating memory interference. In contrast, as in rodents, in humans hippocampal damage causes more generalized episodic and associative memory deficits (Mayes et al., 2004; King et al., 2002; Manns et al., 2003; Vargha-Khadem et al., 1997). Based on the above findings, in the two experiments reported here, we sought to investigate the relationship between binge alcohol patterns, stress, depression and memory performance in university students. We hypothesized that high current binge drinking and elevated stress and depression levels would be associated with selective deficits on high interference memory tasks, while early onset binge drinking would cause broader deficits in memory and executive functions.

2. Experiment 1

We administered a battery of cognitive tests, stress and depression inventories and a lifestyle questionnaire to healthy undergraduate participants. The lifestyle questionnaire included questions about recent and remote drinking patterns. The cognitive battery included a paired associate learning task, a visual reverse digit span test, and a novel high interference test of spatial memory, the Concentration Memory Task. We also analyzed data from a previous study, parts of which had been published (Déry et al., 2013), to assess the effects of recent binge drinking on another high interference memory test.

2.1. Materials and methods

Participants were brought into a quiet testing room and seated at a desk in front of a touchscreen computer. After reading the letter of information and providing written consent, they completed computerized versions of the Beck Depression Inventory (BDI), Cohen's Perceived Stress Scale (PSS), and a lifestyle questionnaire developed by our lab. Next, they performed the three memory tests detailed below: a CANTAB-like paired associates learning task, a reverse digit span task, and the Concentration Memory Task.

2.1.1. Participants

84 McMaster University students were recruited online (at "www.experimetrix.com/mac" and "http://mcmaster.sona-systems.com"). Of these, 76 completed our experiment and met inclusion criteria: normal or corrected to normal vision and no history or previous diagnosis of major depression or other psychiatric disorders. Participants' ages ranged from 17 to 26 (mean 18.5). Participants received course credit in an Introductory Psychology course for their participation. The McMaster Research Ethics Board (MREB) approved all aspects of our study.

2.1.2. Questionnaires

To assess stress, depression, and alcohol consumption levels, we administered Cohen's Perceived Stress Scale (PSS) (http://www.psy.cmu.edu/~scohen/scales.html), the Beck Depression Inventory-II (BDI) (Psychological Corporation) and our own lifestyle questionnaire. The PSS is a freely available, 10-item questionnaire that asks participants to rate various measures of their perceived stress level within the past month, on a 5-point scale. The BDI is a standardized, commercially available neuropsychological test including 21 multiple choice questions, each on a 4-point scale, about the individual's mood during the past week. We have used our lifestyle questionnaire in several previous studies; it probes a number of different variables. The key measures of alcohol consumption included in the analyses reported here were number of drinks consumed on a typical drinking occasion (typical alcohol consumption) and a series of questions probing frequency of binge drinking at ages 13–22. A binge is defined by the United States National Institute on Alcohol Abuse and Alcoholism to be 4 drinks per 2 h for a female and 5 drinks per 2 h for a male.

2.1.3. Paired associate learning test

The paired associates learning (PAL) task was designed to be similar to the PAL task in the Cambridge Automated Neuropsychological Test Battery (CANTAB), and was implemented in e-prime. The CANTAB PAL is a widely used visuo-spatial associative learning task that was predicted to be sensitive to major hippocampal pathology. However, as it lacks a high-interference component, it was not hypothesized to be sensitive to acute levels of binge drinking, stress or depression. Indeed, we have shown previously that performance on this task does not vary as a function of BDI score (Becker et al., 2009; Déry et al., 2013). During each study trial, participants viewed six to eight white boxes displayed in a circle on the screen. The boxes were opened one at a time, in a random order, to reveal concealed patterns. Each box stayed open for 4 s. Once all of the white boxes were opened, after a 2 s delay, a test trial began. In a test trial, while the white boxes remained in a circle around the screen, the studied patterns were displayed in the middle of the screen, one at a time for five seconds, during which the participant had to touch the box where the same pattern was located. There were six levels of difficulty in which '1, 2, 3, 4, 5, 6, and 8' patterns were presented. For levels one through five there were six boxes, while for the final level there were eight boxes. Participants had up to six trials at each stage before moving on to the next, and automatically moved from one stage to the next by correctly locating all of the patterns. When an error was made, the participant was allowed to finish the test trial before all of the patterns were presented again as a reminder of their locations. The total number of errors across trials at each difficulty level was recorded.

2.1.4. Reverse digit span task

To assess working memory, we administered a computerized visual reverse digit span task implemented in e-prime. Participants