Relationship Between Prior Brain Injury and Alcohol Consumption in College Students

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Abstract

This study examined whether the occurrence of a prior brain injury was related to current patterns of alcohol consumption, and whether the existence of a prior brain injury exacerbated the effects of alcohol on cognition. Participants were college students ages 18-22 (N=50; 52% female; 80% Caucasian). Prior brain injury was identified via the HELPS screening tool (Picard, Scarisbrick, & Paluck, 1991). Binge drinkers were identified with the Timeline Follow-Back (TLFB; Sobell *et al*., 1992). Participants were administered a neuropsychological battery using the Cogstate™ (CogState Ltd.) and JAva NEuropsychological Tests (JANET) (Glahn *et al*., 2007). The Cogstate™ consisted of: the One Back, the Two Back, the Groton Maze Learning Test, and the Continuous Paired Associate Learning Task. The JANET included the following tests: the Digit Symbol task, the Balloon Analogue Risk Task (BART), and the Penn Conditional Exclusion Test (PCET). Cognitive measures were categorized a priori as those requiring complex attention/speed of processing (One Back, Two Back, Digit symbol, PCET), those requiring learning and memory (Groton Maze Learning Test, Continuous Paired Associate Learning Task), and those requiring impulse control (BART). Two by two by three analysis of variance (ANOVA) (brain injury, not brain injury) x (binge, no binge) x cognitive domain revealed that: (a) students who reported brain injury (N=20) performed significantly worse than those who reported no brain injury on measures of attention and impulse control; (b) students who binged (N=18) performed worse in all cognitive domains compared to those who did not binge, and (c) binge drinking students with a previous brain injury showed a greater decrement on tests of attention than those who only binge drank and those without brain injury. A similar pattern was found for impulse control. No such interaction was found for learning and memory. This data suggests that individuals with a history of brain injury may be at particular risk for increased cognitive impairment if they also binge. The synergistic effect of brain injury and binge drinking on cognitive function has received relatively little attention in college age samples (Graham and Cardon, 2008). The relationship among prior brain injury, impulsivity, and binge drinking are worthy of further investigation, along with documentation of the natural history of their temporal ordering.

Introduction

- The consumption of greater than five drinks on any one occasions leads to an odds ratio of 3.4:1 of sustaining an injury whereas a BAC at or above 150mg/dl leads to a higher risk of sustaining a head injury compared to other types of trauma. (McLeod *et al*., 1999; Savola *et al*., 2005)
- There is conflicting data regarding patient intoxication at time of trauma. Kraus *et al*. (1989) found that injury severity and mortality are inversely related to intoxication; Ruff *et al*. (1983) found that there was no apparent relationship between intoxication and outcome of trauma. On the other hand, it was also found that cognitive deficits seen in alcoholics cannot be accounted for only by alcohol abuse; TBI interacts with alcohol abuse to cause further neurological impairment. It is also postulated that even normal alcohol use can impact recovery from a TBI (Corrigan *et al*., 1995).
- Studies investigating the incidence of intoxication at time of trauma and outcome of the trauma have various methods of determining level of drunkenness, severity of trauma, and outcome. Thus, not all studies can be directly compared to each other (Corrigan 1995).
- When comparing alcoholics without head injuries to controls, uninjured alcoholics performed significantly worse on a cognitive battery of tests compared to the controls. Furthermore, it is important to note that a significant number of traumatic brain injuries (TBI) in alcoholics remain unidentified (Hilbom *et al*., 1986).
- Intoxication at time of a trauma and the presence of past and/or present alcohol use disorders are highly correlated (Sparadeo *et al*., 1989).
- Individuals that have shown cognitive deterioration six months post injury are more likely to have a history of substance abuse (Corrigan 1995). However, post injury drinking has a complicated effect on various aspects of recovery and can decrease the likelihood of optimum rehabilitation outcomes (Dikmen *et al*., 1995).
- It is hypothesized that the protective effect of alcohol on TBI is derived from its affect on the brain before and during the injury. From the animal model, it has been shown that the consumption of alcohol after the injury does not have the same protective effect on the brain and can be harmful during rehabilitation (Baratz *et al*., 2010).

Methods

Participants

Participants were originally recruited to the study when they were members of the freshman class at two educational institutions, a small liberal arts college and a large university. Recruiting was accomplished through school email, flyers, and classroom visits. The first year students were required to complete a pre-screen and were excluded from the study if they had: a history of major brain injury with loss of consciousness of more than twenty-four hours, multiple sclerosis (MS) or cerebral palsy, a seizure disorder, any concussion with loss of consciousness within the prior thirty days, a current or previous brain tumor, or a DSM-IV-TR Axis I psychotic disorders. The consent form signed by each of the participants clearly stated that researchers would contact the students in two years, when they are juniors, for a follow up time slot. Recruiting for the follow up time slot consisted of emailing participants individually, dorm room visits, flyers, and an iPAD raffle.

Materials

The JANET platform includes a measure of risk taking and impulsivity, the Balloon Analogue Risk Task, and executive functioning through the Penn Conditional Exclusion Test. The Mini Neuropsychiatric Interview is given as a brief measure of psychopathology. The COGSTATE platform includes two memory and learning tasks, the Groton Maze Learning Task and the Continuous Paired Associate Learning Task as well as n–back test, which measures executive function and attention.

Procedure

Each participant participated in a total of two time slots. Each of the two time slots asked the same questions about alcohol and drug use and contained the same cognitive tasks. During the follow up testing session, participants were asked to fill out a number of forms: a compensation form, an Autism-Spectrum Quotient, the HELPS brain injury screening tool, and a demographics form. Following this, individuals were asked to complete a battery of computerized cognitive tasks. The first test administered was the Groton Maze Learning Task, taking approximately 10 minutes to complete. Second, participants were administered the *n*-back, consisting of the one back and two back tests. Each of these tests was approximately 10 minutes in length. Next, participants completed the Continuous Paired Associate Learning Task (CPAL), about 10 minutes in length. As a final measure, participants were administered one additional trial of the Groton Maze Learning Task as a test of recall. The next set of cognitive tests was part of the JANET testing platform. Participants were instructed to follow along with the instructions and complete the tests at their own pace. First, participants were administered the Digit Symbol, then the Penn Conditional Exclusion Test (PCET), and finally, the Balloon Analogue Risk Task (BART). Each of these tests was about 10 minutes in length, for a total of 30 minutes. There was an interval of approximately 5 minutes in between the testing platforms for participants to rest. Participants were also asked to fill out a self report survey regarding their alcohol and drug use throughout their lifetime. After these computer-based assessments, each individual participated in a Mini Neuropsychiatric Interview, in order to diagnose certain disorders such as depression, suicidality, mania, alcohol abuse or dependence, substance abuse or dependence, and assorted others.

Results

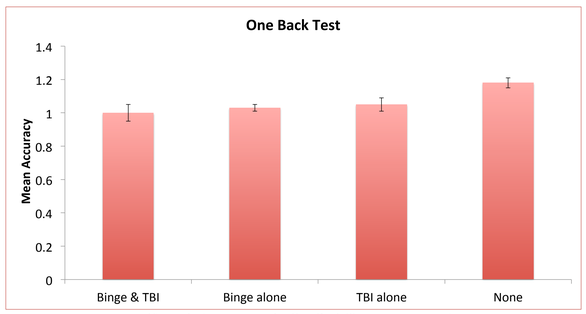

Figure 1: Accuracy on the One Back Test across categories

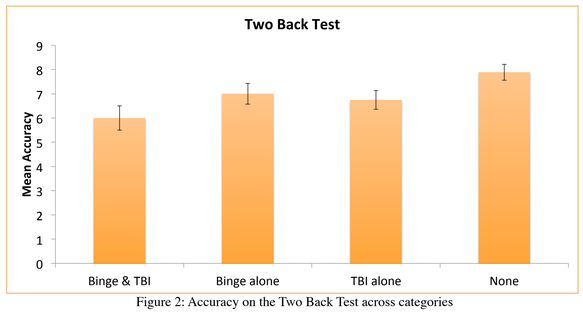

Figure 2: Accuracy on the Two Back Test across categories

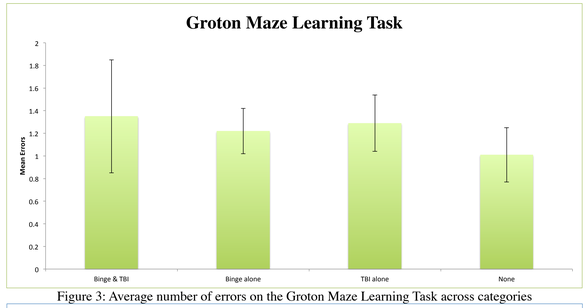

Figure 3: Average number of errors on the Groton Maze Learning Task across categories

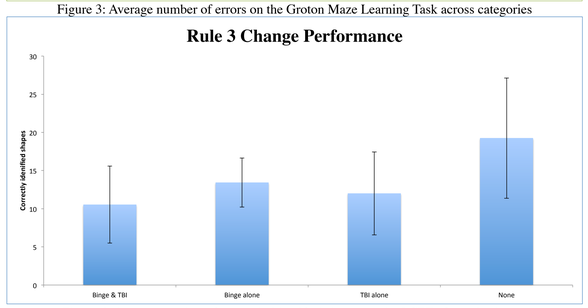

Figure 4: Average number of correctly identified shapes on the PCET after rule change

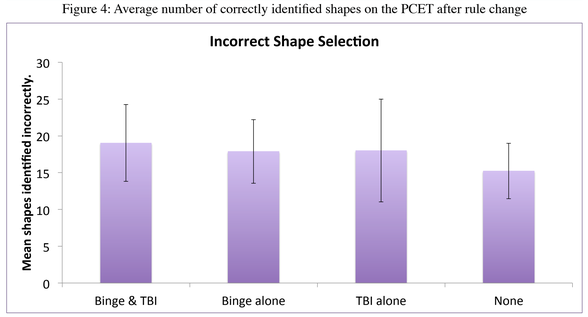

Figure 5: Average number of incorrectly identified shapes on the PCET

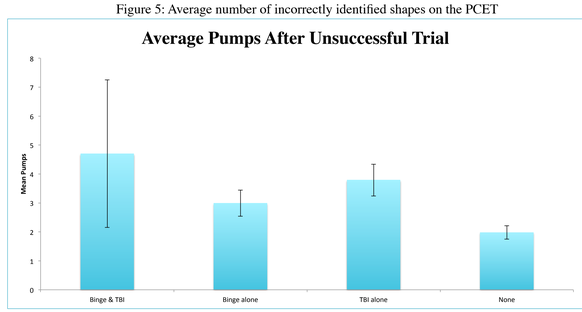

Figure 6: Average number of pumps on the BART after an unsuccessful trial

Discussion

- Students who reported brain injury performed significantly worse than those who reported no brain injury on measures of attention and impulse control.
- Students who binge performed worse in all cognitive domains such as attention, executive function, and impulsivity compared to those who do not binge.
- Binge drinking students with a previous brain injury demonstrated poorer performance on tests of attention compared to those who only binge drank and those without brain injury.
- Similarly, binge drinking students with a previous brain injury demonstrated more impulsivity on the BART measure compared to those who only binge drank and those without brain injury.
- There was not shown to be any association between learning and memory and previous brain injury.
- This data suggests that individual with a history of brain injury may be at particular risk for increased cognitive impairment if they also binge.
- The synergistic effects of brain injury and binge drinking on cognitive function has received relatively little attention in college age samples (Graham and Cardon, 2008). The relations among prior brain injury, impulsivity, and binge drinking are worthy of further investigation, along with documentation of the natural history of their temporal ordering.

References

Baratz, R., Rubovitch, V., Frenk, H., Pick, C.G. (2010). The influence of alcohol on behavioral recovery after mTBI in mice. *Journal of neurotrauma*, 27, 555-563.

Corrigan, J.D. (1995). Substance abuse as a mediating factor in outcome from traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 76, 302-309*.

Dikmen, S.S., Machamer, J.E., Donovan, D.M., Winn, H.R., Temkin, N.R. (1995) Alcohol use before and after traumatic head injury. *Annals of emergency medicine,* 26(2), 167-176.

Hillbom, M., Holm, L. (1986). Contribution of traumatic head injury to neuropsychological deficits in alcoholics. *Journal of Neurology, Neurosurgery, and psychiatry,* 49, 1348-1353.

Kraus, J.F., Morgenstern, H., Fife, D., Conroy, C., Nourjah, P. (1989). Blood alcohol test prevalence of involvement and outcomes following brain injury. *American Journal of Public Health,* 79(3), 294-299.

McLeod, R., Stockwell, T., Stevens, M. *et al*., (1999). The relationship between alcohol consumption patterns and injury. *Addiction,* 94, 1719-1734.

Ruff, R.M., Marshall., L.F., Klauber, M.R., Blunt, B.A., Grant, I., Foulkes, M.A. *et al*. (1993). Alcohol abuse and neurological outcome of the severely head injured. *The Journal of Head Trauma Rehabilitation,* 8(26), 26-33.

Savola, O., Niemela, O., Hillbom, M. (2005). Alcohol intake and the pattern of trauma in young adults and working aged people admitted after trauma. *Alcohol and Alcoholism*, 40, 269-273.

Sparadeo, F.R., Gill, D. (1989). Effects of prior alcohol use on head injury recovery. *The Journal of Head Trauma Rehabilitation,* 4, 75-82.

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