

Undergraduate Research Symposium May 17, 2013 Mary Gates Hall

Online Proceedings

POSTER SESSION 3

Commons West, Easel 30

2:30 PM to 4:00 PM

Interaction of Alcohol and Sleep Medication Use Predicts Negative Consequences

Jason Jeffery (Jason) Van Dyck, Senior, Psychology

Mentor: Haley Douglas, Psychology, Center for the Study of Health and Risk Behaviors

Mentor: Mary Larimer, Psychiatry & Behavioral Sciences

Alcohol use consistently correlates with negative physical and psychological consequences like dependence, memory loss, impaired judgment, etc. Studies also link the use of sleep medications with similar negative consequences. Yet, no study examines the interaction of sleep medication and alcohol use onto these negative consequences. Therefore the present study investigated the relationship between peak blood alcohol content (BAC) in the past 6 months, negative consequences of alcohol use in the last 12 months as measured by the Rutgers Alcohol Problem Index, and lifetime, 3- and 12-month non-prescribed sleep medication use in Washington state and Swedish high school seniors participating in a larger study (N = 3,352, 56.6% Female). Regression analyses revealed a significant main effect of BAC and lifetime use of sleep medication onto negative consequences ($\beta = 18.69$, $F(3, 2875) = 115.86$, $p < .05$) such that individuals with a higher BAC and who had a lifetime incidence of sleep medication usage experienced significantly greater negative consequences than those who only drank or used sleep medications. There was no significant moderating effect of frequency of sleep medication use in the last 3 or 12 months onto BAC and negative consequences ($p > .05$). While these results suggest that use of sleep medication and alcohol interact to contribute to increased risk for negative consequences, there are limitations. Lifetime use of sleep medication may reflect a tendency to act impulsively or self-medicate (e.g. for sleep/depression/anxiety), not a direct effect of the medication per se. Additionally, analyses did not control for other drug usage or mood disturbances, and all sleep medication drugs were grouped under one survey item. Given our observations, additional research testing combined effect of sleep medication and alcohol use as related to negative consequences is warranted.

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Impulsivity in Relation to Alcohol Use and Expectancies

Kayla Jeniece (Kayla) Evans, Senior, Sociology, Psychology

Mentor: Haley Douglas, Psychology, Center for the Study of Health and Risk Behaviors

Mentor: Mary Larimer, Psychiatry & Behavioral Sciences

Impulsivity is often found to be related to alcohol use (Pearson and Henson, 2012; Simons, 2003). Additionally, a correlate of alcohol expectancies is increased alcohol use (Hull and Bond, 1986). However, no research to date has looked directly into the association of impulsivity, alcohol expectancies, and alcohol use. Therefore, the present study looks into impulsivity, alcohol use, and alcohol expectancies in high school seniors (N = 3,352, 56.6% Female) from Washington and Sweden. Participants completed a baseline online survey as part of a larger trial that assessed peak blood alcohol content (BAC) in the past 12 months, impulsivity as measured by the sensation seeking scale (Zuckerman, 1964), and alcohol expectancies as measured by the brief Comprehensive Effects of Alcohol questionnaire which consists of positive (sociability, tension-reduction, liquid courage, and sexuality) and negative (cognitive/behavioral impairment, risk/aggression, and self-perception) classifications (CEOA; Fromme, 1993). Regression analyses revealed a significant main effect of impulsivity, cognitive/behavioral impairment, sociability, risk/aggression, and self-perception onto peak BAC ($p < .05$). However, significant interactions were only found for impulsivity onto risk/aggression and self-perception. A significant moderating effect of risk/aggression onto the relationship between impulsivity and peak BAC ($\beta = .002$, $F(3, 2913) = 114.71$, $p < .05$) was found such that those with lower impulsivity and lower risk aggression scores (alcohol seen positively) have higher peak BAC. A significant moderating effect of self-perception onto the relationship between impulsivity and peak BAC was also found ($\beta = .001$, $F(3, 2908) = 132.04$, $p < .05$) suggesting that individuals with lower self-perception scores (alcohol seen positively) and higher impulsivity have higher peak BAC. These results suggest that individuals who are both highly impulsive and have higher positive expectancies for alcohol drink more. Future research should continue to investigate the relationship between alcohol expectancies and impulsivity in vivo.

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O6-Methylguanine-DNA Methyltransferase Activity

Alaina Julia (Alaina) Goesling, Senior, Biology (General)

Mentor: John Silber, Neurological Surgery

Mentor: Douglas Kolstoe, Neurological Surgery

The project I worked on was part of a larger project in which we examined the activity of O6-methylguanine-DNA methyltransferase activity, CpG promoter methylation, and progression-free survival following alkylating agent therapy in glioblastoma and anaplastic glioma brain tumors. For my part in the research I performed standard biochemical assays to quantify O6-methylguanine-DNA methyltransferase (MGMT) in extracts of glioblastoma (GBM) and anaplastic glioma (AG) tumor samples from adult patients operated on with consent at the University of Washington Medical Center between 1991 and 2008. MGMT is a DNA repair protein that excises methyl adducts from the O6 position of guanine residues. Chemotherapy and other alkylating agents such as methylating triazene derivative temozolomide (TMZ) are responsible for methylating DNA, or damaging the DNA and thus destroy the tumor cell. There has been poor prognosis for GBM due to the intrinsic resistance mechanisms in the MGMT that limit the efficacy of TMZ and other alkylators. GBM and other gliomas display a wide range of MGMT activity and thus the question for research was whether the amount of MGMT in tumors correlated with progression-free survival following alkylator-based chemotherapy. Overall the data strongly supported that MGMT activity in GBM and AG tissue mediates resistance to alkylator therapy.