

UNDERGRADUATE STUDENTS AND RISK FACTORS FOR EXCESSIVE DRINKING

by

Sylvia Anna Nay

A thesis submitted to the Department of Psychology

In conformity with the requirements for

the degree of Doctor of Philosophy

Queen's University

Kingston, Ontario, Canada

(October, 2015)

Copyright © Sylvia Anna Nay, 2015

Abstract

Excessive drinking is associated with significant negative consequences for individuals, and enormous costs to the healthcare system and economy. Around the world, university students comprise a particularly vulnerable population with respect to excessive drinking. This demographic group exhibits heavier drinking patterns than older adults and like-aged peers who do not attend university. Given the widespread negative fallout associated with alcohol misuse, the identification of pre-existing factors that are associated with increased vulnerability for this behaviour is critically important, especially among high-risk groups such as undergraduates. The goal of the current dissertation was to explore, among undergraduates, individual differences in intrapersonal factors that confer risk for excessive drinking, particularly disinhibited behaviour and stress – factors that have been robustly associated with the development of alcohol use disorder. Study 1 examined whether impulsivity, sensation seeking, and anxiety sensitivity were related to escalated drinking behaviour in the transition between high school and the first year of undergraduate studies. Our findings demonstrated that individual differences in trait impulsivity prospectively predicted increases in hazardous drinking and, at a trend level, drinking intensity. Study 2 assessed whether exposure to acute stress increases voluntary alcohol intake and, further, whether intake of alcohol relates to individual differences in anxiety. Consistent with our hypotheses, psychosocial stress increased voluntary intake of alcohol, but not placebo or non-alcoholic beverages. In contrast, *ad libitum* alcohol intake was not related to individual differences in anxiety. Finally, Study 3 examined whether alcohol induces biochemical stress responses in a non-clinical population and, moreover, whether this effect relates to individual differences in impulsivity and subjective alcohol responses. Our data demonstrated that alcohol intoxication was associated with increases in biochemical markers of stress, namely cortisol and alpha amylase, among males. Biochemical responses were highest among individuals who reported the greatest stimulant effects of alcohol. Additionally, trait impulsivity was positively correlated with cortisol responses to alcohol. Taken together, the current studies underscore the importance of individual differences in behavioural

disinhibition (e.g., impulsivity) on the subjective effects of alcohol and drinking behaviour among undergraduates. Clinical implications and avenues for future research are discussed.

Co-Authorship

The manuscripts included in this dissertation are the result of collaboration between the doctoral candidate, Sylvia Nay, and her supervisor, Dr. Mary C. Olmstead. As the primary author, Mrs. Nay assumed primary responsibility for research design, data collection, data analysis, and dissertation writing. Dr. Olmstead assisted with all aspects of the studies, particularly manuscript writing. The first study (Chapter 2) was co-authored by Dr. Olmstead. The second study (Chapter 3) was also co-authored by Dr. Olmstead, and has been published in the Oxford University Press journal *Alcohol & Alcoholism* (Magrys & Olmstead, 2015). The third study (Chapter 4) has been published in the Wiley Publishing journal *Psychophysiology* (Magrys, Wynne-Edwards, Olmstead, & Balodis, 2013). This study is based on archival data collected by Dr. Iris Balodis, who also assisted with data analysis and writing of the manuscript, and is listed as a co-author. Dr. Katherine Wynne-Edwards is also a co-author on the third study, as she conducted the neuroendocrinological analysis.

Acknowledgements

First, I would like to express my sincere gratitude to my supervisor and mentor, Dr. Cella Olmstead. I appreciate everything she has taught me about research, life, and how to balance the two. Thanks to my labmates Megan, Amanda and Scott, for their research support and friendship. I gratefully acknowledge the undergraduate students who assisted with data collection. Overall, I feel fortunate to have been part of an academic ‘home’ as wonderful as the Olmstead lab.

Thank you to my dissertation committee, Dr. Ron Holden and Dr. Chris Bowie, for their guidance and encouragement throughout this project. The work and generous advice of my research collaborator, Dr. Iris Balodis, is much appreciated. Many thanks to Daniela, editor extraordinaire, and to Jolene and Jen, the other two thirds of the Online Dissertation Support Group. Thanks to all the faculty members who, as inspiring teachers, clinical supervisors, and colleagues, have made an impact on my development.

I owe the largest debt of gratitude to my loved ones. Thank you to my dear friends for the many good meals, long conversations, and belly laughs, which were welcome distractions on the long road through grad school. I am immeasurably thankful for my family. Mom, dad, Phillip – thank you for your love, for teaching me the importance of hard work and integrity, and for always believing in me. I sincerely appreciate the continuous support and encouragement of my second family, the Nays. Finally, to my husband, Curtis – it is impossible to put into words how grateful I am to have you by my side. Your thoughtfulness, sincerity, humour, curiosity, fearlessness, and positivity make everything better. Thank you for sharing this journey with me – I love you.

Table of Contents

Abstract.....	ii
Co-Authorship.....	iv
Acknowledgements.....	v
List of Figures.....	x
List of Tables.....	xi
List of Abbreviations.....	xii
Chapter 1 General Introduction.....	1
Defining Alcohol Misuse: Alcoholism and Risky Drinking.....	2
Drinking Behaviour in the General Population.....	5
Drinking Behaviour among Undergraduate Students.....	5
Individual Differences in Risk for Alcoholism.....	8
Risk Factors for Alcoholism.....	10
Stress.....	10
Disinhibited Behaviour.....	12
Response to Alcohol.....	14
Purpose of Current Studies.....	16
References.....	18
Chapter 2 Trait Impulsivity Prospectively Predicts Progression to Hazardous Drinking in Undergraduate Students.....	42
Abstract.....	43
Introduction.....	44
Methods.....	46
Participants and Procedure.....	46
Substance Use Measures.....	47
Alcohol Timeline Follow-Back.....	47
Rutgers Alcohol Problem Index.....	48
Alcohol Use Disorders Identification Test.....	48
Alcohol, Smoking and Substance Involvement Screening Test.....	48
Trait and Behaviour Measures.....	49
Barratt Impulsiveness Scale.....	49
Anxiety Sensitivity Index.....	49
Zuckerman-Kuhlman Personality Questionnaire.....	49

Alcohol Use Onset.....	50
Statistical Analyses.....	50
Participant Demographics.....	50
Measure Descriptives.....	51
Relationship between Predictors and Criterion Variables.....	51
Relationship between Predictors and Alcohol Use Onset.....	51
Results.....	52
Participant Demographics.....	52
Measure Descriptives.....	52
Relationship between Predictors and Criterion Variables.....	55
Relationship between Predictors and Alcohol Use Onset.....	61
Discussion.....	61
Conclusions.....	64
References.....	66
Chapter 3 Acute Stress Increases Voluntary Consumption of Alcohol in Undergraduates.....	72
Abstract.....	73
Introduction.....	74
Methods.....	75
Participants.....	75
Self-Report Measures.....	76
State-Trait Anxiety Inventory.....	76
Perceived Intoxication.....	76
Stress Condition.....	77
Drinking Protocol.....	77
Experimental Procedure.....	78
Results.....	79
Participants.....	79
Manipulation Checks.....	79
Stress.....	79
Perceived Intoxication.....	80
Effect of Stress on Fluid Intake.....	82
Relationship between Self-Report Anxiety Measures and Alcohol Intake.....	83
Discussion.....	83
References.....	87

Chapter 4 Neuroendocrinology Responses to Alcohol Intoxication in Health Males: Relationship with Impulsivity, Drinking Behaviour and Subjective Effects	91
Abstract	92
Introduction.....	93
Methods	95
Participants.....	95
Measures.	96
Self-Report Measures.....	96
Barratt Impulsiveness Scale.	96
Biphasic Alcohol Effects Scale.....	96
Manipulation Check.....	96
Physiological Measures.....	96
Procedure.	97
Statistical Analyses.	98
Self-Report Measures.....	98
Relationship between Self-Report Measures.	99
Physiological Responses.....	99
Relationship between BAL and Physiological Responses.....	99
Relationship between Self-Report Measures and Physiological Responses.....	99
Results.....	99
Self-Report Measures.....	99
Manipulation Check.....	100
Relationship between Self-Report Measures.	101
Physiological Responses.....	101
Cortisol.....	102
Alpha-Amylase.	102
Relationship between BAL and Physiological Responses.....	103
Relationship between Self-Report Measures and Physiological Responses.....	103
Discussion	104
References.....	107
Chapter 5 General Discussion.....	112
Summary of Findings.....	112
Individual Differences in Risk Factors	113
Interaction between Risk Factors	116

Revisiting the Concept of Alcoholic Personality 118
Role of Alcohol Expectancy 120
Limitations and Future Research 123
Conclusions and Practical Implications 124
References..... 127
Appendix A..... 151

List of Figures

Figure 2-1. Sensation seeking correlated against age of recreational drinking onset (years).	61
Figure 3-1. Graphical representation of experimental timeline.	79
Figure 3-2. Effect of stress on state anxiety across experimental time points.	80
Figure 3-3. Relationship between manipulation check items and fluid intake.	82
Figure 3-4. Effect of stress and beverage group on fluid intake.	83
Figure 4-1. Experimental timeline. Diagram shows procedure over time (minutes).	98
Figure 4-2. BAES scores over time.	100
Figure 4-3. Changes in cortisol (a) and alpha-amylase (b) across four sampling points following administration of the first beverage for the 3 beverage groups.	102
Figure 4-4. Relationship between percent change in cortisol levels and scores on the BIS.	104

List of Tables

Table 1-1 Summary of risk factors and drinking behaviours examined in each study	16
Table 2-1 Descriptive statistics for predictor and criterion measures.....	53
Table 2-2. Change in criterion measures between Time 1 and Time 2.....	55
Table 2-3. Bootstrapped correlations (Pearson's r) between predictor and criterion variables ($n = 160$)..	57
Table 2-4. Hierarchical regression analysis predicting substance use behaviour	59
Table 3-1. Mean (SEM) blood alcohol level (BAL) and self-reported perceived intoxication in placebo and alcohol groups.	80

List of Abbreviations

ANOVA	Analysis of variance
AUD	Alcohol use disorder
AUDIT	Alcohol Use Disorders Identification Test
APA	American Psychiatric Association
AS	Anxiety sensitivity
ASI	Anxiety Sensitivity Index
ASSIST	Alcohol, Smoking and Substance Involvement Screening Test
BAES	Biphasic alcohol effects scale
BAL	Breath alcohol level
BIS	Barratt Impulsiveness Scale
DEQ	Drug Effects Questionnaire
HPA	Hypothalamic-pituitary-adrenal
ImpSS	Impulsive sensation seeking scale
RAPI	Rutgers Alcohol Problem Index
SAM	Sympathetic-adrenal-medullary
SS	Sensation seeking
SPSS	Statistical Package for the Social Sciences
STAI	State-Trait Anxiety Inventory
STAIS	State-Trait Anxiety Inventory – State
STAIT	State-Trait Anxiety Inventory – Trait
TLFB	Timeline Follow-back
TSST	Trier Social Stress Test

Chapter 1

General Introduction

Immense individual, societal, and economic costs are attributable to excessive drinking (Mohapatra, Patra, Popova, Duhig, & Rehm, 2010; Rehm et al., 2009) which includes clinically pathological drinking (i.e., alcoholism) as well as risky alcohol consumption by non-alcoholics. The personal consequences associated with excessive drinking include harm to relationships, missed responsibilities, and financial difficulties (Rehm et al., 1996). Alcohol misuse also contributes to injuries, and a variety of health conditions such as diabetes, cirrhosis, and cardiovascular disease (Rehm et al., 2009; World Health Organization, 2009). To add to these negative outcomes, alcoholism is highly comorbid with other psychiatric disorders, such as mood and anxiety disorders (Schuckit, 2006). Globally, excessive alcohol use is one of the top ten risk factors for disability and mortality (World Health Organization, 2009), accounting for 4.6% of disability-adjusted life-years and 3.8% of deaths worldwide (Rehm et al., 2009). Similarly, the economic costs associated with heavy alcohol use are high, averaging 1.6% of the gross domestic product annually in high-income countries, including Canada (Mohapatra et al., 2010). Taken together, the personal and societal burden associated with excessive drinking is staggering.

Given the sizeable negative consequences associated with alcohol misuse, it is critical to identify specific populations and pre-existing factors that are associated with increased risk for this behaviour. In North America and other developed countries, university students have emerged as a particularly vulnerable population with respect to problem drinking (Jackson, Sher, & Park, 2005). Examination of risk factors for excessive drinking has focused on individual differences in both the general population and among university students (Baer, 2002). These include variations in behavioural disinhibition, stress, and alcohol responses all of which are associated with increased risk for alcohol misuse (for reviews see Barr & Goldman, 2006; Quinn & Fromme, 2011; Schuckit, 2002; Sher, Grekin, & Williams, 2005).

Importantly, these risk factors can serve as the foci of psychoeducational, psychological, and behavioural interventions aimed at university students. Thus, research that elucidates the role of risk factors in excessive drinking among undergraduates will not only further theoretical knowledge, but may also provide practical benefit through clinical applications.

The following introductory chapter begins by operationalizing measures of drinking behaviour, first clarifying the distinction between alcohol use, misuse, and clinically-diagnosed abuse. Then, epidemiological overviews of alcohol use are provided as they relate to the general population and, more specifically, to undergraduate students. The latter sub-section highlights the uniqueness of undergraduate students' drinking patterns and consequent vulnerability for alcohol misuse. Theoretical context is provided through discussion of the individual difference model of alcoholism risk, and sub-ordinate hypotheses related to affect regulation and subjective alcohol responses. Grounded in this theoretical framework, prominent intrapersonal risk factors, namely behavioural disinhibition, stress, and alcohol response, are described. Finally, the original studies composing this dissertation are introduced.

Defining Alcohol Misuse: Alcoholism and Risky Drinking

'Alcoholism' is a lay term used to describe the pathological range of alcohol use, and corresponds to the clinical condition known as *alcohol use disorder* (AUD; American Psychiatric Association [APA], 2013). According to the Diagnostic and Statistical Manual of Mental Disorders – 5th Edition (DSM-V), AUD is a maladaptive pattern of drinking that causes significant impairment or distress (APA, 2013). Consistent with current conceptualizations of alcoholism, the most recent DSM definition centers on “a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems (p. 483)” and highlights the involvement of persistent brain changes resulting from repeated use of the substance (APA, 2013). AUD is typified by loss of control over drug use, chronicity, and frequent relapse (Leshner, 1997, 1999). These characteristics distinguish AUD from non-pathological substance use and make recovery from the disorder particularly challenging (Dawson, Grant, Stinson, et al., 2005; Moos & Moos, 2006).

The development of AUD is a dynamic process that begins with controlled, social use and progresses to problem drinking before reaching clinical levels (Becker, 2008). Given the persistent, treatment-refractory nature of alcoholism, there is great benefit to understanding the problem drinking behaviour that precedes AUD. Moreover, risky drinking behaviours are, in their own right, associated with negative outcomes and risk for disease, injury, and mortality (Rehm et al., 2003). Various terms are used interchangeably to describe a pattern of drinking that exceeds what is considered to be healthy or low-risk: ‘excessive’, ‘problem’, ‘problematic’, ‘hazardous’, ‘risky’, ‘immoderate’, and ‘maladaptive’. Although there is no globally-accepted definition of problem drinking, it is often described in terms of two features: patterns of alcohol intake and alcohol-related problems (Baer, 2002).

Patterns of alcohol intake are most commonly described in terms of self-reported ‘usual’ frequency (number of drinking days) and quantity (number of standard alcoholic drinks consumed per drinking day or occasion), with a standard alcoholic drink being defined as a 1.5 oz. shot of liquor, a 5 oz. glass of wine, or 12 oz. beer/ cider/ cooler. Thresholds for low vs. high frequency and light vs. heavy quantity vary considerably across research studies (Rossow & Romelsjö, 2006). For example, thresholds for heavy intake include 40 grams per day (Rehm et al., 2009; WHO, 2009), 1 oz. per day (Dawson, 1999), and 11 L per year (Skog, 1999). Alternatively, other researchers take scale measures of alcohol intake (e.g., average volume) and conceptualize heavy consumption along the upper range of this continuum (Jones et al., 1995). The Canadian Addiction Survey, a large nationwide population study, categorizes respondents’ drinking patterns as frequent or infrequent, using a threshold of five drinks per occasion; light (less than one drinking occasion per week on average) or heavy (one or more drinking occasions per week on average; Adlaf, Begin, & Sawka, 2005). Canada’s Low-Risk Alcohol Drinking Guidelines (Canadian Centre on Substance Abuse, 2012) recommend that women should have no more than 2 drinks per day or 10 drinks per week and men should not exceed 3 drinks per day or 15 drinks per week (Butt, Beirness, Stockwell, Gliksman, & Paradis, 2011). U.S. guidelines define low-risk drinking as 3 drinks per day or 7 drinks per week for women, and 4 drinks per day or 14 drinks per week for men

(National Institute on Alcohol Abuse and Alcoholism, 2010). Additionally, some scholars assign problem drinking status to any alcohol consumption by populations who, for health and safety reasons, should engage in complete abstinence, such as youth under the age of 21 and pregnant women (e.g., Bouchery, Harwood, Sacks, Simon, & Brewer, 2011).

Heavy episodic use, also known as binge drinking, is an increasingly popular measure of risky alcohol consumption (see Courtney & Polich, 2009). Binge drinking is defined as one or more binge episodes over a two-week period, wherein a binge episode is four drinks per occasion for women and five drinks per occasion for men (Thomas, 2012; Wechsler, Dowdall, Davenport, & Rimm, 1995). Some researchers apply these per-session thresholds (i.e., the '4/5 rule') to a 24-hour period to operationalize binge drinking (e.g., Collins, Kashdan, Koutsky, Morsheimer, & Vetter, 2008). Maximum intake per drinking session correlates with traditional measures of binge drinking (i.e., Wechsler et al., 1995), offering an alternative measure of heavy episodic drinking (Esser, Kanny, Brewer, & Naimi, 2012). Subjective-effects measures, such as frequency of drunkenness, have also been used to quantify the intensity of drinking occasions (O'Neill, Parra, & Sher, 2001).

Arguably, alcohol-related problems are an equally important aspect of identifying risky drinking. In fact, some researchers propose that thorough conceptualization of maladaptive drinking necessitates inclusion of both usage pattern measures and indices of alcohol-related problems (Ham & Hope, 2003). Risky alcohol use is associated with a range of negative consequences, including risk for diseases, injury, harm to others, failed responsibilities, academic/vocational problems, and financial strain. Self-report measures, such as the Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) and Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993), are commonly used to measure these alcohol-related negative consequences. Researchers and clinicians use self-report measures differentially to produce varied metrics of alcohol-related problems (Rossow & Romelsjö, 2006). This may include continuous ranges, wherein higher sum scores signify greater alcohol-related problems (Gmel, Kuntsche, & Rehm, 2011), cut-scores, which delineate hazardous drinking or

high alcohol-related problems (Aalto, Alho, Halme, & Seppä, 2009), and frequency/ number of alcohol-related problems (Wyllie et al., 2000). Alternatively, specific behaviours, such as alcohol-related absenteeism, serve as proxy measures of alcohol-related problems in general (Jones et al., 1995).

Drinking Behaviour in the General Population

Approximately 80% of Canadians over the age of 15 endorse past-year drinking (Adlaf, Begin, & Sawka, 2005; Health Canada, 2012). Most Canadians are moderate drinkers, with 38.7% classifying as *light infrequent* drinkers and 28.7% falling into the *light frequent* category, according to the Canadian Addiction Survey thresholds described above (Adlaf et al., 2005). However, *heavy infrequent* and *heavy frequent* drinkers make up 5.6% and 7.1% of the Canadian population, respectively (Adlaf et al., 2005). Thirteen percent of Canadians are considered high-risk drinkers, based on self-reported hazardous drinking behaviour, and 10% of Canadian drinkers report experiencing drinking-related personal harm related to their relationships, physical health, vocational/academic life, and/or finances, over the past year (Adlaf et al., 2005). The 12-month and lifetime prevalence of AUD in the U.S. is 8.5% and 30%, respectively (Hasin, Stinson, Ogburn, & Grant, 2007).

Notably, past-year drinking rates are highest among young adults aged 20-24 years (83%-90% report drinking over the past year), and up to 30% of this age group engages in hazardous drinking behaviour (Adlaf, Begin, & Sawka, 2005; Health Canada, 2012). In general, alcohol use follows a typical developmental trajectory: drinking typically begins in adolescence, gradually increases until young adulthood (Chassin, Forthofer, & King, 2004; White, Labouvie, & Papadaratsakis, 2005), after which individuals undergo ‘maturing out’ – a normative steady decline in alcohol use (Lee, Chassin, & Villalta, 2013).

Drinking Behaviour among Undergraduate Students

Alcohol use is particularly problematic among undergraduate students, whose drinking patterns are distinct from the general population (Ham & Hope, 2003). In keeping with typical developmental trends, drinking rates increase in the transition from high school to university (Fromme, Corbin, & Kruse,

2008), remain elevated throughout the undergraduate years, and decline following graduation (O’Neill et al., 2001). As adolescents, individuals who do not go on to attend university exhibit heavier drinking patterns than their university-bound peers, but this pattern reverses in young adulthood (Timberlake et al., 2007). Specifically, in early adulthood, undergraduate students show a faster rate of increase in alcohol use, consume alcohol more heavily, binge-drink more frequently, exhibit higher rates of alcohol abuse, and are more likely to die from impaired driving compared to like-aged peers who do not attend university (Hingson, Zha, & Weitzman, 2009; Slutske, 2005; Timberlake et al., 2007; White et al., 2005). Most notably, approximately 45% of undergraduates report engaging in risky drinking behaviour, such as bingeing, in the previous year (Hingson et al., 2009; Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994; Wechsler, Molnar, Davenport, & Baer, 1999). In a large, representative survey of U.S. university students, binge drinkers comprised just under half of the total sample, but accounted for 91% of the total alcohol consumed (Wechsler et al., 1999). An estimated 15% of undergraduate students’ meet criteria for AUD based on their past-year drinking patterns (Grekin & Sher, 2006), which exceeds general population base rates of approximately 8.5% (Hasin et al., 2007).

Additionally, undergraduate students regularly engage in unique drinking behaviour such as “pregaming” – drinking before going out to a social function (also known as “pre-partying”, “pre-drinking”, or “drinking before drinking”; Borsari et al., 2007; Zamboanga, Schwartz, Ham, Borsari, & Van Tyne, 2010). Undergraduates also commonly participate in drinking games (Pedersen & LaBrie, 2006) and consume caffeinated cocktails, such as alcoholic energy drinks (Brache & Stockwell, 2011; O’Brien, McCoy, Rhodes, Wagoner, & Wolfson, 2008). These activities are associated with heavier drinking patterns, and more alcohol-related problems in university samples (Brache & Stockwell, 2011; LaBrie & Pedersen, 2008; O’Brien et al., 2008; Pedersen & LaBrie, 2006; Price, Hilchey, Darredeau, Fulton, & Barrett, 2010).

Not surprisingly, undergraduate students experience a range of alcohol-related problems affecting different areas of their lives. Heavy drinking is associated with academic consequences, such as

decreased scholastic achievement, impaired athletic performance, campus infractions, and university attrition (Martinez, Sher, & Wood, 2008; Perkins, 2002; Wechsler et al., 1994). Additionally, drinking among university students can lead to high-risk behaviours such as unsafe sex and impaired driving (Neal & Fromme, 2007; Perkins, 2002), personal injury, and unintentional death (Hingson et al., 2009; Perkins, 2002; Wechsler et al., 1994). Undergraduate drinking is also associated with harm to others, such as property damage, mischief, and interpersonal violence (Hingson et al., 2009; Perkins, 2002). There is a positive relationship between the degree of alcohol involvement (e.g., greater frequency of binge episodes) and alcohol-related consequences in undergraduates (Wechsler et al., 1999). Importantly, maladaptive drinking during the undergraduate years significantly predicts future progression to AUDs (O'Neill et al., 2001).

The unique patterns of alcohol consumption and alcohol-related problems among undergraduates likely relate to factors associated with the particular cultural milieu of university. For most students, undergraduate studies mark a time of newfound independence, socialization, and stress (Robotham & Julian, 2006; Vaughan, Corbin, & Fromme, 2009) – all of which may be conducive to increased alcohol intake. Related to independence and flexibility, students' schedules appear to contribute to their drinking patterns, with the majority (71%) of total alcohol consumption occurring on Thursdays, Fridays, and Saturdays (Del Boca, Darkes, Greenbaum, & Goldman, 2004). Interestingly, excessive drinking behaviour on Thursday nights is moderated by whether students have Friday classes (Wood, Sher, & Rutledge, 2007). Similarly, breaks in the regular academic schedule, such as spring break and orientation week (a.k.a., 'Frosh Week'), are high-risk periods for heavy alcohol use (Mallett et al., 2013).

Regarding social influences, social engagement and motives (i.e., personal importance of socializing) are positively associated with alcohol intake among undergraduates (Vaughan et al., 2009; Wechsler, Dowdall, Davenport, & Castillo, 1995). Additionally, university students vastly overestimate the consumption rates of their fellow students, and these misperceptions of peer norms significantly predict personal alcohol consumption (Perkins, 2007). Stress has also been shown to affect undergraduate

drinking, in that students consume a greater volume of alcohol on days that they perceive as stressful (Park et al., 2004). Genetic studies using sibling-pair designs have shown that university attendance moderates the relationship between genetic risk and drinking behaviour, supporting a gene by environment interaction (Timberlake et al., 2007). Such data suggest that it is the exposure to a university environment that increases risk for excessive drinking, as opposed to university-attending individuals being inherently vulnerable to this pattern of behaviour.

Longitudinal analyses have shown that rates of drinking and alcohol-related mortality among undergraduates have remained stable over recent years, despite increased on-campus interventions aimed at reducing heavy drinking (Henson, Pearson, & Carey, 2015; Hingson et al., 2009; Wechsler et al., 2002). Although most undergraduate students will exhibit a normative decline in alcohol use as they move on from university, some will go on to develop AUD (Lee et al., 2013; O'Neill et al., 2001). Thus, it remains critically important to inform, and hopefully improve, early interventions by identifying factors that confer direct or indirect risk for progression to alcoholism in this population.

Individual Differences in Risk for Alcoholism

The development of AUD is a complex and multifactorial phenomenon. At the most basic level, risk for alcoholism has been framed according to two paradigms: an exposure, or drug-centered, model and an individual-differences model (Piazza & Le Moal, 1996; Swendsen & Le Moal, 2011). In support of the exposure model, a large body of animal and human research has demonstrated that repeated exposure to drugs of abuse, including alcohol, is associated with persistent neural changes that are, in turn, implicated in the behavioural pathology of addiction (Koob & Volkow, 2010). This model has contributed significantly to our understanding of addiction and, especially, its neurobiological underpinnings. Despite its utility, the exposure model does not explain why many undergraduate students drink heavily during university, but not all go on to develop AUD.

The fact that repeated exposure to alcohol is insufficient to account for progression to alcoholism suggests that there is variability between individuals in terms of vulnerability. Consistently, the

individual-centered model proposes that the propensity for developing alcoholism reflects inter-individual differences in trait, behavioural, physiological, or genetic factors (Swendsen & Le Moal, 2011). Integrating this view with the exposure model, addiction is considered “a behavioral disorder occurring in a vulnerable phenotype, in which an intrinsic predisposed state determines the neuroplasticity that is induced by psychoactive substances (p. 74; Swendsen & Le Moal, 2011)”. In other words, various environmental, biological, and psychological factors drive individuals to drink, and continued repetitive exposure to alcohol produces brain changes that are critical to the compulsivity that typifies alcoholism.

Among the etiologic models of AUD that center on individual differences, theories related to affect regulation have gained considerable support (Barr & Goldman, 2006; Kuntsche, Knibbe, Gmel, & Engels, 2005; Sher et al., 2005). The view that individuals drink to regulate both positive and negative emotional experiences has been widely supported in the scientific literature (Cooper, Frone, Russell, & Mudar, 1995; Khantzian, 1997, 1999; Koob, 2008). Alcohol can be a positive reinforcer, in that individuals consume alcohol to attain or enhance positive subjective states, such as pleasure. As proposed by the self-medication hypothesis (Khantzian, 1997), some individuals drink to diminish or avoid aversive affective states, such as anxiety, with alcohol acting as a negative reinforcer. Respectively, these drinking behaviours are sometimes referred to as ‘reward drinking’ and ‘relief drinking’ (e.g., Barr, 2013). The affective benefits associated with drinking can relate directly to the pharmacological or expectancy effects of alcohol, or from secondary effects, such as social inclusion (Kuntsche et al., 2005).

Alternatively, drinking may be reinforced by the pharmacological or subjective effects of alcohol. According to the pharmacological vulnerability hypothesis and similar views, individual differences in response to alcohol may confer variability in vulnerability to alcoholism (Sher et al., 2005). Two prominent paradigms have been put forward to explain the relationship between subjective alcohol response and excessive drinking. The low level of response model (e.g., Schuckit, 1987) posits that attenuated responses to the range of alcohol effects may drive an individual to consume more alcohol, and the differentiator model (Newlin & Thomson, 1990) suggests that high risk is related to increased

sensitivity to positive effects and/or decreased sensitivity to aversive effects of alcohol (Morean & Corbin, 2010).

Risk Factors for Alcoholism

Across the body of preclinical and human research on individual differences and excessive drinking, the most prominent factors affecting vulnerability are: (1) stress, (2) behavioural disinhibition, and (3) alcohol response (for reviews see Barr & Goldman, 2006; Quinn & Fromme, 2011b; Schuckit, 2002; Sher et al., 2005). The first two factors relate to affective regulation models of alcoholism, and the latter factor is in line with the view that inter-individual variability in subjective alcohol response is related to risk for AUD. These factors appear to be similarly implicated in excessive drinking among undergraduate populations (for reviews see Baer, 2002; Ham & Hope, 2003).

Stress.

In the context of psychology, stress is operationalized as an individual's response to disruption of physical or psychological equilibrium by a challenge (i.e., stressor), which can be internal or external (Johnson, Kamilaris, Chrousos, & Gold, 1992; Selye, 1936). Stress response manifests as a number of subjective psychological and physiological effects (Schlotz et al., 2008). The psychological effects of stress involve cognitive and affective changes, such as subjective worry, rumination, nervousness, tension, and anxiety. These subjective effects are typically measured by self-report questionnaires wherein individuals rate the extent to which they are experiencing a stress-related trait or state, such as anxiety or tension. For example, the State-Trait Anxiety Inventory (Spielberger, 1983) is a questionnaire assessing subjective anxiety, in which statements (e.g., "I am tense") are rated on a Likert scale.

Physiological stress response involves a cascade of autonomic nervous system and neuroendocrine changes (Johnson et al., 1992). Stress-induced sympathetic nervous system responses include various changes such as elevated heart rate, blood pressure, and respiration (Chrousos & Gold, 1992; Habib, Gold, & Chrousos, 2001), which are collectively referred to as the 'fight-or-flight' response (Cannon, 1915). Two primary pathways of the biochemical stress system are the hypothalamic-pituitary-

adrenal (HPA) axis and the sympathetic-adrenal-medullary (SAM) system (Habib et al., 2001). Cortisol, a glucocorticoid end-product released by the adrenal cortex, is a reliable indicator of HPA axis activity (Kudielka, Hellhammer, & Wüst, 2009). The enzyme alpha amylase is a popular proxy measure of SAM activation (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996). Conveniently, these compounds can be easily measured through biochemical assays of saliva samples (Kirschbaum & Hellhammer, 1994). The physiological stress response exhibits high temporal covariance with stress-induced psychological states, supporting the view that both types of metrics represent the same theoretical construct (Schlotz et al., 2008).

The relationship between stress and alcohol use is well established, and supported by several lines of evidence. Epidemiological studies show that AUDs are highly comorbid with anxiety disorders, such as social anxiety and generalized anxiety disorder (Dawson, Grant, Stinson, et al., 2005; Kushner, Abrams, & Borchardt, 2000). Consistently, abnormal HPA activation has been associated with a range of psychiatric disorders, including AUD (Chrousos & Gold, 1992). Compared to normal controls, individuals with AUD typically exhibit abnormal baseline activity and blunted stress responsivity of the HPA axis (Lovallo, 2006; Sinha et al., 2011). In fact, preclinical and human studies have implicated stress in all facets of AUD, including drug-seeking, craving, and relapse (for reviews see Gianoulakis, 1998; Koob, 2008; Piazza & Le Moal, 1996; Uhart & Wand, 2009). Similarly, HPA axis hypo-responsivity is seen among individuals at increased risk for AUD, such as children of alcoholics (Sorocco, Lovallo, Vincent, & Collins, 2006). In rodent models, pharmacological blockade of the stress system attenuates heavy, but not moderate, drinking (Cippitelli et al., 2012), which highlights the role of the stress system in pathological drinking.

In the general population, greater exposure to stressful life events, such as job loss or divorce, is associated with heavy drinking and increased AUD symptoms, and this relationship is stronger with increased subjective distress (Boden, Fergusson, & Horwood, 2014; Dawson, Grant, & Ruan, 2005). Longitudinal analyses support the view that exposure to stressful life events leads to AUD symptoms, as

opposed to the opposite unidirectional or bidirectional models (Boden et al., 2014). Among university students, there is a positive relationship between daily changes in subjective stress and anxiety, and alcohol intake (Park, Armeli & Tennen, 2004). Moreover, this relationship is stronger among students who report using alcohol for the express purpose of managing anxiety (Grant, Stewart, & Mohr, 2009). Some studies suggest that acute stress directly increases drinking behaviour in humans or animals, however this finding is not consistently reported across the literature (see Sher et al., 2005). Taken together, this body of research demonstrates that individual differences in subjective and physiological responses to stress are important factors for vulnerability to excessive alcohol consumption.

Disinhibited Behaviour.

Across personality dimensions, those related to behavioural disinhibition appear to have the most consistent and strongest link to misuse of substances, including alcohol (Baer, 2002). Personality refers to a pattern of cognition, emotion, and behaviour that remain stable over time and across situations within an individual (Steyer, Schmitt, & Eid, 1999). This construct is also referred to as trait factors or temperament, and can be thought of as a stable behavioural phenotype. Personality is among the most commonly studied predictors of drinking behaviour, in general and among undergraduate students in particular. Despite a proliferation of related research over the past several decades, no particular personality type or group of traits has emerged as a specific predictor of alcohol misuse (i.e., “alcoholic personality” (Sher et al., 2005). Nonetheless, disinhibited behaviour has emerged as a stable phenotype associated with increased risk for excessive alcohol use.

Impulsivity and sensation seeking (SS) are arguably the most researched traits related to disinhibited behaviour (Hittner & Swickert, 2006). Although impulsivity has been described a number of ways by different researchers and clinicians, integrative biopsychosocial conceptualizations have attempted to reconcile the various models, defining impulsivity as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others (p. 1784; Moeller, Barratt, Dougherty, Schmitz, &

Swann, 2001)”. In contrast, SS is conceptualized as the tendency to seek out and participate in novel experiences despite significant risks (Zuckerman, 1979). Although impulsivity and SS are related in terms of disinhibition, they form conceptually and psychometrically distinct constructs. SS is typified by a strong drive for intense and novel experiences while disregarding the serious risks, as opposed to acting quickly without consideration for negative consequences, as is seen in impulsivity (Moeller et al., 2001). The distinction between impulsivity and SS has also been corroborated via factor analysis, and has exhibited differential influences on measures of drinking behaviour (Magid, MacLean, & Colder, 2007; Smith et al., 2007).

Behavioural disinhibition may be particularly important in the initial stages of substance use (Koob, 2009; Kreek, Nielsen, & LaForge, 2004). In support of this view, drinking onset typically occurs in adolescence, a period that coincides with high levels of behavioural disinhibition (Crews & Boettiger, 2009; Spear, 2000). Additionally, cross-sectional and longitudinal studies consistently show that high impulsivity and SS are significantly implicated in the progression to risky drinking and AUDs (for reviews see De Wit, 2009; Hittner & Swickert, 2006; Perry & Carroll, 2008), findings that have been replicated in animal models (for reviews see Blanchard, Mendelsohn, & Stamp, 2009; Dick et al., 2010). Impulsivity and SS, respectively, are positively associated with coping (i.e., negative affect regulation) and enhancement (i.e., positive affect regulation) motives for drinking (Cooper et al., 1995; Magid et al., 2007), both of which relate to increased risk for AUD.

The role of disinhibited behaviour in drinking has drawn particular attention in the context of undergraduate students. To illustrate, nearly 50% of studies examining the relationship between SS and alcohol consumption involve university populations (see Hittner & Swickert, 2006). Personality theorists have proposed that, in general, individual differences in trait factors will exert the greatest effect on behaviour during critical transitional periods, as individuals adjust to novel circumstances (Caspi & Moffitt, 1993). Likewise, the relationship between disinhibited behaviour and drinking may be particularly salient in undergraduate populations (Borsari, Murphy, & Barnett, 2007). For example, SS is

related to frequency of alcohol use and binge drinking in university students, as well as increases in these measures between high school and university (Del Boca et al., 2004; White et al., 2006), whereas these associations do not emerge in age-matched peers who do not attend university (Raskin White et al., 2006).

Response to Alcohol.

Drinking is associated with a number of subjective effects, such as feelings of intoxication, stimulation, sedation, ‘high’, and pleasure (Quinn & Fromme, 2011a). Alcohol responses are commonly assessed using alcohol-challenge experiments, wherein participants complete self-report and/or physiological measures of alcohol effects after drinking. Alcohol challenge designs are further strengthened by the inclusion of appropriate controls, ideally placebo and non-alcoholic beverage groups as well as multiple doses of alcohol (Morean & Corbin, 2010). Common self-report measures of subjective alcohol responses include the Biphasic Alcohol Effects Scale (Martin, Earleywine, Musty, Perrine, & Swift, 1993), which assesses symptoms of stimulation and sedation, and the Drug Effects Questionnaire (Kirk & de Wit, 2000), which examines a range of subjective effects, such as feelings of intoxication, liking the effects and desire for more of the drug. Physiological responses to alcohol are assessed by a variety of measures, including heart rate, skin conductance, and endocrine response such as prolactin and cortisol (Brunelle & Pihl, 2007; King, Munisamy, De Wit, & Lin, 2006; Newlin & Thomson, 1990; Soyka, Gorig, & Naber, 1992).

There are vast inter-individual differences in alcohol responses across the range of both subjective and physiological effects and these relate to risk for excessive drinking (Sher et al., 2001). Consistent with the low level of response theory (e.g., Schuckit, 1987), diminished sensitivity to the effects of alcohol is robustly associated with levels of alcohol consumption and alcohol-related problems (Schuckit et al., 2012), and prospectively predicts the progression to AUD (King, de Wit, McNamara, & Cao, 2011; Schuckit, Smith, Anderson, & Brown, 2004). Among a sample of heavy-drinking young adults, retrospectively-reported low subjective response to alcohol during their first five drinking

experiences significantly predicted weekly alcohol consumption, when controlling for acquired tolerance (Corbin et al., 2013). In other words, individuals who needed more drinks to perceive alcohol effects in their earliest drinking experiences went on to exhibit heavier patterns of intake as young adults. These findings raise the possibility that individual differences in alcohol responsivity may predate the progression to excessive drinking.

As proposed by the differentiator model (Newlin & Thomson, 1990), greater risk for excessive drinking is associated with increased sensitivity to reinforcing effects of alcohol and decreased sensitivity to the aversive effects of alcohol. This view is closely related to the observation that alcohol produces both subjective stimulating and sedating effects – so-called ‘biphasic’ effects – as blood alcohol levels rise and fall, respectively (Martin et al., 1993). Greater stimulation related to alcohol intoxication is positively associated with desire for more of the drug (Childs, O’Connor, & de Wit, 2011). Similarly, at-risk groups (e.g., heavy drinkers) exhibit greater stimulation and decreased sedation by alcohol, which, in turn, prospectively predict AUDs (King et al., 2011).

In general, both the low level of response and differentiator models are supported in the literature (for reviews see Morean & Corbin, 2010; Quinn & Fromme, 2011). Theorists have attempted to reconcile the two prevailing theories, suggesting that both low- and high-sensitivity to the positive effects of alcohol, along with diminished response to the aversive effects of alcohol, are important in explaining variability in risk for AUD (e.g., King et al., 2011). Consistently, in undergraduate populations, all three types of subjective response to alcohol have been shown to distinguish healthy social drinkers from high-risk groups, such as children of alcoholics and binge drinkers (Balodis, Potenza, & Olmstead, 2009; Newlin & Thomson, 1990). Emerging data demonstrate that each of these patterns of subjective alcohol responses is associated with different gene variants (Roh et al., 2011; Uhart et al., 2012). Thus, one way in which genetic variation may confer risk for AUD is by moderating the behavioural phenotype related to subjective responses to alcohol.

Purpose of Current Studies

Consistent with the individual difference model of alcoholism, the studies presented in the subsequent chapters seek to elucidate the role of inter-individual variability in risk factors for AUD among undergraduate students. As demonstrated in the literature, undergraduates comprise a high-risk population in terms of excessive drinking. The main individual-difference factors of interest for the current studies are behavioural disinhibition, stress/anxiety, and subjective alcohol response. Past research has confirmed that variation in these factors is significantly linked to excessive alcohol use. The current dissertation explores how these risk factors relate to measures of drinking-related behaviour that, in turn, have been implicated in risk for AUD. These include heavy and hazardous patterns of drinking, drinking in response to stress, and increased neuroendocrine response to alcohol (as summarized in Table 1-1 below).

Table 1-1 Summary of risk factors and drinking behaviours examined in each study

Study	Risk Factor(s)	Drinking Behaviour
1	Disinhibited behaviour Stress/ anxiety	Hazardous drinking patterns
2	Disinhibited behaviour Subjective alcohol response	Neuroendocrine response to alcohol
3	Stress/ anxiety	Stress-related drinking

The first study (Chapter 2) explores how individual differences in stable trait factors predict drinking pattern changes in the transition from high-school to undergraduate studies. The selected trait factors relate to known risk factors for alcoholism, namely disinhibited behaviour (impulsivity and sensation seeking) and stress (anxiety sensitivity). By employing a prospective design, this study is able to assess how pre-existing variability in intrapersonal risk factors predicts changes in alcohol consumption, hazardous drinking, and alcohol-related problems in the first year of university.

Despite the fact that stress is well-established as a risk factor for increased drinking, surprisingly few studies have examined whether individuals actually increase their intake of alcohol following a stressor. The second experiment (Chapter 3; Magrys & Olmstead, 2015) examines whether exposure to an acute stressor moderates individuals' voluntary consumption of alcohol. Moreover, it assesses whether individual differences in state and trait anxiety affect stress-related alcohol consumption.

In Study 3 (Chapter 4; Magrys, Olmstead, Wynne-Edwards, & Balodis, 2013), individual differences in impulsivity and subjective alcohol responses are examined in relation to neuroendocrine responses to alcohol. Alcohol, a physiological stressor, activates the biochemical stress system; understanding how individual difference factors interact with this phenomenon may help to explain the link between hormonal stress responsivity and risk for AUD.

References

- Aalto, M., Alho, H., Halme, J. T., & Seppä, K. (2009). AUDIT and its abbreviated versions in detecting heavy and binge drinking in a general population survey. *Drug and Alcohol Dependence, 103*(1-2), 25–29. <http://doi.org/10.1016/j.drugalcdep.2009.02.013>
- Abrams, K., & Kushner, M. G. (2004). The moderating effects of tension-reduction alcohol outcome expectancies on placebo responding in individuals with social phobia. *Addictive Behaviors, 29*(6), 1221–1224. <http://doi.org/10.1016/j.addbeh.2004.03.020>
- Abrams, K., Kushner, M. G., Medina, K. L., & Voight, A. (2002). Self-administration of alcohol before and after a public speaking challenge by individuals with social phobia. *Psychology of Addictive Behaviors, 16*(2), 121–128. <http://doi.org/10.1037/h0092445>
- Armeli, S., Conner, T. S., Cullum, J., & Tennen, H. (2010). A longitudinal analysis of drinking motives moderating the negative affect-drinking association among college students. *Psychology of Addictive Behaviors, 24*(1), 38–47. <http://doi.org/10.1037/a0017530>
- Baer, J. S. (2002). Student factors: understanding individual variation in college drinking. *Journal of Studies on Alcohol. Supplement, 1986*(14), 40–53.
- Balodis, I. M., Potenza, M. N., & Olmstead, M. C. (2009). Binge drinking in undergraduates: Relationship with gender, drinking behaviors, impulsivity and the perceived effects of alcohol. *Behavioral Pharmacology, 20*(5-6), 518–526. <http://doi.org/10.1097/FBP.0b013e328330c779>.Binge
- Balodis, I. M., Wynne-Edwards, K. E., & Olmstead, M. C. (2011). The stress-response-dampening effects of placebo. *Hormones and Behavior, 59*(4), 465–472. <http://doi.org/10.1016/j.yhbeh.2011.01.004>

- Bari, A., Robbins, T., & Dalley, J. (2011). Impulsivity. In M. C. Olmstead (Ed.), *Animal Models of Drug Addiction* (pp. 379–401). New York: Springer Science.
- Barr, C. S., & Goldman, D. (2006). Non-human primate models of inheritance vulnerability to alcohol use disorders. *Addiction*, 374–385. <http://doi.org/10.1111/j.1369-1600.2006.00034.x>
- Blanchard, M. M., Mendelsohn, D., & Stamp, J. A. (2009). The HR/LR model: Further evidence as an animal model of sensation seeking. *Neuroscience and Biobehavioral Reviews*, 33(7), 1145–1154. <http://doi.org/10.1016/j.neubiorev.2009.05.009>
- Boden, J. M., Fergusson, D. M., & Horwood, L. J. (2014). Associations between exposure to stressful life events and alcohol use disorder in a longitudinal birth cohort studied to age 30. *Drug and Alcohol Dependence*, 142, 154–160. <http://doi.org/10.1016/j.drugalcdep.2014.06.010>
- Borsari, B., Boyle, K. E., Hustad, J. T. P., Barnett, N. P., O’Leary Tevyaw, T., & Kahler, C. W. (2007). Drinking before drinking: Pregaming and drinking games in mandated students. *Addictive Behaviors*, 32(11), 2694–2705. <http://doi.org/10.1016/j.addbeh.2007.05.003>
- Borsari, B., & Carey, K. B. (2001). Peer influences on college drinking: A review of the research. *Journal of Substance Abuse*, 13(4), 391–424. [http://doi.org/10.1016/S0899-3289\(01\)00098-0](http://doi.org/10.1016/S0899-3289(01)00098-0)
- Borsari, B., Murphy, J. G., & Barnett, N. P. (2007). Predictors of alcohol use during the first year of college: Implications for prevention. *Addictive Behaviors*, 32(10), 2062–2086. <http://doi.org/10.1016/j.addbeh.2007.01.017>
- Bouchery, E. E., Harwood, H. J., Sacks, J. J., Simon, C. J., & Brewer, R. D. (2011). Economic costs of excessive alcohol consumption in the U.S., 2006. *American Journal of Preventive Medicine*, 41(5), 516–524. <http://doi.org/10.1016/j.amepre.2011.06.045>

- Brache, K., & Stockwell, T. (2011). Drinking patterns and risk behaviors associated with combined alcohol and energy drink consumption in college drinkers. *Addictive Behaviors, 36*(12), 1133–1140. <http://doi.org/10.1016/j.addbeh.2011.07.003>
- Brunelle, C., & Pihl, R. O. (2007). Effects of conditioned reward and nonreward cues on the heart rate response to alcohol intoxication in male social drinkers. *Alcoholism: Clinical and Experimental Research, 31*(3), 383–389. <http://doi.org/10.1111/j.1530-0277.2006.00318.x>
- Butt, P., Beirness, D., Stockwell, T., Gliksman, L., & Paradis, C. (2011). *Alcohol and health in Canada : A summary of evidence and guidelines for low-risk drinking*. Ottawa, ON: Canadian Centre on Substance Abuse
- Carroll, M. E., & Meisch, R. A. (2011). Acquisition of drug self-administration. In M. C. Olmstead (Ed.), *Animal models of drug addiction* (pp. 237–265). New York: Springer Science.
- Caspi, A., & Moffitt, T. E. (1993). When do individual differences matter? A paradoxical theory of personality coherence. *Psychological Inquiry, 4*(4), 247–271. http://doi.org/10.1207/s15327965pli0404_1
- Chassin, L., Forthofer, D. B., & King, K. M. (2004). Trajectories of alcohol and drug use and dependence from adolescence to adulthood: the effects of familial alcoholism and personality. *Journal of Abnormal Psychology, 113*(4), 483–498. <http://doi.org/10.1037/0021-843X.113.4.483>
- Childs, E., O'Connor, S., & de Wit, H. (2011). Bidirectional interactions between acute psychosocial stress and acute intravenous alcohol in healthy men. *Alcoholism: Clinical and Experimental Research, 35*(10), 1794–1803. <http://doi.org/10.1111/j.1530-0277.2011.01522.x>

- Christiansen, P., Rose, A. K., Cole, J. C., & Field, M. (2013). A comparison of the anticipated and pharmacological effects of alcohol on cognitive bias, executive function, craving and ad-lib drinking. *Journal of Psychopharmacology*, *27*(1), 84-92. <http://doi.org/10.1177/0269881112450787>
- Chrousos, G. P. (1998). Stressors, stress and neuroendocrine integration of the adaptative r-response. *Annals of the New York Academy of Sciences*, *30*(851), 311-335. <http://doi.org/10.1111/j.1749-6632.1998.tb09006.x>
- Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *The Journal of the American Medical Association*, *267*(9), 1244–1252. <http://doi.org/10.1001/jama.267.9.1244>
- Chutuape, M. A. D., & de Wit, H. (1995). Preferences for ethanol and diazepam in anxious individuals: an evaluation of the self-medication hypothesis. *Psychopharmacology*, *121*(1), 91–103. <http://doi.org/10.1007/BF02245595>
- Chutuape, M. A. D., Mitchell, S. H., & de Wit, H. (1994). Ethanol preloads increase ethanol preference under concurrent random-ratio schedules in social drinkers. *Experimental and Clinical Psychopharmacology*, *2*(4), 310–318. <http://doi.org/10.1037/1064-1297.2.4.310>
- Cippitelli, A., Damadzic, R., Singley, E., Thorsell, A., Ciccocioppo, R., Eskay, R. L., & Heilig, M. (2012). Pharmacological blockade of corticotropin-releasing hormone receptor 1 (CRH1R) reduces voluntary consumption of high alcohol concentrations in non-dependent Wistar rats. *Pharmacology Biochemistry and Behavior*, *100*(3), 522–529. <http://doi.org/10.1016/j.pbb.2011.10.016>
- Collins, R. L., Kashdan, T. B., Koutsky, J. R., Morsheimer, E. T., & Vetter, C. J. (2008). A self-administered Timeline Followback to measure variations in underage drinkers' alcohol intake and binge drinking. *Addictive Behaviors*, *33*(1), 196–200. <http://doi.org/10.1016/j.addbeh.2007.07.001>

- Conrad, M., McNamara, P., & King, A. (2012). Alternative substance paradigm: Effectiveness of beverage blinding and effects on acute alcohol responses. *Experimental and Clinical Psychopharmacology*, 20(5), 382–389. <http://doi.org/10.1037/a0029261>
- Conrod, P. J., Stewart, S. H., Comeau, N., & Maclean, A. M. (2006). Efficacy of cognitive-behavioral interventions targeting personality risk factors for youth alcohol misuse. *Journal of Clinical Child and Adolescent Psychology*, 35(4), 550–563. http://doi.org/10.1207/s15374424jccp3504_6
- Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: a motivational model of alcohol use. *Journal of Personality and Social Psychology*, 69(5), 990–1005. <http://doi.org/10.1037/0022-3514.69.5.990>
- Corbin, W. R., Gearhardt, A., & Fromme, K. (2008). Stimulant alcohol effects prime within session drinking behavior. *Psychopharmacology*, 197(2), 327–337. <http://doi.org/10.1007/s00213-007-1039-x>
- Corbin, W. R., Scott, C., Leeman, R. F., Fucito, L. M., Toll, B. A., & O'Malley, S. S. (2013). Early subjective response and acquired tolerance as predictors of alcohol use and related problems in a clinical sample. *Alcoholism: Clinical and Experimental Research*, 37(3), 490–497. <http://doi.org/10.1111/j.1530-0277.2012.01956.x>
- Courtney, K. E., & Polich, J. (2009). Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin*, 135(1), 142–156. <http://doi.org/10.1037/a0014414>.Binge
- Crews, F. T., & Boettiger, C. A. (2009). Impulsivity, frontal lobes and risk for addiction. *Pharmacology Biochemistry and Behavior*, 93(3), 237–247. <http://doi.org/10.1016/j.pbb.2009.04.018>

- Dawson, D. A., Grant, B. F., & Ruan, W. J. (2005). The association between stress and drinking: Modifying effects of gender and vulnerability. *Alcohol and Alcoholism*, *40*(5), 453–460.
<http://doi.org/10.1093/alcalc/agh176>
- Dawson, D. A., Grant, B. F., Stinson, F. S., Chou, P. S., Huang, B., & Ruan, W. J. (2005). Recovery from DSM-IV alcohol dependence: United States, 2001–2002. *Addiction*, *100*(3), 281–292.
<http://doi.org/10.1111/j.1360-0443.2004.00964.x>
- De Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, *14*(1), 22–31. <http://doi.org/10.1111/j.1369-1600.2008.00129.x>
- Del Boca, F. K., Darkes, J., Greenbaum, P. E., & Goldman, M. S. (2004). Up close and personal: temporal variability in the drinking of individual college students during their first year. *Journal of Consulting and Clinical Psychology*, *72*(2), 155–164. <http://doi.org/10.1037/0022-006X.72.2.155>
- Dick, D. M., Smith, G., Olausson, P., Mitchell, S. H., Leeman, R. F., O'Malley, S. S., & Sher, K. (2010). Understanding the construct of impulsivity and its relationship to alcohol use disorders. *Addiction Biology*, *15*(2), 217–226. <http://doi.org/10.1111/j.1369-1600.2009.00190.x>
- Dixon, L. J., Stevens, E. N., & Viana, A. G. (2014). Anxiety sensitivity as a moderator of the relationship between trait anxiety and illicit substance use. *Psychology of Addictive Behaviors*, *28*(4), 1284–1289.
- Esser, M. B., Kanny, D., Brewer, R. D., & Naimi, T. S. (2012). Binge drinking intensity: A comparison of two measures. *American Journal of Preventive Medicine*, *42*(6), 625–629.
<http://doi.org/10.1016/j.amepre.2012.03.001>

- Fernie, G., Christiansen, P., Cole, J. C., Rose, A.K., & Field, M. (2012). Effects of 0.4g/kg alcohol on attentional bias and alcohol-seeking behaviour in heavy and moderate social drinkers. *Journal of Psychopharmacology*, *26*(7), 1017–1025. <http://doi.org/10.1177/0269881111434621>
- Field, M., Wiers, R.W., Christiansen, P., Fillmore, M.T., & Verster, J.C. (2010). Acute alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking. *Alcoholism: Clinical and Experimental Research*, *34*(8), 1346–1352. <http://doi.org/10.1111/j.1530-0277.2010.01218.x>
- Fitzgerald, K.A., & Long, C.G. (2012). The influence of drinking motives and anxiety on low-, moderate- and high-risk drinkers. *Journal of Substance Use*, *17*(3), 240–248. <http://doi.org/10.3109/14659891.2011.565109>
- Fromme, K., Corbin, W.R., & Kruse, M. I. (2008). Behavioral risks during the transition from high school to college. *Developmental Psychology*, *44*(5), 1497–1504. <http://doi.org/10.1037/a0012614>
- Gianoulakis, C. (1998). Alcohol-seeking behavior. *Alcohol Health & Research World*, *22*(3), 202–210.
- Gmel, G., Kuntsche, E., & Rehm, J. (2011). Risky single-occasion drinking: Bingeing is not bingeing. *Addiction*, *106*(6), 1037–1045. <http://doi.org/10.1111/j.1360-0443.2010.03167.x>
- Goldman, D., Oroszi, G., & Ducci, F. (2005). The genetics of addictions: uncovering the genes. *Nature Reviews. Genetics*, *6*(7), 521–532. <http://doi.org/10.1038/nrg1635>
- Gotham, H. J., Sher, K. J., & Wood, P. K. (1997). Predicting stability and change in frequency of intoxication from the college years to beyond: individual-difference and role transition variables. *Journal of Abnormal Psychology*, *106*(4), 619–629. <http://doi.org/10.1037/0021-843X.106.4.619>

- Grant, V. V., Stewart, S. H., & Mohr, C. D. (2009). Coping-anxiety and coping-depression motives predict different daily mood-drinking relationships. *Psychology of Addictive Behaviors, 23*(2), 226–237. <http://doi.org/10.1037/a0015006>
- Greeley, J. D., & Oei, T. P. S. (1999). Alcohol and tension reduction. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (pp. 14–53). New York: The Guilford Press.
- Grekin, E. R., & Sher, K. J. (2006). Alcohol dependence symptoms among college freshmen: prevalence, stability, and person-environment interactions. *Experimental and Clinical Psychopharmacology, 14*(3), 329–338. <http://doi.org/10.1037/1064-1297.14.3.329>
- Habib, K. E., Gold, P. W., & Chrousos, G. P. (2001). Neuroendocrinology of stress. *Endocrinology and Metabolism Clinics of North America, 30*(3), 695–728. [http://doi.org/10.1016/S0889-8529\(05\)70208-5](http://doi.org/10.1016/S0889-8529(05)70208-5)
- Ham, L. S., & Hope, D. A. (2003). College students and problematic drinking: A review of the literature. *Clinical Psychology Review, 23*(5), 719–759. [http://doi.org/10.1016/S0272-7358\(03\)00071-0](http://doi.org/10.1016/S0272-7358(03)00071-0)
- Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry, 64*(7), 830–842. <http://doi.org/10.1001/archpsyc.64.7.830>
- Henson, J. M., Pearson, M. R., & Carey, K. B. (2015). Defining and characterizing differences in college alcohol intervention efficacy : A growth mixture modeling application, *Journal of Consulting and Clinical Psychology, 83*(2), 370–381. <http://doi.org/10.1037/a0038897>

- Hingson, R. W., Zha, W., & Weitzman, E. R. (2009). Magnitude of and trends in alcohol-related mortality and morbidity among U.S. college students ages 18-24, 1998-2005. *Journal of Studies on Alcohol and Drugs. Supplement*, (16), 12–20.
<http://doi.org/10.1146/annurev.publhealth.26.021304.144652>
- Hittner, J. B., & Swickert, R. (2006). Sensation seeking and alcohol use: A meta-analytic review. *Addictive Behaviors*, 31(8), 1383–1401. <http://doi.org/10.1016/j.addbeh.2005.11.004>
- Jackson, K. M., Sher, K. J., & Park, A. (2005). Drinking among college students. Consumption and consequences. *Recent Developments in Alcoholism*, 17, 85–117. http://doi.org/10.1007/0-306-48626-1_5
- Johnson, E. O., Kamilaris, T. C., Chrousos, G. P., & Gold, P. W. (1992). Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neuroscience and Biobehavioral Reviews*, 16(2), 115–130. [http://doi.org/10.1016/S0149-7634\(05\)80175-7](http://doi.org/10.1016/S0149-7634(05)80175-7)
- Kaim, S. C., Brill, H., Cloud, L. A., Knott, D. H., Lieber, C. S., McIsaac, W. M., et al. (1972). Rationale for the diagnosis. *American Journal of Psychiatry*, 129(2), 127–134.
- Kerr, J. S. (1996). Two myths of addiction: The addictive personality and the issue of free choice. *Human Psychopharmacology*, 11(1), S9-S13. [http://doi.org/10.1002/\(SICI\)1099-1077\(199602\)11:1+<S9::AID-HUP747>3.0.CO;2-6](http://doi.org/10.1002/(SICI)1099-1077(199602)11:1+<S9::AID-HUP747>3.0.CO;2-6)
- Khantzian, E. J. (1997). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, 4(5), 231–244.
<http://doi.org/10.3109/10673229709030550>

- Khantzian, E. J. (1999). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, *4*(5), 231–244.
<http://doi.org/10.3109/10673229709030550>
- King, A. C., de Wit, H., McNamara, P. J., & Cao, D. (2011). Rewarding, stimulant, and sedative alcohol responses and relationship to future binge drinking. *Archives of General Psychiatry*, *68*(4), 389–399.
<http://doi.org/10.1001/archgenpsychiatry.2011.26>
- King, A., Munisamy, G., De Wit, H., & Lin, S. (2006). Attenuated cortisol response to alcohol in heavy social drinkers. *International Journal of Psychophysiology*, *59*(3), 203–209.
<http://doi.org/10.1016/j.ijpsycho.2005.10.008>
- King, K. M., Patock-Peckham, J. A., Dager, A. D., Thimm, K., & Gates, J. R. (2014). On the mismeasurement of impulsivity: Trait, behavioral, and neural models in alcohol research among adolescents and young adults. *Current Addiction Reports*, *1*(1), 19–32.
<http://doi.org/10.1007/s40429-013-0005-4>
- Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology*, *19*(4), 313–333.
[http://doi.org/10.1016/0306-4530\(94\)90013-2](http://doi.org/10.1016/0306-4530(94)90013-2)
- Koob, G. F. (2008). A role for brain stress systems in addiction. *Neuron*, *59*(1), 11–34.
<http://doi.org/10.1016/j.neuron.2008.06.012>
- Koob, G. F. (2009). Brain stress systems in the amygdala and addiction. *Brain Research*, *1293*, 61–75.
<http://doi.org/10.1016/j.brainres.2009.03.038>
- Koob, G. F., & Volkow, N. D. (2010). Neurocircuitry of addiction. *Neuropsychopharmacology*, *35*(1), 217–238. <http://doi.org/10.1038/npp.2010.4>

- Kreek, M. J., Nielsen, D. a, Butelman, E. R., & LaForge, K. S. (2005). Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience*, 8(11), 1450–1457. <http://doi.org/10.1038/nn1583>
- Kreek, M. J., Nielsen, D. A, & LaForge, K. S. (2004). Genes associated with addiction: alcoholism, opiate, and cocaine addiction. *Neuromolecular Medicine*, 5(1), 85–108. <http://doi.org/10.1385/NMM:5:1:085>
- Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology*, 34(1), 2–18. <http://doi.org/10.1016/j.psyneuen.2008.10.004>
- Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2005). Why do young people drink? A review of drinking motives. *Clinical Psychology Review*, 25(7), 841–861. <http://doi.org/10.1016/j.cpr.2005.06.002>
- Kushner, M. G., Abrams, K., & Borchardt, C. (2000). The relationship between anxiety disorders and alcohol use disorders: A review of major perspectives and findings. *Clinical Psychology Review*, 20(2), 149–171. [http://doi.org/10.1016/S0272-7358\(99\)00027-6](http://doi.org/10.1016/S0272-7358(99)00027-6)
- LaBrie, J. W., & Pedersen, E. R. (2008). Prepartying promotes heightened risk in the college environment: An event-level report. *Addictive Behaviors*, 33(7), 955–959. <http://doi.org/10.1016/j.addbeh.2008.02.011>
- Lee, M. R., Chassin, L., & Villalta, I. K. (2013). Maturing out of alcohol involvement: transitions in latent drinking statuses from late adolescence to adulthood. *Development and Psychopathology*, 25(4 Pt 1), 1137–53. <http://doi.org/10.1017/S0954579413000424>

- Leshner, a I. (1999). Science-based views of drug addiction and its treatment. *The Journal of the American Medical Association*, 282(14), 1314–1316. <http://doi.org/10.1001/jama.282.14.1314>
- Leshner, A. I. (1997). Addition is a brain disease, and it matters. *Sciences, New Series*, 278(5335), 45–47. <http://doi.org/10.1126/science.278.5335.45>
- Levine, H. G. (1978). The discovery of addiction. Changing conceptions of habitual drunkenness in America. *Journal of Studies on Alcohol*, 39(1), 143–174. [http://doi.org/10.1016/0740-5472\(85\)90022-4](http://doi.org/10.1016/0740-5472(85)90022-4)
- Littlefield, A. K., & Sher, K. J. (2010). The multiple, distinct ways that personality contributes to alcohol use disorders. *Social and Personality Psychology Compass*, 4(9), 767–782. <http://doi.org/10.1111/j.1751-9004.2010.00296.x>
- Littlefield, A. K., Stevens, A. K., & Sher, K. J. (2013). Impulsivity and alcohol involvement: Multiple, distinct constructs and processes. *Current Addiction Reports*, 1(1), 33–40. <http://doi.org/10.1007/s40429-013-0004-5>
- Lovallo, W. R. (2006). Cortisol secretion patterns in addiction and addiction risk. *International Journal of Psychophysiology*, 59(3), 195–202. <http://doi.org/10.1016/j.ijpsycho.2005.10.007>
- MacDonald, T. K., Fong, G. T., Zanna, M. P., & Martineau, A. M. (2000). Alcohol myopia and condom use: can alcohol intoxication be associated with more prudent behavior? *Journal of Personality and Social Psychology*, 78(4), 605–619. <http://doi.org/10.1037/0022-3514.78.4.605>
- Magid, V., MacLean, M. G., & Colder, C. R. (2007). Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors*, 32(10), 2046–2061. <http://doi.org/10.1016/j.addbeh.2007.01.015>

- Magrys, S. A., & Olmstead, M. C. (2015). Acute stress increases voluntary consumption of alcohol in undergraduates. *Alcohol and Alcoholism*, *50*(2), 213–218. <http://doi.org/10.1093/alcalc/agu101>
- Magrys, S. A., Olmstead, M. C., Wynne-Edwards, K. E., & Balodis, I. M. (2013). Neuroendocrinological responses to alcohol intoxication in healthy males: Relationship with impulsivity, drinking behavior, and subjective effects. *Psychophysiology*, *50*(2), 204–209. <http://doi.org/10.1111/psyp.12007>
- Mallett, K. A., Varvil-Weld, L., Borsari, B., Read, J. P., Neighbors, C., & White, H. R. (2013). An update of research examining college student alcohol-related consequences: New perspectives and implications for interventions. *Alcoholism: Clinical and Experimental Research*, *37*(5), 709–716. <http://doi.org/10.1111/acer.12031>
- Martinez, J. A., Sher, K. J., & Wood, P. K. (2008). Is heavy drinking really associated with attrition from college? The alcohol-attrition paradox. *Psychology of Addictive Behaviors : Journal of the Society of Psychologists in Addictive Behaviors*, *22*(3), 450–456. <http://doi.org/10.1037/0893-164X.22.3.450>
- McGue, M., Slutske, W., & Iacono, W. G. (1999). Personality and substance use disorders: II. Alcoholism versus drug use disorders. *Journal of Consulting and Clinical Psychology*, *67*(3), 394–404. <http://doi.org/10.1037/0022-006X.67.3.394>
- McLellan, A. T., Lewis, D. C., O'Brien, C. P., & Kleber, H. D. (2000). Drug dependence, a chronic medical illness. *Journal of the American Medical Association*, *284*(13), 1689. <http://doi.org/10.1001/jama.284.13.1689>
- Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, a. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, *158*(11), 1783–1793. <http://doi.org/10.1176/appi.ajp.158.11.1783>

- Mohapatra, S., Patra, J., Popova, S., Duhig, A., & Rehm, J. (2010). Social cost of heavy drinking and alcohol dependence in high-income countries. *International Journal of Public Health, 55*(3), 149–157. <http://doi.org/10.1007/s00038-009-0108-9>
- Monahan, C. J., McDevitt-Murphy, M. E., Dennhardt, A. A., Skidmore, J. R., Martens, M. P., & Murphy, J. G. (2013). The impact of elevated posttraumatic stress on the efficacy of brief alcohol interventions for heavy drinking college students. *Addictive Behaviors, 38*(3), 1719–1725. <http://doi.org/10.1016/j.addbeh.2012.09.004>
- Moos, R. H., & Moos, B. S. (2006). Rates and predictors of relapse after natural and treated remission from alcohol use disorders. *Addiction, 101*(2), 212–222. <http://doi.org/10.1111/j.1360-0443.2006.01310.x>
- Morean, M. E., & Corbin, W. R. (2010). Subjective response to alcohol: A critical review of the literature. *Alcoholism: Clinical and Experimental Research, 34*(3), 385–395. <http://doi.org/10.1111/j.1530-0277.2009.01103.x>
- Morris, E. P., Stewart, S. H., & Ham, L. S. (2005). The relationship between social anxiety disorder and alcohol use disorders: A critical review. *Clinical Psychology Review, 25*(6), 734–760. <http://doi.org/10.1016/j.cpr.2005.05.004>
- Morse, R. M., & Flavin, D. K. (1992). The definition of alcoholism. *Alcohol and Alcoholism, 28*(6), 617–620. <http://doi.org/10.1001/jama.269.5.586>
- Mulder, R. T. (2002). Alcoholism and personality. *Australian and New Zealand Journal of Psychiatry, 36*, 44–52. <http://doi.org/10.1046/j.1440-1614.2002.00958.x>

- Neal, D. J., & Fromme, K. (2007). Event-level covariation of alcohol intoxication and behavioral risks during the first year of college. *Journal of Consulting and Clinical Psychology, 75*(2), 294–306. <http://doi.org/10.1037/0022-006X.75.2.294>
- Newlin, D. B., & Thomson, J. B. (1990). Alcohol challenge with sons of alcoholics: a critical review and analysis. *Psychological Bulletin, 108*(3), 383–402. <http://doi.org/10.1037/0033-2909.108.3.383>
- O'Brien, M. C., McCoy, T. P., Rhodes, S. D., Wagoner, A., & Wolfson, M. (2008). Caffeinated cocktails: Energy drink consumption, high-risk drinking, and alcohol-related consequences among college students. *Academic Emergency Medicine, 15*(5), 453–460. <http://doi.org/10.1111/j.1553-2712.2008.00085.x>
- O'Neill, S. E., Parra, G. R., & Sher, K. J. (2001). Clinical relevance of heavy drinking during the college years: cross-sectional and prospective perspectives. *Psychology of Addictive Behaviors, 15*(4), 350–359. <http://doi.org/10.1037/0893-164X.15.4.350>
- Ogborne, A. C. (2004). Theories of addiction and implications for counselling. In *Alcohol & Drug Problems: A Practical Guide for Counsellors* (pp. 3–21). CAMH: Toronto.
- Pedersen, E. R., & LaBrie, J. (2006). Drinking game participation among college students: Gender and ethnic implications. *Addictive Behaviors, 31*(11), 2105–2115. <http://doi.org/10.1016/j.addbeh.2006.02.003>
- Perkins, H. W. (2002). Surveying the damage: A review of research on consequences of alcohol misuse in college populations. *Journal of Studies on Alcohol. Supplement, (14)*, 91–100.
- Perkins, H. W. (2007). Misperceptions of peer drinking norms in Canada: Another look at the “reign of error” and its consequences among college students. *Addictive Behaviors, 32*(11), 2645–2656. <http://doi.org/10.1016/j.addbeh.2007.07.007>

- Perry, J. L., & Carroll, M. E. (2008). The role of impulsive behavior in drug abuse. *Psychopharmacology*, 200(1), 1–26. <http://doi.org/10.1007/s00213-008-1173-0>
- Piazza, P. V., & Le Moal, M. L. (1996). Pathophysiological basis of vulnerability to drug abuse: role of an interaction between stress, glucocorticoids, and dopaminergic neurons. *Annual Review of Pharmacology and Toxicology*, 36(1), 359–378. <http://doi.org/10.1146/annurev.pa.36.040196.002043>
- Piazza, P. V., & Le Moal, M. (1998). The role of stress in drug self-administration. *Trends in Pharmacological Sciences*, 19(2), 67–71. [http://doi.org/10.1016/S0165-6147\(97\)01115-2](http://doi.org/10.1016/S0165-6147(97)01115-2)
- Pope, J., Ionescu-Pioggia, M., & Pope, K. W. (2001). Drug use and life style among college undergraduates: A 30-year longitudinal study. *American Journal of Psychiatry*, 158(9), 1519–1521. <http://doi.org/10.1176/appi.ajp.158.9.1519>
- Price, S. R., Hilchey, C. A., Darredeau, C., Fulton, H. G., & Barrett, S. P. (2010). Energy drink co-administration is associated with increased reported alcohol ingestion. *Drug and Alcohol Review*, 29(3), 331–333. <http://doi.org/10.1111/j.1465-3362.2009.00163.x>
- Quinn, P. D., & Fromme, K. (2011a). Predictors and outcomes of variability in subjective alcohol intoxication among college students: An event-level analysis across 4 years. *Alcoholism: Clinical and Experimental Research*, 35(3), 484–495. <http://doi.org/10.1111/j.1530-0277.2010.01365.x>
- Quinn, P. D., & Fromme, K. (2011b). Subjective response to alcohol challenge: A quantitative review. *Alcoholism: Clinical and Experimental Research*, 35(10), 1759–1770. <http://doi.org/10.1111/j.1530-0277.2011.01521.x>

- Rehm, J., Ashley, M. J., Room, R., Single, E., Bondy, S., Ferrence, R., & Giesbrecht, N. (1996). On the emerging paradigm of drinking patterns and their social and health consequences. *Addiction*, *91*(11), 1615–1621. <http://doi.org/10.1080/09652149639178>
- Rehm, J., Mathers, C., Popova, S., Thavorncharoensap, M., Teerawattananon, Y., & Patra, J. (2009). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. *The Lancet*, *373*(9682), 2223–2233. [http://doi.org/10.1016/S0140-6736\(09\)60746-7](http://doi.org/10.1016/S0140-6736(09)60746-7)
- Rehm, J., Rehn, N., Room, R., Monteiro, M., Gmel, G., Jernigan, D., & Frick, U. (2003). The global distribution of average volume of alcohol consumption and patterns of drinking. *European Addiction Research*, *9*(4), 147–156. <http://doi.org/10.1159/000072221>
- Reid, A. E., Carey, K. B., Merrill, J. E., & Carey, M. P. (2015). Social network influences on initiation and maintenance of reduced drinking among college students. *Journal of Consulting and Clinical Psychology*, *83*(1), 36–44.
- Robotham, D., & Julian, C. (2006). Stress and the higher education student: A critical review of the literature. *Journal of Further & Higher Education*, *30*(2), 107–117. <http://doi.org/10.1080/03098770600617513>
- Roerecke, M., & Rehm, J. (2013). Alcohol use disorders and mortality: A systematic review and meta-analysis. *Addiction*, *108*(9), 1562–1578. <http://doi.org/10.1111/add.12231>
- Roh, S., Matsushita, S., Hara, S., Maesato, H., Matsui, T., Suzuki, G., & Higuchi, S. (2011). Role of GABRA2 in moderating subjective responses to alcohol. *Alcoholism: Clinical and Experimental Research*, *35*(3), 400–407. <http://doi.org/10.1111/j.1530-0277.2010.01357.x>
- Rose, A. K., Hobbs, M., & Drummond, C. (2013). Differentiating the contribution of pharmacological from alcohol expectancy effects to changes in subjective response and priming over successive

drinks. *Alcoholism: Clinical and Experimental Research*, 37(4), 687–695.

<http://doi.org/10.1111/acer.12022>

Rossow, I., & Romelsjö, A. (2006). The extent of the “prevention paradox” in alcohol problems as a function of population drinking patterns. *Addiction*, 101(1), 84–90. <http://doi.org/10.1111/j.1360-0443.2005.01294.x>

Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption--II. *Addiction*, 88(6), 791–804.

<http://doi.org/10.1111/j.1360-0443.1993.tb02093.x>

Schlauch, R. C., Waesche, M. C., Riccardi, C. J., Donohue, K. F., Blagg, C. O., Christensen, R. L., & Lang, A. R. (2010). A meta-analysis of the effectiveness of placebo manipulations in alcohol-challenge studies. *Psychology of Addictive Behaviors*, 24(2), 239–253.

<http://doi.org/10.1037/a0017709>

Schlotz, W., Kumsta, R., Layes, I., Entringer, S., Jones, A., & Wüst, S. (2008). Covariance between psychological and endocrine responses to pharmacological challenge and psychosocial stress: a question of timing. *Psychosomatic Medicine*, 70(7), 787–796.

<http://doi.org/10.1097/PSY.0b013e3181810658>

Schmidt, N. B., Buckner, J. D., & Keough, M. E. (2007). Anxiety sensitivity as a prospective predictor of alcohol use disorders. *Behavior Modification*, 31(2), 202–219.

<http://doi.org/10.1177/0145445506297019>

Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology*, *197*(1), 169–178.

<http://doi.org/10.1007/s00213-007-1023-5>

Schuckit, M. A. (1987). Biological vulnerability to alcoholism. *Journal of Consulting and Clinical Psychology*, *55*(3), 301–309. <http://doi.org/10.1037/0022-006X.55.3.301>

Schuckit, M. A. (2002). Vulnerability factors for alcoholism. In K. L. Davis, D. Charney, J.T. Coyle, and C. Nemeroff (Eds.), *Neuropsychopharmacology* (1399-1411). Philadelphia, Pennsylvania: Lippincott, Williams, & Wilkins.

Schuckit, M. A., Smith, T. L., Kalmijn, J., Trim, R. S., Cesario, E., Saunders, G., Sanchez, C., & Campbell, N. (2012). Comparison across two generations of prospective models of how the low level of response to alcohol affects alcohol outcomes. *Journal of Studies on Alcohol and Drugs*, *73*, 195–204. <http://dx.doi.org/10.15288/jsad.2012.73.195>

Schuckit, M. A. (2006). Comorbidity between substance use disorders and psychiatric conditions. *Addiction*, *101*, 76–88. <http://doi.org/10.1111/j.1360-0443.2006.01592.x>

Schuckit, M. A., Smith, T. L., Anderson, K. G., & Brown, S. A. (2004). Testing the level of response to alcohol: Social information processing model of alcoholism risk: A 20-year prospective study. *Alcoholism: Clinical*, *28*(12), 1890–1897. <http://doi.org/10.1097/01.ALC.0000148111.43332.A5>

Shaffer, H. J. (1985). The discovery of addiction: Levine and the philosophical foundations of drug abuse treatment. *Journal of Substance Abuse Treatment*, *2*(1), 41–57. [http://doi.org/10.1016/0740-5472\(85\)90021-2](http://doi.org/10.1016/0740-5472(85)90021-2)

Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology*, *1*, 493–523. <http://doi.org/10.1146/annurev.clinpsy.1.102803.144107>

- Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology, 103*(1), 92–102.
<http://dx.doi.org/10.1037/0021-843X.103.1.92>
- Sinha, R., Fox, H. C., Hong, K.I.A., Hansen, J., Tuit, K., & Kreek, M. J. (2011). Effects of adrenal sensitivity, stress- and cue-induced craving, and anxiety on subsequent alcohol relapse and treatment outcomes. *Archives of General Psychiatry, 68*(9), 942–952.
<http://doi.org/10.1001/archgenpsychiatry.2011.49>
- Slutske, W. S. (2005). Alcohol use disorders among US college students and their non-college-attending peers. *Archives of General Psychiatry, 62*(3), 321–327. [http://doi.org/10.1016/S0084-3717\(08\)70186-7](http://doi.org/10.1016/S0084-3717(08)70186-7)
- Slutske, W. S., Heath, A. C., Madden, P. A. F., Bucholz, K. K., Statham, D. J., & Martin, N. G. (2002). Personality and the genetic risk for alcohol dependence. *Journal of Abnormal Psychology, 111*(1), 124–133. <http://doi.org/10.1037/0021-843X.111.1.124>
- Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D. M. (2007). On the validity and utility of discriminating among impulsivity-like traits. *Assessment, 14*(2), 155–170.
<http://doi.org/10.1177/1073191106295527>
- Sobell, L. C., Brown, J., Leo, G. I., & Sobell, M. B. (1996). The reliability of the Alcohol Timeline Followback when administered by telephone and by computer. *Drug and Alcohol Dependence, 42*(1), 49–54. [http://doi.org/10.1016/0376-8716\(96\)01263-X](http://doi.org/10.1016/0376-8716(96)01263-X)
- Söderpalm, A., Nikolayev, L., & De Wit, H. (2003). Effects of stress on responses to methamphetamine in humans. *Psychopharmacology, 170*(2), 188–199. <http://doi.org/10.1007/s00213-003-1536-5>

- Söderpalm, A. H. V., & de Wit, H. (2002). Effects of stress and alcohol on subjective state in humans. *Alcoholism, Clinical and Experimental Research*, 26(6), 818–826.
<http://doi.org/http://dx.doi.org/10.1111/j.1530-0277.2002.tb02610.x>
- Sorocco, K. H., Lovallo, W. R., Vincent, A. S., & Collins, F. L. (2006). Blunted hypothalamic-pituitary-adrenocortical axis responsivity to stress in persons with a family history of alcoholism. *International Journal of Psychophysiology*, 59(3), 210–217.
<http://doi.org/10.1016/j.ijpsycho.2005.10.009>
- Soyka, M., Gorig, E., & Naber, D. (1992). Serum prolactin increase induced by ethanol: a dose-dependent effect not related to stress. *Psychoneuroendocrinology*, 16(5), 441–446.
[http://dx.doi.org/10.1016/0306-4530\(91\)90009-I](http://dx.doi.org/10.1016/0306-4530(91)90009-I)
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews*, 24(4), 417–463. [http://doi.org/10.1016/S0149-7634\(00\)00014-2](http://doi.org/10.1016/S0149-7634(00)00014-2)
- Stewart, S. H., Taylor, S., & Baker, J. M. (1997). Gender differences in dimensions of anxiety sensitivity. *Journal of Anxiety Disorders*, 11(2), 179–200. [http://dx.doi.org/10.1016/S0887-6185\(97\)00005-4](http://dx.doi.org/10.1016/S0887-6185(97)00005-4)
- Stewart, S. H., Zvolensky, M. J., & Eifert, G. H. (2001). Negative-reinforcement drinking motives mediate the relation between anxiety sensitivity and increased drinking behavior. *Personality and Individual Differences*, 31(2), 157–171. [http://doi.org/10.1016/S0191-8869\(00\)00213-0](http://doi.org/10.1016/S0191-8869(00)00213-0)
- Steyer, R., Schmitt, M., & Eid, M. (1999). Latent State - Trait Theory and research in personality and individual differences. *European Journal of Personality*, 13, 389–408.
[http://doi.org/10.1002/\(SICI\)1099-0984\(199909/10\)13:5<389::AID-PER361>3.0.CO;2-A](http://doi.org/10.1002/(SICI)1099-0984(199909/10)13:5<389::AID-PER361>3.0.CO;2-A)
- Swendsen, J., & Le Moal, M. (2011). Individual vulnerability to addiction. *Annals of the New York Academy of Sciences*, 1216(1), 73–85. <http://doi.org/10.1111/j.1749-6632.2010.05894.x>

- Testa, M., Fillmore, M. T., Norris, J., Abbey, A., Curtin, J. J., Leonard, K. E., & Hayman, L. W. (2006). Understanding alcohol expectancy effects: Revisiting the placebo condition. *Alcoholism: Clinical and Experimental Research*, *30*(2), 339–348. <http://doi.org/10.1111/j.1530-0277.2006.00039.x>
- Thomas, G. (2012). *Levels and patterns of alcohol use in Canada*. (Alcohol Price Policy Series: Report 1) Ottawa, ON: Canadian Centre on Substance Abuse
- Timberlake, D. S., Hopfer, C. J., Rhee, S. H., Friedman, N. P., Haberstick, B. C., Lessem, J. M., & Hewitt, J. K. (2007). College attendance and its effect on drinking behaviors in a longitudinal study of adolescents. *Alcoholism: Clinical and Experimental Research*, *31*(6), 1020–1030. <http://doi.org/10.1111/j.1530-0277.2007.00383.x>
- Uhart, M., & Wand, G. S. (2009). Stress, alcohol and drug interaction: An update of human research. *Addiction Biology*, *14*(1), 43–64. <http://doi.org/10.1111/j.1369-1600.2008.00131.x>
- Uhart, M., Weerts, E. M., McCaul, M. E., Guo, X., Yan, X., Kranzler, H. R., & Wand, G. S. (2012). GABRA2 markers moderate the subjective effects of alcohol. *Addiction Biology*, *18*(2), 357–369. <http://doi.org/10.1111/j.1369-1600.2012.00457.x>
- Vaughan, E. L., Corbin, W. R., & Fromme, K. (2009). Academic and social motives and drinking behavior. *Psychology of Addictive Behaviors*, *23*(4), 564–576. <http://doi.org/10.1037/a0017331>
- Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience and Biobehavioral Reviews*, *32*(4), 777–810. <http://doi.org/10.1016/j.neubiorev.2007.11.003>
- Vincent, K. B., Kasperski, S. J., Caldeira, K. M., Garnier, L. M., Pinchevsky, G. M., Grady, K. E. O., & Arria, A. M. (2013). Maintaining superior follow-up rates in a longitudinal study: Experiences from

the College Life Study. *International Journal of Multiple Research Approaches*, 6(1), 56-72.
<http://doi.org/10.5172/mra.2012.6.1.56>; PMC3255097

Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology*, 201(3), 315–324.
<http://doi.org/10.1007/s00213-008-1284-7>

Wechsler, H., Davenport, A., Dowdall, G., Moeykens, B., & Castillo, S. (1994). Health and behavioral consequences of binge drinking in college. *Journal of the American Medical Association*, 272(21), 1672–1677.

Wechsler, H., Dowdall, G. W., Davenport, A., & Castillo, S. (1995). Correlates of college student binge drinking. *American Journal of Public Health*, 85(7), 921–926. <http://doi.org/10.2105/AJPH.85.7.921>

Wechsler, H., Dowdall, G. W., Davenport, A., & Rimm, E. B. (1995). A gender-specific measure of binge drinking among college students. *American Journal of Public Health*, 85(7), 982–985.
<http://doi.org/10.2105/AJPH.85.7.982>

Wechsler, H., Lee, J. E., Kuo, M., Seibring, M., Nelson, T. F., & Lee, H. (2002). Trends in college binge drinking during a period of increased prevention efforts. Findings from 4 Harvard School of Public Health College Alcohol Study surveys: 1993-2001. *Journal of American College Health*, 50(5), 203–217. <http://doi.org/10.1080/07448480209595713>

Wechsler, H., Molnar, B. E., Davenport, A. E., & Baer, J. S. (1999). College alcohol use: A full or empty glass? *Journal of American College Health : J of ACH*, 47(6), 247–252.
<http://doi.org/10.1080/07448489909595655>

- White, H. R., Labouvie, E. W., & Papadaratsakis, V. (2005). Changes in substance use during the transition to adulthood: A comparison of college students and their noncollege age peers. *Journal of Drug Issues, 35*(2), 281–306. <http://doi.org/10.1177/002204260503500204>
- White, H. R., McMorris, B. J., Catalano, R. F., Fleming, C. B., Haggerty, K. P., & Abbott, R. D. (2006). Increases in alcohol and marijuana use during the transition out of high school into emerging adulthood: The effects of leaving home, going to college, and high school protective factors. *Journal of Studies on Alcohol, 67*(6), 810–822.
- White, W. L. (2000). Addiction as a disease: Birth of a concept. *Counselor, 1*(1), 46–51.
- Wood, P. K., Sher, K. J., & Rutledge, P. C. (2007). College student alcohol consumption, day of the week, and class schedule. *Alcoholism: Clinical and Experimental Research, 31*(7), 1195–1207. <http://doi.org/10.1111/j.1530-0277.2007.00402.x>
- Zamboanga, B. L., Schwartz, S. J., Ham, L. S., Borsari, B., & Van Tyne, K. (2010). Alcohol expectancies, pregameing, drinking games, and hazardous alcohol use in a multiethnic sample of college students. *Cognitive Therapy and Research, 34*(2), 124–133. <http://doi.org/10.1007/s10608-009-9234-1>
- Zimmermann, U., Spring, K., Kunz-Ebrecht, S. R., Uhr, M., Wittchen, H.-U., & Holsboer, F. (2004). Effect of ethanol on hypothalamic-pituitary-adrenal system response to psychosocial stress in sons of alcohol-dependent fathers. *Neuropsychopharmacology, 29*(6), 1156–1165. <http://doi.org/10.1038/sj.npp.1300395>

Chapter 2

Trait Impulsivity Prospectively Predicts Progression to Hazardous Drinking in Undergraduate Students

Abstract

Aims: Impulsivity, sensation seeking, and anxiety sensitivity have been associated with drinking behaviour and risk for alcohol use disorders. Our objective was to assess the extent to which these intrapersonal factors longitudinally predict change in drinking and alcohol-related problems in the transition from high school to university. A secondary aim was to examine whether these same variables relate to reported age of drinking onset. **Methods:** Participants ($n = 183$) completed an online survey during the summer prior to entering university (Time 1) and six months into the first year of university (Time 2). Hierarchical regression analyses assessed whether self-reported trait predictors (impulsivity, sensation seeking, and anxiety sensitivity) at Time 1 predicted change in drinking behaviour (use patterns, hazardous drinking, alcohol-related problems) between Time 1 and Time 2. Correlation analyses examined bivariate relationships between the trait predictors and reported age of drinking onset. **Results:** Impulsivity prospectively predicted an increase in hazardous drinking and, at a trend level, increased quantity of alcohol consumption per drinking day. Sensation seeking related to drinking behaviour cross-sectionally and significantly correlated with age of drinking onset. Anxiety sensitivity was not related to any measure of drinking behaviour. **Conclusions:** These data support the role of sensation seeking and impulsivity in the establishment and progression of drinking behaviour among young adults, and suggest that these factors are important targets for early intervention programs.

Introduction

Maladaptive drinking is a serious issue on university campuses across North America. Undergraduate students exhibit heavy drinking patterns, which exceed those of age-group peers who do not attend university (Slutske, 2005). Moreover, the majority (72%) of undergraduates report hazardous drinking, such as bingeing (Balodis, Potenza, & Olmstead, 2009). Increased drinking during this developmental period is related to risky behaviours, such as driving while intoxicated (Beck et al., 2010), and to negative outcomes, including university attrition (Martinez, Sher, & Wood, 2008). Importantly, high alcohol intake in university significantly predicts alcohol use disorders up to 10 years later (O'Neill, Parra & Sher, 2001; Zucker et al., 2006).

An important step in reducing alcohol misuse among undergraduate students is to identify factors that predict this behaviour. This includes behavioural traits, because these could serve as targets in prevention and early-intervention programs aimed at reducing risky drinking in this population. Among university students, two factors have been consistently associated with increased alcohol intake and alcohol-related problems: disinhibited personality traits and stress/anxiety (Baer, 2002).

In terms of disinhibited personality traits, impulsivity and sensation seeking (SS) are arguably the most commonly studied in the context of drinking behaviour. These factors are related under the broad concept of behavioural undercontrol, but form distinct constructs (Magid, Maclean & Colder, 2007). Impulsivity is commonly defined as the tendency for rapid, unplanned reactions to stimuli (internal or external) while lacking regard for the possible negative consequences (Moeller et al., 2001). Both trait and behavioral impulsivity have been implicated in the onset and maintenance of drug abuse (for reviews see Littlefield, Stevens, & Sher, 2013, de Wit, 2009; Perry & Carroll, 2008). For example, among healthy social drinkers, higher trait impulsivity is cross-sectionally associated with increased drinking and alcohol-related problems (Fox et al., 2010). In contrast, SS refers to the tendency to seek out and engage in novel experiences even if these include risk (Zuckerman, 1979). SS is positively associated with drinking behaviour and early onset of drinking (for review see Hittner & Swickert, 2006).

In contrast to disinhibition-based drinking, some individuals drink in response to negative emotionality. In particular, there is a well-established relationship between subjective stress/anxiety and alcohol use, alcoholism, and relapse (for reviews see Kushner, Abrams & Borchardt, 2000; Sinha, 2001, 2008). More recently, anxiety sensitivity (AS) has been identified as a cognitive vulnerability factor that amplifies anxiety, and moderates the relationship between anxiety and substance use (Dixon, Stevens, & Viana, 2014). AS is defined as the fear of anxious arousal-related sensations, related to beliefs that these sensations will have harmful or catastrophic consequences (Reiss, Peterson, Gursky, & McNally, 1986). High AS is related to greater alcohol consumption (Stewart, Peterson, & Pihl, 1995; Stewart, Zvolensky, & Eifert, 2001) and predicts future development of alcohol use disorders (Schmidt, Buckner, & Keough, 2007).

The vast majority of studies examining predictors of alcohol use have been cross-sectional (for reviews see Baer, 2002; Hittner & Swickert, 2006). However, the number of longitudinal studies is increasing, in line with the view that examination of change in drinking over time is particularly useful in estimating risk for future AUD (Chassin, Flora, & King, 2004). Moreover, alcohol use follows a typical developmental trajectory across the lifespan, wherein drinking normally increases steadily throughout adolescence, peaks in young adulthood (Chassin, Flora, & King, 2004), and begins to decline in the mid-twenties in a process known as ‘maturing out’ (Lee, Chassin, & Villalta, 2013). Interestingly, the correlation between heavy drinking and alcohol-related problems is strongest on first entering university (O’Neill, Parra, & Sher, 2001). This suggests that analyses of drinking behaviour should be approached from a developmental perspective with a focus on critical transitions, such as the move from high school to university, because this period is associated with increased alcohol use (Baer, Kivlahan, & Marlatt, 1995).

Large-scale survey studies (e.g., Monitoring the Future Study, Pope, Ionescu-Pioggia, & Pope, 2001; College Life Study, Vincent et al., 2013) that collect longitudinal data on U.S. university students’ drinking behaviour have focused, most heavily, on epidemiological descriptions of drinking patterns, and

demographic predictors of substance use. More recently, these research programmes have expanded to include single trait predictors, such as sensation seeking (Kaynak et al., 2013). Likewise, some individual studies have prospectively examined the role of intrapersonal factors in the progression of undergraduates' substance use. For example, Cyders and colleagues (2009) tested impulsivity and sensation seeking as predictors of change in drinking across the first year of undergraduate studies, specifically between the beginning of the fall and the end of the spring. Despite its merits, this study is limited by its use of basic measures of alcohol intake (simple quantity-frequency), the absence of internalizing-related predictors (e.g., anxiety sensitivity), and the fact that the initial measurement point likely captures the isolated spike in drinking behaviour that coincides with arrival at university (e.g., "Frosh Week"). To our knowledge, no study has examined the variables of interest in our study, namely sensation seeking, impulsivity, and anxiety sensitivity, during the critical transition period between high school and undergraduate studies.

The present study examined the unique contributions of particular trait factors in predicting change in thoroughly-characterized drinking behaviour during the transition from high school to university. We hypothesized that higher impulsivity, sensation seeking, and anxiety sensitivity at the first sampling point, prior to university, would predict increased alcohol use and alcohol-related problems during the first year of university. A secondary goal of this study was to assess whether these predictors relate to the individuals' reported age of drinking onset.

Methods

Participants and Procedure.

The experimental protocol was approved by the Graduate Research Ethics Board at Queen's University. All incoming undergraduates in 2012 ($N = 4,079$; Queen's University Senate, 2012) were contacted via email in August, following graduation from high school and before entering their first year of university. Participants who completed the survey in August (Time 1; $n = 636$) were emailed to request that they complete an additional questionnaire the following March (Time 2), six months into their first

undergraduate year. A reminder email was sent once per sampling time. One hundred-eighty three individuals participated in both Time 1 and Time 2 surveys. Numerical identifiers were assigned to each participant and used to match participants' responses at Time 1 and Time 2.

At each time point, participants provided informed consent and completed online questionnaires (detailed below) related to substance use (types of substances, patterns, substance-related problems), trait/behavioural factors (impulsivity, sensation seeking, anxiety sensitivity), and demographic information, which took approximately 30 minutes to complete. Participants were asked to report on the three months prior to each sampling period, which, for some measures, was a modification on the original instructions. As compensation at each time point, individuals were offered entry into a draw to win a \$25 gift certificate from a popular chain coffee shop or bookstore.

Substance Use Measures.

Alcohol Timeline Follow-Back.

Participants completed the alcohol Timeline Follow-Back (TLFB; Sobell & Sobell, 1992), a widely-used retrospective self-report measure of drinking behaviour, in which participants are asked to indicate how many standard drinks they consumed on each day of a specified time period (Sobell & Sobell, 2008). A standard drink was defined as 12 oz. beer, 5 oz. wine, 1.5 oz. hard liquor, or 3 oz. fortified wine. To increase accuracy of reporting, the TLFB employs a calendar format and encourages recall of important dates and events. The TLFB provides an accurate retrospective estimate of overall drinking levels when compared against real-time recordings of alcohol use (Carney, Tennen, Affleck, del Boca, & Kranzler, 1998). This questionnaire is preferable to aggregate measures of alcohol consumption that tend to be insensitive to variations in drinking patterns, such as heavy drinking occasions (del Boca & Darkes, 2003). Computerized self-administration of the TLFB is as reliable as administration of the measure by a trained interviewer (Sobell, Brown, Leo, & Sobell, 1996). The main summary variables from the TLFB were Drinking Frequency, the average number of drinking days per week; Drinking Quantity, the average number of alcohol beverages per drinking day; Total Intake, the total number of

alcoholic beverages over sampling period; and Maximum Quantity, the maximum number of alcoholic beverages consumed on a single drinking day.

Rutgers Alcohol Problem Index.

The Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) is a measure of alcohol-related negative consequences that is specifically designed for young adults. This 23-item questionnaire lists potential problems associated with alcohol use and asks respondents to rate each item on a scale from 0 (“never”) to 4 (“more than 10 times”). Total scores on the RAPI demonstrate good reliability in identifying alcohol-related problems among college students (e.g., Cronbach’s $\alpha = .86$; Martens et al., 2007).

Alcohol Use Disorders Identification Test.

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a measure of harmful or hazardous drinking behavior. The 10 items on this questionnaire are scored on a scale from 0 (“never”) to 4 (“four or more times per week”) and assess three main factors: alcohol consumption, drinking behaviour/ dependence, and alcohol-related consequences. The AUDIT is a unique alcohol screening measure, in that its focus is on detection of risky drinking as opposed to alcohol dependence (Kokotailo et al., 2004). The AUDIT demonstrates good internal consistency (Cronbach’s $\alpha = 0.81$) and predictive validity for alcohol use disorders among college students (Fleming, Barry, & MacDonald, 1991).

Alcohol, Smoking and Substance Involvement Screening Test.

The Alcohol, Smoking and Substance Involvement Screening Test version 3.0 (ASSIST; World Health Organization, 2002) is an 8-question measure that assesses drug use and associated problems for each of ten drug categories: tobacco, alcohol, cannabis, cocaine, stimulants, inhalants, sedatives/hypnotics, hallucinogens, opioids, and “other drugs”. The ASSIST demonstrates high concurrent validity with other alcohol risk measures, such as the AUDIT ($r = 0.82$; Humeniuk et al,

2008). The test-retest reliability of the ASSIST ranges from good to excellent for periods of up to three weeks (average kappas for drug classes = 0.61-0.78; World Health Organization, 2002).

Trait and Behaviour Measures.

Barratt Impulsiveness Scale.

The Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) is self-report measure of behavioural or trait impulsivity, which has been used extensively in clinical and research settings for over 50 years (Stanford et al., 2009). The questionnaire consists of 30 items that are rated on a 4-point scale (rarely/never, occasionally, often or almost always). The BIS exhibits strong psychometric properties, including high internal consistency (Cronbach's $\alpha = 0.83$) and one-month test-retest reliability (Spearman's $\rho = 0.83$; Stanford et al., 2009). The current study examined total BIS score, which provides a valid global measure of impulsivity in undergraduate populations (Patton et al., 1995).

Anxiety Sensitivity Index.

The Anxiety Sensitivity Index (ASI; Peterson & Heilbronner, 1987) is a 16-item questionnaire about individuals' negative beliefs about the physical and social consequences of anxiety symptoms. Individuals rank each item on a 5-point scale: very little, a little, some, much, or very much. Our measure of anxiety sensitivity was the ASI Total score, which has high internal consistency (Cronbach's $\alpha = 0.88$), and exhibits good two-week test-retest reliability (Pearson product-moment correlation = 0.75; Reiss, Peterson, Gursky, & McNally, 1986).

Zuckerman-Kuhlman Personality Questionnaire.

Sensation seeking was assessed using the Impulsive Sensation Seeking subscale (ImpSS) of the Zuckerman-Kuhlman Personality Questionnaire (Zuckerman & Link, 1968). This ImpSS involves 19 true-false questions that provide a total score, as well as Impulsivity and Sensation Seeking subscales. The latter subscale was our particular variable of interest. Test-retest reliability (for up to four weeks) of the ImpSS has been shown to be adequate (Pearson product-moment correlation = 0.80; Zuckerman,

2002). The ImpSS subscales exhibit moderate to high internal consistency among undergraduates (Cronbach's $\alpha = 0.67-0.79$; Buelow & Suhr, 2013).

Alcohol Use Onset.

As part of the demographic questionnaire, participants answered two items regarding onset of alcohol use – “At what age did you have your first alcoholic drink?” and “At what age did you begin to drink alcohol recreationally (i.e., for social participation and/or pleasure)?” – to which they provided a numeric response. Single-item measures of self-reported drinking onset are commonly used in alcohol use research, including large-scale studies such as the Ontario Health Survey (de Wit, Adlaff, Offord, & Ogborne, 2000) and the U.S. National Epidemiologic Survey on Alcohol and Related Conditions (Hingson, Heeren & Winter, 2006). Self-reported age of alcohol use onset exhibits moderate one-year test-retest reliability (Johnson & Mott, 2001; Parra, O’Neill, & Sher, 2003).

Statistical Analyses

Data were analyzed using SPSS version 22.0. For cases with missing data within a particular measure, summary scores were pro-rated using an average of the existing responses. If respondents were missing more than 10-15% of items within a scale, their data were not included in the analyses for that particular measure. The dependent measures were analyzed as described below. Initial inspection of skewness, kurtosis, histograms and normality plots revealed that most variables were non-normal. Thus, non-parametric bootstrapped analyses, which do not require assumptions (e.g., normality) to be met, were employed for all analyses. Statistical significance was set at a probability of $p < .05$. Correction for multiple comparisons (Bonferroni) was employed when independent variables underwent multiple comparisons against the same dependent variable, which is associated with inflation of Type I error rate.

Participant Demographics.

A power analysis was conducted using G*Power 3.1.2 software (Faul, Erdfeder, Lang, & Buchner, 2007) for a multiple regression with the following specifications: small-medium effect size (Cohen's $f^2 = 0.07$), alpha of .05, beta of .20, and four predictors. Attrition analyses were conducted to

assess differences in our variables of interest (predictors, criterion measures, demographic variables) at Time 1 between individuals who participated at Time 2 ($n = 183$) and those who did not ($n = 453$). Group differences were examined using bootstrapped (10, 000 re-samples) independent samples t-tests and Chi-square (χ^2) tests for continuous and categorical data, respectively. Basic descriptive statistics (mean, standard deviation, sample size) were used to describe the participant demographics.

Measure Descriptives.

Initial descriptive statistics (sample size, mean, standard deviation, median, skewness, kurtosis) were analyzed for all variables of interest. Reliability analysis was conducted to verify that the predictor and criterion measures exhibited adequate internal consistency. Reliability analysis was not completed for the TLFB, in which participants simply report usage patterns (as opposed to responding to question items). Bootstrapped (10,000 re-samples) correlation (Pearson's r) assessed correspondence between measures of drinking onset (i.e., age of first alcoholic beverage and age of recreational drinking onset).

Relationship between Predictors and Criterion Variables.

To assess whether criterion variables changed between Time 1 and Time 2, bootstrapped (10,000 re-samples) paired samples t-tests were used. Bootstrapped (10,000 re-samples) correlations (Pearson's r) were conducted to examine bivariate relationships between all variables of interest, in order to assess for multicollinearity among predictor variables and to see whether predictors are significantly correlated with criterion variables. Bootstrapped (10,000 re-samples) hierarchical linear regressions were used to assess prediction of drinking behaviour (i.e., criterion variables) at Time 2. To control for drinking behaviour at Time 1, these variables were entered as the first step in the regression, followed by the predictor variables.

Relationship between Predictors and Alcohol Use Onset.

Bootstrapped (10,000 re-samples) correlations (Pearson's r) examined the bivariate relationship between the predictors (i.e., BIS, SS, and ASI) and the two self-report measures of drinking onset: age at first alcoholic drink and age of recreational drinking onset.

Results

Participant Demographics.

The power analysis revealed that a sample size of 176 was required for the most complex planned analysis (i.e., multiple regression) with a small-medium effect size (Cohen's $f^2 = 0.07$). The participant sample was comprised of 183 individuals (28 men, 141 women, 14 unreported) who participated in both Time 1 and Time 2 surveys, which is in keeping with the required sample size per the power analysis. Attrition analysis, comparing individuals who participated at Time 2 ($n = 183$) and those who did not ($n = 453$), revealed no group differences at Time 1 in any of the variables of interest, all p -values $> .05$. At Time 1, the average age of the sample was 17.80 ($SD = 0.60$, Range: 15-19; $n = 166$). Additional demographic information (living arrangement and relationship status) of the participants at each sampling point can be found in Appendix A. In the 2012 academic year (i.e., the year in which the study sample entered university), 68% of first-year undergraduate students were female (Queens's Senate, 2012); with 77% women, our study sample overestimates to some degree the gender proportion of the population.

Measure Descriptives.

Table 2-1 presents descriptive statistics for the variables of interest at Time 1 and Time 2. As mentioned in the Methods section, nearly all variables of interest exhibited significant non-normality. Coefficient alpha reliabilities for all variables of interest exceeded the conventional cut-off of .70, indicating that reliability was adequate to justify inclusion in subsequent analyses. The drinking onset variables (reported age of first alcoholic beverage, and reported age of recreational drinking onset) were significantly correlated, $r = .51$, $p < .001$.

Table 2-1 Descriptive statistics for predictor and criterion measures

Measure	<i>n</i>	M (SD)	Median	Range	Skewness (SE)	Kurtosis (SE)	α
<i>Predictors (Time 1)</i>							
Drinking Frequency	180	0.41 (0.54)	0.20	0 – 2.93	1.63 (0.81)	2.79 (0.36)	-
Drinking Quantity	180	2.76 (2.85)	2.13	0 – 15.52	1.07 (0.18)	1.51 (0.36)	-
Total Intake	180	29.82 (47.60)	8.00	0 – 326	2.66 (0.18)	9.78 (0.36)	-
Maximum Quantity	180	4.80 (5.36)	4.00	0 – 28.00	1.29 (0.18)	2.00 (0.36)	-
ASSIST Total	182	8.75 (15.66)	4.00	0 – 124	3.93 (0.18)	20.30 (0.34)	.92
RAPI	180	1.90 (4.53)	0.00	0 – 48	6.61 (0.36)	60.88 (0.36)	.90
AUDIT	180	0.63 (1.49)	0.00	0 – 12	3.89 (0.81)	21.03 (0.36)	.81
BIS	175	54.75 (8.31)	54.00	34.14 – 80	0.22 (0.18)	0.09(0.36)	.77
ASI	170	22.82 (11.13)	22.00	0 – 53	0.47 (0.19)	-0.09 (0.37)	.88
SS	170	5.32 (2.73)	5.00	0 – 11	-0.09 (0.19)	-0.69 (0.37)	.74
<i>Criterion Measures (Time 2)</i>							
Drinking Frequency	183	0.56 (0.67)	0.33	0 – 4.33	1.80 (0.18)	4.96 (0.36)	-
Drinking Quantity	183	3.12 (2.81)	3.00	0 – 13.55	0.87 (0.18)	0.78 (0.36)	-
Total Intake	183	42.56 (64.07)	19.00	0 – 393	2.44 (0.18)	7.21(0.36)	-
Maximum Quantity	183	5.61 (5.64)	5.00	0 – 32	1.33 (0.18)	2.61 (0.36)	-
ASSIST	183	9.81 (16.16)	5.00	0 – 139	4.22	25.61	.92
RAPI	176	2.96 (5.34)	1.00	0 – 41	3.98	21.61	.90
AUDIT	176	4.73 (4.70)	4.00	0 – 20	0.82	-0.20	.82

Note. Due to missing data, some variables include fewer participants than the total sample ($n = 183$). n = sample size, M = mean, SD = standard deviation, Drinking Frequency = average number of drinking days per week, Drinking Quantity = average number of alcohol beverages consumed per drinking day, Total Intake = total number of alcoholic beverages consumed over the sampling period, Maximum Quantity = maximum number of alcoholic beverages consumed on a single drinking day, ASSIST = Alcohol, Smoking, and Substance Involvement Screening Test total score, RAPI = Rutgers Alcohol Problems Index total score, AUDIT = Alcohol Use Disorders Identification Test total score, BIS = Barratt Impulsiveness Scale total score, ASI = Anxiety Sensitivity Index total score, SS = Sensation Seeking subscale score.

Relationship between Predictors and Criterion Variables.

Bootstrapped paired samples t-tests were used to assess significant differences between individuals' scores at Time 1 and Time 2 on each measure (see Table 2-2). There were significant increases in most criterion measures, namely drinking frequency and total intake, as well as RAPI and AUDIT total scores, $p < .05$. The change in maximum quantity score between Time 1 and Time 2 reached a trend level, $p = .06$. The difference between Time 1 and Time 2 ASSIST Total scores was non-significant, $p > .05$.

Table 2-2. Change in criterion measures between Time 1 and Time 2

Criterion Measures	Time 1		Time 2		$t(df)$	p	d
	n	$M (SD)$	n	$M (SD)$			
Drinking Frequency	180	0.41 (0.54)	183	0.56 (0.67)	3.68 (179)	<.000**	0.25
Drinking Quantity	180	2.76 (2.85)	183	3.12 (2.81)	1.88 (179)	.060	0.13
Total Intake	180	29.82 (47.60)	183	42.56 (64.07)	3.26 (179)	.001**	0.22
Maximum Quantity	180	4.80 (5.35)	183	5.61 (5.64)	2.21 (179)	.029*	0.15
ASSIST Total	182	8.75 (15.66)	183	9.81 (16.16)	1.00 (181)	.319	0.07
RAPI Total	180	1.90 (4.53)	176	2.96 (5.34)	3.24 (172)	.001**	0.22
AUDIT Total	180	3.21 (4.05)	176	4.73 (4.70)	6.02 (172)	<.001**	0.35

Note. Due to missing data, some variables include fewer participants than the total sample ($n = 183$). n = sample size, M = mean, SD = standard deviation, t = Bootstrapped (10,000 re-samples) paired samples t-test, p = p -value, d = Cohen's d , Drinking Frequency = average number of drinking days per week, Drinking Quantity = average number of alcohol beverages per drinking day, Total Intake = total number of alcoholic beverages over sampling period, Heavy Drinking Frequency = number of drinking days on which females and males consumed greater than 4 and 5 alcoholic beverages, respectively, Maximum Quantity = maximum number of alcoholic beverages consumed on a single drinking day, ASSIST = Alcohol, Smoking, and Substance Involvement Screening Test, RAPI = Rutgers Alcohol Problems Index, AUDIT = Alcohol Use Disorders Identification Test. * = significance at $p < .05$ (two-tailed), ** = significance at $p < .05$ (two-tailed)

Bivariate correlations (bootstrapped Pearson's correlations with 10,000 re-samples) between all predictor and criterion variables are presented in Table 2-3. The BIS and SS exhibited significant correlations with all criterion variables, with the exception of a non-significant relationship between SS at

Time 1 and RAPI at Time 2, $r = .15, p > .05$. In contrast, the ASI was not significantly correlated with any of the criterion variables. Inter-correlations between the predictor variables ranged from .00-.54, suggesting that multicollinearity was not an issue.

Table 2-4 summarizes the results of the hierarchical linear regressions. Together the predictors explained a significant proportion of AUDIT scores, $R^2_{\text{change}} = .04, F_{\text{change}}(3, 157) = 4.17, p < .01$. Specifically, the overall model accounted for 4% of the variance in AUDIT scores. BIS was a significant predictor of AUDIT scores, $\beta = .17, t(157) = 2.68, p < .01$, whereas ASI and SS were not, p -values $> .05$. The overall model also significantly predicted ASSIST scores, $R^2_{\text{change}} = .04, F_{\text{change}}(3, 163) = 3.49, p < .05$; however, upon confirmation with bootstrapping, none of the variables remained significant as predictors. A trend emerged when the predictor model was regressed against drinking quantity, $R^2_{\text{change}} = .02, F_{\text{change}}(3, 161) = 2.82, p = .081$. ASI and SS did not emerge as significant predictors, $p > .05$, whereas BIS was a significant predictor of drinking quantity at a trend level, $\beta = .14, t(161) = 2.42, p = .054$.

Table 2-3. Bootstrapped correlations (Pearson's *r*) between predictor and criterion variables (*n* = 160)

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
<u>Predictors</u>																	
<u>(Time 1)</u>																	
1. Drinking Frequency	-																
2. Drinking Quantity	.57**	-															
3. Total Intake	.83**	.78**	-														
4. Maximum Quantity	.67**	.90**	.79**	-													
5. ASSIST	.64**	.61**	.72**	.69**	-												
6. RAPI	.62**	.45**	.57**	.47**	.58**	-											
7. AUDIT	.77**	.79**	.84**	.74**	.67**	.66**	-										
8. BIS	.19*	.29**	.27**	.24**	.24**	.17*	.31**	-									
9. ASI	-.03	-.13	-.06	-.11	-.06	.04	-.05	.02	-								
10. SS	.37**	.34**	.39**	.33**	.33**	.30**	.41**	.54**	-.00								
<u>Criteria</u>																	
<u>(Time 2)</u>																	
11. Drinking Frequency	.71**	.54**	.63**	.62**	.62**	.54**	.63**	.24**	-.06	.31**	-						
12. Drinking	.50**	.66**	.54**	.65**	.50**	.48**	.63**	.32**	-.13	.33**	.55**	-					

Quantity																	
13. Total Intake	.65**	.59**	.66**	.64**	.59**	.61**	.66**	.26**	-.06	.29**	.88**	.72**	-				
14. Maximum	.54**	.66**	.57**	.75**	.61**	.52**	.62**	.26**	-.13	.30**	.65**	.90**	.77**	-			
Quantity																	
15. ASSIST	.51**	.40**	.47**	.43**	.68**	.52**	.54**	.28**	-.00	.43**	.60**	.46**	.51**	.53**	-		
16. RAPI	.35**	.27**	.27**	.29**	.28**	.48**	.40**	.20*	.04	.15	.42**	.41**	.42**	.40**	.40**	-	
17. AUDIT	.58**	.64**	.57**	.63**	.55**	.53**	.75**	.38**	-.06	.37**	.72**	.78**	.71**	.76**	.64**	.64**	-

Note. Due to missing data, some variables include fewer participants than the total sample ($n = 183$). Drinking Quantity = average number of alcohol beverages per drinking day, Total Intake = total number of alcoholic beverages over sampling period, Maximum Quantity = maximum number of alcoholic beverages consumed on a single drinking day, ASSIST = Alcohol, Smoking, and Substance Involvement Screening Test total score, RAPI = Rutgers Alcohol Problems Index total score, AUDIT = Alcohol Use Disorders Identification Test total score, BIS = Barratt Impulsiveness Scale total score, ASI = Anxiety Sensitivity Index, SS = Sensation Seeking Scale subscale score. * = significance at $p < .05$ (two-tailed), ** = significance at $p < .01$ (two-tailed). Six month test-retest correlations between Time 1 and Time 2 are in bold.

Table 2-4. Hierarchical regression analysis predicting substance use behaviour

Criterion Measure (Time 2)	Steps	R ² Change	<i>p</i>	β (SE)	<i>p</i>	<i>f</i> ²
Drinking Frequency	1. Time 1 Drinking Frequency	.43**	.000	.64 (.07)**	.000	
	2. Predictors (Time 1)	.01	.284			0.02
	BIS			.12 (.01)	.099	
	ASI			-.04 (.01)	.464	
	SS			-.02 (.02)	.811	
Drinking Quantity	1. Time 1 Drinking Quantity	.40**	.000	.57 (.06)**	.000	
	2. Predictors (Time 1)	.02	.081			0.04
	BIS			.14 (.02)	.054	
	ASI			-.06 (.01)	.334	
	SS			.03 (.08)	.714	
Total Intake	1. Time 1 Total Intake	.39**	.000	.61 (.08)**	.000	
	2. Predictors (Time 1)	.01	.544			0.01
	BIS			.10 (.57)	.200	
	ASI			-.04 (.35)	.524	
	SS			-.03 (1.83)	.710	
Maximum Quantity	1. Time 1 Maximum Quantity	.51**	.000	.68 (.06)**	.000	
	2. Predictors (Time 1)	.01	.334			0.02
	BIS			.09 (.04)	.163	
	ASI			-.05 (.03)	.343	

	SS			-.00 (.14)	.977	
ASSIST Total	1. Time 1 ASSIST Total	.34**	.000	.51 (.06)**	.000	
	2. Predictors (Time 1)	.04*	.017			0.06
	BIS			.02 (.12)	.765	
	ASI			.01 (.07)	.909	
	SS			.20 (.38) [†]	.011	
RAPI Total	1. Time 1 RAPI Total	.20**	.000	.44 (.12)**	.000	
	2. Predictors (Time 1)	.02	.189			0.03
	BIS			.17(.05)	.040	
	ASI			.00 (.03)	>.999	
	SS			-.48 (.15)	.632	
AUDIT Total	1. Time 1 AUDIT Total	.50**	.000	.65 (.07)	.000	
	2. Predictors (Time 1)	.04**	.007			0.08
	BIS			.17 (.04)**	.008	
	ASI			-.04 (.02)	.414	
	SS			.04 (.12)	.600	

Note. Drinking Quantity = average number of alcohol beverages per drinking day, Total Intake = total number of alcoholic beverages over sampling period, Maximum Quantity = maximum number of alcoholic beverages consumed on a single drinking day, ASSIST = Alcohol, Smoking, and Substance Involvement Screening Test, RAPI = Rutgers Alcohol Problems Index, AUDIT = Alcohol Use Disorders Identification Test, BIS = Barratt Impulsiveness Scale, ASI = Anxiety Sensitivity Index, SSS-SS = Sensation Seeking Scale – Sensation Seeking. All results were confirmed using non-parametric bootstrapping with 10,000 samples. * = significance at $p < .05$ (two-tailed), ** = significance at $p < .01$ (two-tailed), [†] No longer significant upon verification with bootstrapping.

Relationship between Predictors and Alcohol Use Onset.

Reported age of first alcoholic drink and age of recreational drinking (i.e., for pleasure and/or social participation) onset were, on average, 14.77 years ($SD= 2.45$, Range: 4-18; $n = 128$) and 16.19 years ($SD= 1.34$, Range: 13-19; $n = 99$), respectively. There was a weak, but marginally significant, negative relationship between SS and the age of recreational drinking onset (i.e., for pleasure or social participation), $r = -.24$, $p = .017$ (with Bonferroni correction for multiple comparisons; $\alpha = .05/3 = .017$). The relationship between SS and the age at first alcohol drink was not significant, $r = -.18$, $p > .017$. There was no significant relationship between BIS or ASI with either age at first alcoholic drink or age of recreational drinking onset, p -values $> .017$.



Figure 2-1. Sensation seeking correlated against age of recreational drinking onset (years).
Note. SS = Sensation Seeking subscale score.

Discussion

The current study is the first, to our knowledge, to examine the contribution of impulsivity, sensation seeking, and anxiety sensitivity to the progression of drinking behaviour during the critical developmental transition period between high school and undergraduate studies. In this study, individual differences in impulsivity predicted increases in both hazardous drinking and the quantity of alcohol consumed per drinking day during the first few months of undergraduate studies, although the latter finding was at a trend level. This supports the well-established relationship between impulsivity and

pathological alcohol use, including the finding that alcoholics exhibit higher trait impulsivity compared to non-alcoholics (de Wit, 2009). Moreover, as with behavioural measures (Goudriaan, Grekin, & Sher, 2011), our assessment of trait impulsivity was more predictive of hazardous drinking than of either the frequency or quantity of alcoholic consumption in this population.

In contrast to impulsivity, sensation seeking did not significantly predict changes in drinking behaviour, despite being positively correlated with all drinking measures at each time point. Specifically, sensation seeking correlated with Time 1 and Time 2 drinking behaviour, but did not predict changes in drinking behaviour between these two time points. That is, once the variance accounted for by Time 1 was removed (as was the case in the regression analysis), sensation seeking was no longer significantly related to drinking behaviour at Time 2. Statistically, this raises the possibility that the correlation between sensation seeking and Time 2 drinking behaviour was confounded by the correlation between sensation seeking and Time 1 drinking behaviour, highlighting the importance of sensation seeking in early patterns of drinking behaviour. This notion was corroborated by the fact that sensation seeking, but not impulsivity, was negatively correlated with recreational drinking onset. Taken together, these findings highlight an interesting dissociation, in that sensation seeking was related to initial alcohol use (i.e., drug approach) whereas impulsivity predicted the progression of hazardous drinking.

Impulsivity and sensation seeking relate to the general notion of behavioural disinhibition, and have a similar influence on drinking behaviour in pathological populations, in that both predict early onset alcoholism and greater symptom severity (Dom, Julstijn, & Sabbe, 2006). The distinction between these two factors (Magid, MacLean, & Colder, 2007), however, may become apparent in non-pathological alcohol use, as has been previously hypothesized (Dom, Julstijn, & Sabbe, 2006). This includes the onset of regular alcohol use and early drinking patterns, as measured in our study. Specifically, sensation seeking involves the drive for novel experiences, and is strongly implicated in initial drug-seeking behaviour, both in animal models and human studies (Bardo et al., 1996). Impulsivity, on the other hand, includes behavioural dyscontrol and a diminished consideration of future consequences, which could

plausibly contribute to a progression into hazardous patterns of drinking. This behavioural dissociation is supported by underlying neural processes: the subcortical socioemotional system and the cortical cognitive control system, which mediate sensation seeking and impulsivity, respectively (Harden & Tucker-Drob, 2011; Steinberg, 2008). In other words, sensation seeking reflects heightened responsiveness of limbic structures to novel stimuli, whereas impulsivity is associated with diminished behavioural inhibition by the prefrontal cortex (Harden & Tucker-Drob, 2011; Steinberg, 2008). These neurobehavioural processes could differentially influence initial alcohol use and the progression to hazardous drinking, explaining our finding that sensation seeking relates to initial use but not progression of drinking, whereas impulsivity shows the opposite pattern.

Unlike impulsivity and sensation seeking, anxiety sensitivity was not associated with drinking behaviour in the current sample, either cross-sectionally or longitudinally. This contradicts some findings (Stewart, Zvolensky & Eifert, 2001; Stewart, Peterson, Pihl, 1995), but is in line with other evidence that anxiety sensitivity is unrelated to drinking behaviour in this population (Novak et al., 2003; Stewart, Karp, Pihl, & Peterson, 1997). In fact, the latter studies demonstrated that anxiety sensitivity, although not predicting levels of alcohol consumption, was predictive of coping motives for drinking (Novak et al., 2003; Stewart, Karp, Pihl & Peterson, 1997). Thus, anxiety sensitivity likely has a greater, or more direct, influence on why individuals drink, as opposed to their patterns of drinking. Additionally, the relationship between anxiety sensitivity and intake may be more pronounced in pathological anxiety and/or alcohol use (Cox et al., 1993; Schmidt, Buckner, & Keough, 2007).

The findings of the current study should be considered in light of potential limitations. Firstly, the initial response rate for the survey at Time 1 (15%) fell below the commonly cited acceptable threshold of 60% (Dillman, 1991). This may relate to a number of factors, such as poor student involvement in university-affiliated activities prior to beginning their studies, or reticence to participate in research that is disseminated by the university registrar's office. The impact of nonresponse bias (i.e., the extent to which non-responders differ from Time 1 participants) – arguably, a more important consideration than response

rates per se (Johnson & Wislar, 2012) – is, unfortunately, difficult to ascertain in the absence of ‘complete coverage’ (i.e., whole-population) data. Although the attrition rate between Time 1 and Time 2 was also high (71%), attrition analyses did not reveal significant group differences on any variables of interest, including predictor, criterion, and demographic variables.

Another potential limitation in the current study relates to oversampling of women. As a result, we may not have detected effects that are driven by male participants due to reduced power in this group. For example, because the relationship between alcohol use and sensation seeking is more pronounced in males in some studies (e.g., Hittner & Swickert, 2006), our null findings with respect to sensation seeking and the progression in drinking behaviour may be due to the low number of males. Additionally, our study employed a broad operationalization of impulsivity (Total Score on the BIS), however, this construct can also be measured by lower order facets such as positive urgency and lack of perseverance (Littlefield et al., 2013), as well as behavioural measures (King, Patock-Peckham, Dager, Thimm, & Gates, 2014). These different metrics of impulsivity may exhibit different relationships with the intrapersonal factors that were measured in the current study. Future research examining impulsivity, sensation seeking, and anxiety sensitivity and their relationship with drinking behaviour should maintain a longitudinal design, ideally examining patterns of drinking prior to, during, and following post-secondary education. Furthermore, as previous research has supported the notion that university students exhibit drinking patterns that are distinct from non-university attending peers (e.g., Slutske, 2005), comparing our results to data from age-matched controls who do not attend a post-secondary institution would provide insight into the role of social and environmental factors in the progression of maladaptive drinking behaviours.

Conclusions

In sum, our findings demonstrate that sensation seeking and impulsivity, respectively, are significantly related to initial drinking onset and increased hazardous drinking during the first year of university. In contrast, anxiety sensitivity was not related to any of our self-reported measures of drinking

behaviour. These data add to research on intrapersonal predictors of alcohol use by focusing on the transition between secondary and post-secondary school, an important developmental period that coincides with normative increases in alcohol consumption. Additionally, our prospective design and thorough characterization of drinking behaviour extends the current literature base, in which cross-sectional designs and frequency-quantity measures of alcohol use are more common. Our study may help to inform psychological targets for individualized prevention programs, which are more effective than generalized approaches in reducing risky drinking among adolescents and young adults (Stewart et al., 2005).

References

- Baer, J. S. (2002). Student factors: understanding individual variation in college drinking. *Journal of Studies on Alcohol*, S14, 40-53. <http://dx.doi.org/10.15288/jsas.2002.s14.40>
- Baer, J. S., Kivlahan, D. R., & Marlatt, G. A. (1995). High-risk drinking across the transition from high school to college. *Alcoholism: Clinical and Experimental Research*, 19(1), 54-61. <http://dx.doi.org/10.1111/j.1530-0277.1995.tb01472.x>
- Balodis, I., Potenza, M., & Olmstead, M. (2009). Binge drinking in undergraduates: Relationships with sex, drinking behaviors, impulsivity, and the perceived effects of alcohol. *Behavioural Pharmacology*, 20(5-6; SI), 518-526. <http://dx.doi.org/10.1097/FBP.0b013e328330c779>
- Bardo, M. T., Donohew, R. L., & Harrington, N. G. (1996). Psychobiology of novelty seeking and drug seeking behavior. *Behavioural Brain Research*, 77, 23-43. [http://dx.doi.org/10.1016/0166-4328\(95\)00203-0](http://dx.doi.org/10.1016/0166-4328(95)00203-0)
- Beck, K. H., Kasperski, S. J., Caldeira, K. M., Vincent, K. B., O'Grady, K. E. & Arria, A.M. (2010). Trends in alcohol-related traffic risk behaviors among college students. *Alcoholism: Clinical and Experimental Research*, 34(8), 1472-1478.
- Buelow, M. T., & Suhr, J. A. (2013). Personality characteristics and state mood influence individual deck selections on the Iowa Gambling Task. *Personality and Individual Differences*, 54, 593-597. <http://dx.doi.org/10.1016/j.paid.2012.11.019>
- Carney, M. A., Tennen, H., Affleck, G., del Boca, F. K., & Kranzler, H. R. (1998). Levels and patterns of alcohol consumption using timeline follow-back, daily diaries and real-time. *Journal of Studies on Alcohol and Drugs*, 59(4), 447-454. <http://dx.doi.org/10.15288/jsa.1998.59.447>
- Chassin, L., King, K. M., & Flora, D. B. (2004). Trajectories of alcohol and drug use and dependence from adolescence to adulthood: the effects of familial alcoholism and personality. *Journal of Abnormal Psychology*, 113(4), 483-498. <http://dx.doi.org/10.1037/0021-843X.113.4.483>

- Cox, B. J., Swinson, R. P., Shulman, I. D., Kuch, K., & Reichman, J. T. (1993). Gender effects and alcohol use in the panic disorder with agoraphobia. *Behavior Research & Therapy*, 31(4), 413-416. [http://dx.doi.org/10.1016/0005-7967\(93\)90099-G](http://dx.doi.org/10.1016/0005-7967(93)90099-G)
- de Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, 14, 22-31. <http://dx.doi.org/10.1111/j.1369-1600.2008.00129.x>
- Del Boca, F. K., & Darkes, J. (2003). The validity of self-reports of alcohol consumption: state of the science and challenges for research. *Addiction*, 98(S2), 1-12. <http://dx.doi.org/10.1046/j.1359-6357.2003.00586.x>
- Dixon, L. J., Viana, A. G., & Stevens, E. N. (2014). Anxiety sensitivity as a moderator of the relationship between trait anxiety and illicit substance use. *Psychology of Addictive Behaviors*, 28(4), 1284-1289. <http://dx.doi.org/10.1037/a0037643>
- Dom, G., Hulstijn, W., & Sabbe, B. (2006). Differences in impulsivity and sensation seeking between early- and late-onset alcoholics. *Addictive Behaviors*, 31, 298-308. <http://dx.doi.org/10.1016/j.addbeh.2005.05.009>
- Faul, F., Erdfelder, E., Lang, A., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175-191. <http://dx.doi.org/10.3758/BF03193146>
- Fleming, M. F., Barry, K. L., & MacDonald, R. (1991). The Alcohol Use Disorders Identification Test (AUDIT) in a college sample. *The International Journal of the Addictions*, 26(11), 1173-1185. <http://dx.doi.org/10.3109/10826089109062153>
- Fox, H. C., Bergquist, P. G., & Sinha, R. (2010). Interactive effects of cumulative stress and impulsivity on alcohol consumption. *Alcoholism: Clinical & Experimental Research*, 34(8), 1376-1385.
- Goudriaan, A. E., Grekin, E. R., & Sher, K. J. (2011). Decision making and response inhibition as predictors of heavy alcohol use: A prospective study. *Alcoholism: Clinical and Experimental Research*, 35(6), 1050-1057. <http://dx.doi.org/10.1111/j.1530-0277.2011.01437.x>

- Harden, K. P., & Tucker-Drob, E. M. (2011). Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for a dual systems model. *Developmental Psychology*, 47(3), 739-746. <http://dx.doi.org/10.1037/a0023279>
- Hittner, J. B., & Swickert, R. (2006). Sensation seeking and alcohol use: A meta-analytic review. *Addictive Behaviors*, 1383-1401. <http://dx.doi.org/10.1016/j.addbeh.2005.11.004>
- Humeniuk, R., Ali, R., Babor, T. F., Farrell, M., Formigoni, M. L., Jittiwutikarn, J., de Lacerda, R. B., Ling, W., Marsden, J., Monteiro, M., Nihwatiwa, S., Pal, H., Poznyak, V., & Simon, S. (2008). Validation of the alcohol, smoking and substance involvement screening test (ASSIST). *Addiction*, 103(6), 1039-1047. <http://dx.doi.org/10.1111/j.1360-0443.2007.02114.x>
- Kokotailo, P. K., Egan, J., Gangnon, R., Brown, D., Mundt, M., & Fleming, M. (2004). Validity of the Alcohol Use Disorders Identification Test in college students. *Alcoholism: Clinical and Experimental Research*, 28(6), 914-920. <http://dx.doi.org/10.1097/01.ALC.0000128239.87611.F5>
- Kushner, M. G., Abrams, K., & Borchardt, C. (2000). The relationship between anxiety disorders and alcohol use disorders: a review of major perspectives and findings. *Clinical Psychology Review*, 20(2), 149-171. [http://dx.doi.org/10.1016/S0272-7358\(99\)00027-6](http://dx.doi.org/10.1016/S0272-7358(99)00027-6)
- Lee, M. R., Chassin, L., & Villalta, I. K. (2013). Maturing out of alcohol involvement: Transitions in latent drinking statuses from late adolescence to adulthood. *Development and Psychopathology*, 25, 1137-1153. <http://dx.doi.org/10.1017/S0954579413000424>
- Magid, V., MacLean, M.G., & Colder, C. R. (2007). Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors*, 32, 2046-2061. <http://dx.doi.org/10.1016/j.addbeh.2007.01.015>
- Martens, M. P., Neighbors, C., Lewis, M. A., Lee, C. M., Oster-Aaland, L., & Larimer, M. E. (2007). Relationship between alcohol use and alcohol-related problems among college students. *Journal of Studies on Alcohol and Drugs*, 69(3), 412-419. <http://dx.doi.org/10.15288/jsad.2008.69.412>

- Martinez, J., Sher, K., & Wood, P. K. (2008) Is heavy drinking really associated with attrition from college?: The alcohol-attrition paradox. *Psychology of Addictive Behaviors*, 22, 450-456.
<http://dx.doi.org/10.1037/0893-164X.22.3.450>
- Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, 158(11), 1783-1793.
<http://dx.doi.org/10.1176/appi.ajp.158.11.1783>
- Novak, A., Burgess, E. S., Clark, M., Zvolensky, M. J., & Brown, R. A. (2003). Anxiety sensitivity, self-reported motives for alcohol and nicotine use, and level of consumption. *Journal of Anxiety Disorders*, 17, 165-180. [http://dx.doi.org/10.1016/S0887-6185\(02\)00175-5](http://dx.doi.org/10.1016/S0887-6185(02)00175-5)
- O'Neill, S., Parra, G., & Sher, K. J. (2001). Clinical relevance of heavy drinking during the college years: Cross-sectional and prospective perspectives. *Psychology of Addictive Behaviors*, 15, 350-359.
<http://dx.doi.org/10.1037/0893-164X.15.4.350>
- Patton, J. H., Stanford, M. S., & Barratt, E. S. (. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*, 51(6), 768-774. [http://dx.doi.org/10.1002/1097-4679\(199511\)51:6<768::AID-JCLP2270510607>3.0.CO;2-1](http://dx.doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1)
- Perry, J. L., & Carroll, M. E. (2008). The role of impulsive behavior in drug abuse. *Psychopharmacology*, 200, 1-26. <http://dx.doi.org/10.1007/s00213-008-1173-0>
- Peterson, R. A., & Heilbronner, R. L. (1987). The Anxiety Sensitivity Index: construct validity and factor analytic structure. *Journal of Anxiety Disorders*, 1: 117-121. [http://dx.doi.org/10.1016/0887-6185\(87\)90002-8](http://dx.doi.org/10.1016/0887-6185(87)90002-8)
- Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. *Behaviour Research and Therapy*, 24(1), 1-8.
[http://dx.doi.org/10.1016/0005-7967\(86\)90143-9](http://dx.doi.org/10.1016/0005-7967(86)90143-9)
- Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection

- of persons with harmful alcohol consumption: II. *Addiction*, 88, 791-804.
<http://dx.doi.org/10.1111/j.1360-0443.1993.tb02093.x>
- Sinha, R. (2008). Chronic stress, drug use, and vulnerability to addiction. *Annals of the New York Academy of Sciences*, 1141, 105-130. <http://dx.doi.org/10.1196/annals.1441.030>
- Sinha, R. (2001). How does stress increase risk of drug abuse and relapse? *Psychopharmacology*, 158, 343-359. <http://dx.doi.org/10.1007/s002130100917>
- Schmidt, N. B., Buckner, J. D., & Keough, M. E. (2007). Anxiety sensitivity as a prospective predictor of alcohol use disorders. *Behavior Modification*, 31(2), 202-219.
<http://dx.doi.org/10.1177/0145445506297019>
- Slutske, W.S. (2005) Alcohol use disorders among US college students and their non-college-attending peers. *Archives of General Psychiatry*, 62(3), 321-327.
<http://dx.doi.org/10.1001/archpsyc.62.3.321>
- Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S. L., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt Impulsiveness Scale: an update and review. *Personality and Individual Differences*, 47, 385-395. <http://dx.doi.org/10.1016/j.paid.2009.04.008>
- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, 28, 78-106. <http://dx.doi.org/10.1016/j.dr.2007.08.002>
- Sobell, L. C., & Sobell, M. B. (2008). Alcohol Timeline Followback (TLFB). In American Psychiatric Association (Ed.), *Textbook of psychiatric measures* (pp. 477-479). Washington, DC: American Psychiatric Association.
- Stewart, S. H., Conrod, P. J., Marlatt, A., Comeau, M. N., Thush, C., & Krank, M. (2005). New developments in prevention and early intervention for alcohol abuse in youths. *Alcoholism: Clinical and Experimental Research*, 29(2), 278-286. [http://dx.doi.org/10.1016/S0899-3289\(97\)90018-3](http://dx.doi.org/10.1016/S0899-3289(97)90018-3)

- Stewart, S. H., Karp, J., Pihl, R. O., & Peterson, R. A. (1997). Anxiety sensitivity and self-reported reasons for drug use. *Journal of Substance Abuse*, 9, 223-240. [http://dx.doi.org/10.1016/S0899-3289\(97\)90018-3](http://dx.doi.org/10.1016/S0899-3289(97)90018-3)
- Stewart, S. H., Peterson, J. B., & Pihl, R. O. (1995). Anxiety sensitivity and self-reported alcohol consumption rates in university women. *Journal of Anxiety Disorders*, 9(4), 283-292. [http://dx.doi.org/10.1016/0887-6185\(95\)00009-D](http://dx.doi.org/10.1016/0887-6185(95)00009-D)
- Stewart, S. H., Zvolensky, M. J., & Eifert, G. H. (2001). Negative-reinforcement drinking motives mediate the relation between anxiety sensitivity and increased drinking behavior. *Personality and Individual Differences*, 31, 157-171. [http://dx.doi.org/10.1016/S0191-8869\(00\)00213-0](http://dx.doi.org/10.1016/S0191-8869(00)00213-0)
- White, H. R., & Labouvie, E. W. (1989). Towards the assessment of adolescent problem drinking. *Journal of Studies on Alcohol*, 50, 30–37.
- World Health Organization (WHO) Working Group (2002). The alcohol, smoking and substance involvement screening test (ASSIST): development, reliability and feasibility. *Addiction*, 97(9), 1183-1194. <http://dx.doi.org/10.15288/jsa.1989.50.30>
- Zucker, R. A., Wong, M. M., Clark, D. B., Leonard, K. E., Schulenberg, J. E., Cornelius, J. R., Fitzgerald, H. E., Homish, G. G., Merline, A., Nigg, J. T., O'Malley, P. M., & Puttler, L. I. (2006). Predicting risky drinking outcomes longitudinally: what kind of advance notice can we get? *Alcoholism: Clinical and Experimental Research*, 30(2), 243–252. <http://dx.doi.org/10.1111/j.1530-0277.2006.00033.x>
- Zuckerman, M. (1979). *Sensation seeking: Beyond the optimal level of arousal*. Hillsdale, NJ: Erlbaum.
- Zuckerman, M. (2002). Zuckerman–Kuhlman personality questionnaire (ZKPQ): an alternative five-factorial model. In M. Perugini, & B. DeRaad (Eds.), *Big five assessment* (pp. 377-396). Seattle, WA: Hogrefe and Huber Publishers.
- Zuckerman, M., & Link, K. (1968). Construct validity for the sensation-seeking scale. *Journal of Consulting and Clinical Psychology*, 32(4), 420-426. <http://dx.doi.org/10.1037/h0026047>

Chapter 3

Acute Stress Increases Voluntary Consumption of Alcohol in Undergraduates

Abstract

Aims: The primary aim of this study was to assess whether an acute stressor directly increases alcohol intake among undergraduates. A secondary aim was to examine whether individual differences in state anxiety predict alcohol intake. **Method:** Following random assignment, undergraduate students (47% men; mean age = 20.1 years \pm 2.8) completed the Trier Social Stress Test or no-stress protocol, and then engaged in a 30-min free-drinking session (alcohol, placebo, or non-alcoholic beverage). The State-Trait Anxiety Inventory was completed upon arrival, post-stressor, and after drinking. **Results:** Planned comparisons demonstrated that psychosocial stress increased voluntary intake of alcohol, but not placebo or non-alcoholic beverages. In linear regression analyses, individual differences in anxiety did not predict voluntary alcohol consumption. **Conclusion:** A proximal relationship exists between acute stress and single-session alcohol intake in undergraduates, which may explain the relationship between life stressors and increased drinking in this group. These findings demonstrate that stress management is an important target for reducing heavy episodic drinking on university campuses.

Introduction

Risky drinking is a significant problem among undergraduate students, many of whom exhibit high rates of alcohol consumption (Balodis et al., 2009). Heavy drinking in this group is not only associated with social, academic and health problems (Wechsler and Nelson, 2001), but also significantly predicts later development of alcoholism (O'Neill et al., 2001). Thus, factors that contribute to excessive consumption of alcohol during college and university may indirectly confer risk for later alcohol abuse. Stress is one of the most likely contributing factors in that stressful life events are associated with elevated alcohol use, as well as increased likelihood of alcohol abuse (Enoch, 2011; Boden et al., 2014). Moreover, students studying in higher education institutions experience elevated levels of stress related to new time demands, greater workload, financial strain and examinations (Robotham and Julian, 2006). As with other populations, university students report increased alcohol consumption during periods of increased life stress (Park et al., 2004).

Given the relationship between life stressors and alcohol use, it seems plausible that acute stress directly increases alcohol consumption. Human studies addressing this hypothesis, however, have produced mixed results. Early findings showed that exposure to an acute stressor increases alcohol intake (Hull and Young, 1983), although more recent research suggests that the effect is no greater than a placebo control, at least in healthy social drinkers (de Wit et al., 2003). It is possible that the stress-alcohol association may be limited to, or at least more pronounced in, pathological populations. This appears to be the case with high anxiety, in that alcohol use disorders are highly comorbid with anxiety disorders (Grant et al., 2004), and the association between stress and drinking is particularly strong in these groups (Waldrop et al., 2007). The co-occurrence of anxiety disorders and alcohol use disorders may reflect the fact that individuals with pathological anxiety are motivated to 'self-medicate' by drinking (Khantzian, 1997). These findings fit with preclinical studies showing that pharmacological blockade of stress systems decreases excessive intake of alcohol to a greater extent in dependent, versus non-dependent, rats (see Silberman et al., 2009; Mahoney and Olmstead, 2013). It should not be surprising,

therefore, that much of the research examining stress effects on voluntary alcohol intake has focused on populations with substance abuse and/or anxiety disorders. For example, stress-induced increases in voluntary alcohol intake are seen in social phobia (Abrams et al., 2002) and among individuals at risk for alcohol use disorders, such as those with a positive family history of alcoholism (Soderpalm Gordh et al., 2011).

In contrast to this work with clinical populations, the relationship between acute stress and alcohol consumption in healthy individuals is not well understood. The relationship may be explained by individual differences in anxiety, given that everyday alcohol consumption in non-pathological populations depends on an individual's perceived level of stress (Park et al., 2004). Moreover, both normal drinkers and alcoholics commonly endorse using alcohol for the express purpose of reducing anxiety (Boys et al., 2001; Robinson et al., 2009). Taken together, these data highlight the subjective nature of perceived stress and suggest that some individuals consume alcohol to directly manage dynamic states of subjective anxiety. Thus, state anxiety may be an important direct predictor of voluntary alcohol intake, irrespective of exposure to acute stress. This is particularly relevant to undergraduate populations who face a variety of dynamic stressors, some of which may be transient in nature (Robotham and Julian, 2006).

The primary purpose of the current study was to examine whether acute stress increases alcohol consumption in an undergraduate population. In order to control for the effects of alcohol expectancies, both placebo and sober control groups were included. Another aim of this experiment was to examine whether individual differences in state anxiety predict single session alcohol intake.

Methods

Participants.

The protocol for the current study was approved by Queen's University Graduate Research Ethics Board. Seventy-five undergraduate students (35 men and 40 women) were recruited using the subject pool from an introductory undergraduate psychology course, as well as print advertisements on campus.

To be eligible for participation, students were required to be at least 19 years of age (the legal drinking age in Ontario) and report drinking alcohol at least once per month. Exclusion criteria included previous medical history contraindicating the use of alcohol, allergy to alcohol and/or use of medication that may interact with the effects of alcohol. These inclusion and exclusion criteria were assessed using a screening questionnaire that was completed before individuals arrived at the laboratory. Due to the deleterious effects of alcohol use during pregnancy, women were only permitted to participate on the day of testing if they were menstruating, or had not had sexual intercourse since their last menstruation. Prior to the beginning of experimental procedures, participants were randomly assigned to one of three drinking conditions: alcohol ($n = 24$), placebo ($n = 26$) or sober ($n = 25$). Within each of these groups, participants were randomly assigned to a stress ($n = 24$) or no-stress ($n = 51$) condition. The discrepancy in size between these two groups is owing to the fact that the stress manipulation requires additional personnel and laboratory resources, restricting the rate at which participants were run in this condition over time.

Self-Report Measures.

State-Trait Anxiety Inventory.

The State-Trait Anxiety Inventory (STAI) is a questionnaire that measures feelings associated with anxiety, such as tension, apprehension, nervousness and worry (Spielberger, 1983). The STAI includes two subscales, the STAI-Trait (STAIT) that measures trait anxiety (long-standing individual quality), and the STAI-State (STAIS) that measures state anxiety (temporary status associated with reactivity to acute stressors). Each subscale is comprised of 20 items and provides a rating from 20 to 80, with higher scores relating to greater anxiety.

Perceived Intoxication.

A modified version of the Drug Effects Questionnaire (de Wit et al., 2003; Ortner et al., 2003) was used to assess subjective feelings associated with intoxication. This brief self-report questionnaire asked participants to estimate how much alcohol they consumed, their blood alcohol level, as well as rate how intoxicated they feel, how much they enjoy how they feel, and the extent to which they want more

alcohol. It served as a manipulation check to assess subjective intoxication and the effectiveness of the placebo condition.

Stress Condition.

For individuals assigned to the stress condition, the Trier Social Stress Test (TSST) was used to induce a stress response (Kirschbaum et al., 1993). The TSST is a psychosocial stressor that capitalizes on highly stressful factors including uncontrollability, forced failure, and social-evaluative threat. The procedure reliably elicits elevations in stress measures in healthy normal populations (Kudielka et al., 2007) including a robust hypothalamic–pituitary–adrenal (HPA) axis response with a long recovery time (Kirschbaum et al., 1993). This task involved performing a 5-min speech in front of a panel of student actors, followed by a 5-min mental arithmetic task. The TSST was conducted in a separate room, where there were two student-actors who were introduced to the participant as a doctoral student in linguistics specializing in non-verbal behaviour, and a professor in the Psychology department. The participant was asked to give a mock job talk for a position as a research assistant in the Psychology department, then was given a pen and paper and allowed 5 min to prepare. When the preparation time had expired, any notes the participant had made were taken away and the individual was told that their performance would be compared against their written information. The participant was able to see his or her image on the LED screen of a camcorder during their performance. Participants assigned to the no-stress condition were brought into a separate room, where they sat quietly and completed simple crossword puzzles, in order to provide some cognitive stimulation, for the same amount of time as the stress protocol.

Drinking Protocol.

Participants were requested to fast for 3 h prior to testing in order to minimize variability in the rate of alcohol absorption. A modified version of a validated ad libitum drinking protocol was employed (de Wit et al., 2003) in which participants were instructed to consume an initial drink of their beverage (alcohol, placebo, or non-alcoholic), and were then permitted to continue consuming the beverage ad libitum for the next half hour, up to a maximum of six drinks. For the alcohol group, the initial drink

contained 0.2 g/kg alcohol for women and 0.3 g/kg alcohol for men; subsequent beverages contained 0.1 g/kg alcohol for both sexes. The alcoholic beverages were comprised of two parts calorie-free soda to one part vodka. Placebo beverages contained flattened tonic water instead of alcohol and, to mimic smell and taste cues, a small amount of alcohol was placed on the rim of the glass and floated on top of the beverage.

In the sober condition, participants received a non-alcoholic beverage (calorie-free soda). For both the placebo and alcohol groups, participants were told they were receiving moderate-dose alcoholic beverages, whereas participants in the sober group were told they were receiving non-alcoholic beverages. In order to maintain ecological validity, all participants completed the drinking protocol in a laboratory setting that simulated a comfortable living room environment while watching DVDs of a popular TV show. Consistently, participants always completed the free-drinking portion with one or more other individuals, as drinking alone occurs rarely (~ 15%) among university students (O'Hare, 1990). Participants and, when necessary, confederates completed the protocol in a staggered fashion in order to allow individuals to drink together without participants knowing how much alcohol the other individual had consumed. If only one individual participated in a given testing block, a student-actor served as a confederate, posing as another participant, during the free-drinking portion.

Experimental Procedure.

After arriving in the laboratory, participants underwent a final screening for eligibility and provided written consent. Participants were weighed for beverage dosage, and an initial blood alcohol level (BAL) reading was taken using a breathalyser (Intoxilyzer 400D; CMI, Inc., Owensboro, KY, USA). Participants first completed a set of self-report questionnaires, the STAIT and STAIS (T1), before going to a separate room to undergo the stress or no-stress protocol depending on their group assignment. Participants then returned to the laboratory and completed the STAIS again (T2). They were then told whether their beverage was alcoholic or non-alcoholic, and began the drinking protocol. After the free-drinking period, participants completed the modified Drug Effects Questionnaire as a manipulation check as well as the

final STAIS (T3), and provided another BAL reading. A brief behavioural measure was completed as part of a separate study, before participants were debriefed and compensated with course credit through the psychology subject pool or with cash remuneration (\$10). Participants in the alcohol group stayed in the laboratory until their BAL was below 0.06% and were provided with a taxi ride home. The procedural timeline is depicted in Fig. 3-1.

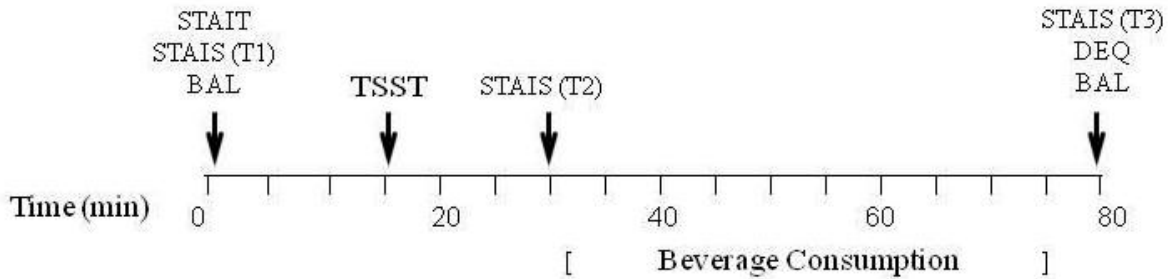


Figure 3-1. Graphical representation of experimental timeline.

Note. STAIT = State-Trait Anxiety Inventory – Trait. STAIS = State-Trait Anxiety Inventory – State. BAL = Blood alcohol level. TSST = Trier Social Stress Test. DEQ = Drug Effects Questionnaire. T1 = Time 1, T2 = Time 2, T3 = Time 3.

Results

Participants.

Participants were between 19 and 36 years old with a mean age of 20.12 years ($SD = 2.81$). They reported drinking an average of 1.69 times per week ($SD = 0.85$). The proportion of men versus women did not differ by beverage group, $\chi^2(2, N = 75) = 0.89, p = 0.51$, or stress condition ($p = 0.81$). Average weight (kg) did not differ between the three beverage groups, $F(2, 74) = 2.01, p = 0.14$.

Manipulation Checks.

Stress.

A 2 x 3 repeated measures ANOVA (see Fig. 3-2), with stress condition as the between-subjects variable and time as the within subjects variable revealed a main effect of stress, $F(1, 73) = 16.85, p < 0.001$, and a main effect of time, $F(2, 133.02) = 45.52, p < 0.001$ (Huynh-Feldt corrected) on STAIS scores. Following a significant stress \times time interaction, $F(2, 146) = 43.76, p < 0.001$, simple effects

analyses with Bonferroni correction ($\alpha = 0.008$) showed the effect of time occurred only within the stress group, $F(2, 72) = 48.75, p < 0.001$, wherein scores at T2 were significantly higher than at T1 and T3, and scores at T3 were significantly higher than at T1, all $p < 0.008$. The effect of stress only emerged at T2, $F(1, 73) = 59.59, p < 0.001$, in that the stress group reported higher STAIS scores than the no-stress group, $p < 0.008$.

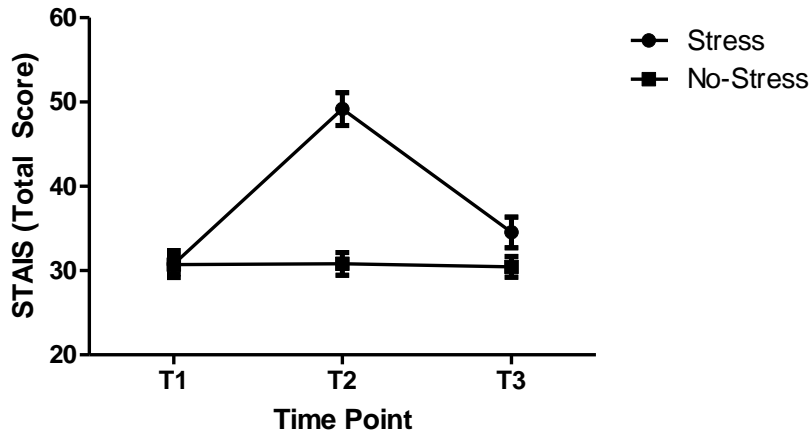


Figure 3-2. Effect of stress on state anxiety across experimental time points.

Note. STAIS = State-Trait Anxiety Inventory – State. T1 = Time 1, T2 = Time 2, T3 = Time 3.

Perceived Intoxication.

Table 3-1 shows BAL and self-reports of perceived intoxication levels in the placebo and alcohol groups. The sober group is not included in this table because they did not receive alcohol and did not provide reports of perceived intoxication.

Table 3-1. Mean (SEM) blood alcohol level (BAL) and self-reported perceived intoxication in placebo and alcohol groups.

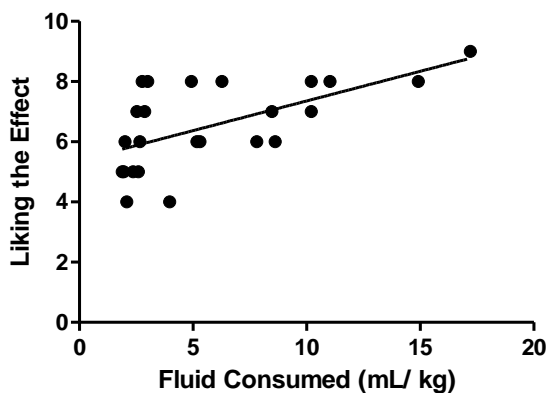
Beverage Group	BAL	Perceived Intoxication				
		Alcohol Consumed (Bottles of Beer)	Estimated Intoxication Level	Estimated BAL	Liking the Effects	Desiring More Alcohol
Placebo	0.00 (0.00)	3.38 (.30)	2.87 (.20)	0.07 (0.05)	6.28 (.29)	5.24 (.52)

Alcohol	0.04 (0.01)	4.71(.36)	3.96 (.29)	0.11 (0.03)	6.54 (.29)	5.54 (.50)
----------------	-------------	-----------	------------	-------------	------------	------------

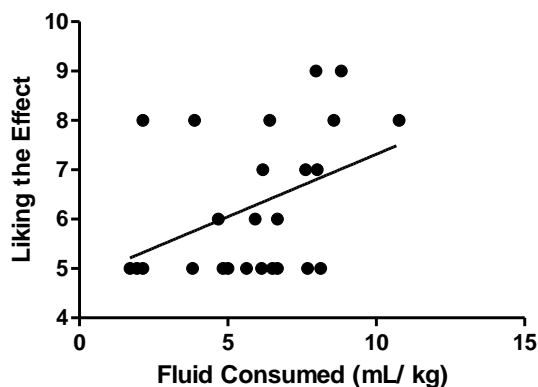
Note. Participants estimated the alcohol they had consumed in terms of the number of drinks consumed. In addition, they rated their level of intoxication, how much they liked the effects they felt, and how much they desired more alcohol (all scales 1-9).

Pearson correlation analysis was used to examine the relationship between fluid intake and manipulation check items in the alcohol and placebo groups. Within the alcohol group, fluid consumption per body weight (ml/kg) was significantly positively correlated with liking the effects of alcohol, $r = 0.60$, $p < 0.01$, and wanting more alcohol, $r = 0.70$, $p < 0.01$, but was not correlated with estimations of intoxication, all p -values > 0.05 . The same pattern emerged in the placebo group, in that fluid consumption by weight was significantly positively correlated with liking the effects of alcohol, $r = 0.41$, $p < 0.05$, and wanting more alcohol, $r = 0.62$, $p < 0.01$, but not with estimations of intoxication, all p -values > 0.05 . These relationships are shown in Figure 3-3.

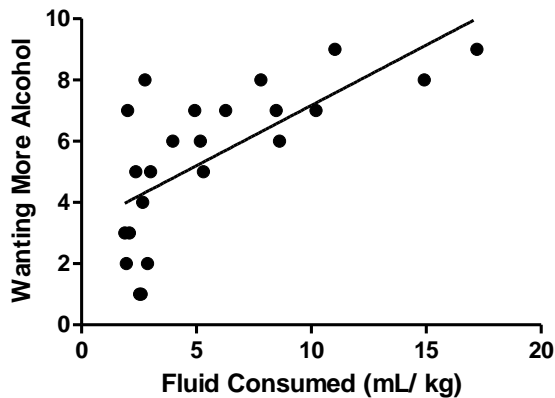
(a)



(b)



(c)



(d)

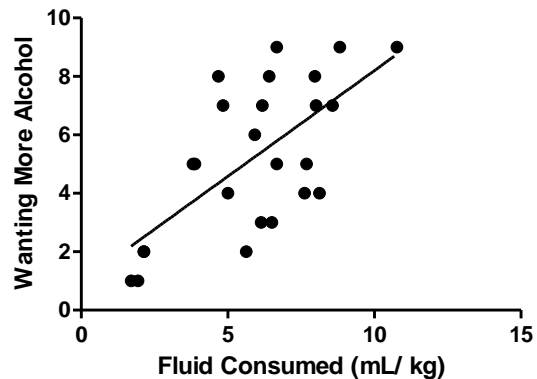


Figure 3-3. Relationship between manipulation check items and fluid intake.

Note. The top scatter plots show correlations between fluid intake (mL/ kg) and liking the effects of the beverage (scale 1-9) in the alcohol (a) and placebo (b) groups; the bottom scatter plots show correlations between fluid intake (mL/ kg) and wanting more beverage (scale 1-9) in the alcohol (c) and placebo (d) groups.

Effect of Stress on Fluid Intake.

Planned comparisons were conducted to test the specific a priori hypothesis that participants would consume more fluid in the stress condition compared with the no-stress condition, but only in the alcohol group. Comparisons using orthogonal weighted contrasts to compare the stress and no-stress groups were conducted at each level of the beverage group variable. Given a priori predictions about directionality of the effect— mean fluid consumption will be higher for the stress condition compared with the no-stress condition—the tests were one tailed. The contrast analyses demonstrated that there was no significant difference in mean fluid consumed between the stress and no-stress conditions in the sober (Contrast Estimate [CE] = 37.72, SE = 85.48), $p = 0.66$, or placebo (CE = -0.78, SE = 84.72), $p = 0.99$, groups. In contrast, within the alcohol group (CE = 169.06, SE = 86.33), mean fluid consumption was significantly higher in the stress condition compared with the no-stress condition, $p < 0.05$. These results are summarized in Figure 3-4.

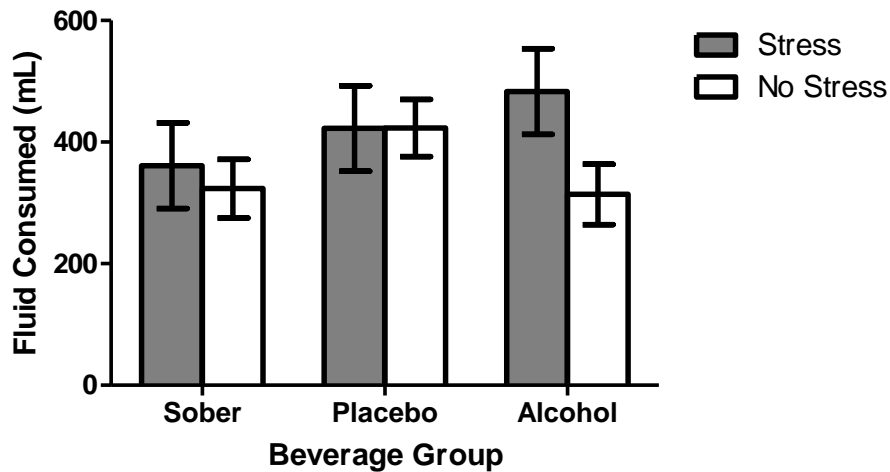


Figure 3-4. Effect of stress and beverage group on fluid intake.

Note. Bars represent mean (+SEM) fluid intake (total mL) for participants in sober, placebo, and alcohol groups in the stress and no-stress conditions.

Relationship between Self-Report Anxiety Measures and Alcohol Intake.

Nonparametric (bootstrapped with 10,000 re-samples) linear regression analysis was used to examine the relationship between alcohol intake and self-report anxiety measures within the alcohol group. Trait anxiety (STAIT scores) did not significantly predict alcohol intake (ml), $\beta = -0.05$, $t(22) = -0.25$, $p = 0.81$. State anxiety, (STAIS scores) did not significantly predict alcohol intake (ml) at T1, $\beta = -0.14$, $t(22) = -0.25$, $p = 0.51$, or T2, $\beta = 0.04$, $t(22) = 0.20$, $p = 0.84$.

Discussion

A primary finding of our study was that acute stress selectively increases voluntary intake of alcohol. Earlier work demonstrated that alcohol intake increases following exposure to an acute stressor (e.g. Higgins and Marlatt, 1975), a finding that was replicated in subsequent work (e.g. Pelham et al., 1997; de Wit et al., 2003; Nestic & Duka, 2006; Soderpalm Gordh et al., 2011). Nonetheless, these results are difficult to interpret without sober and placebo control groups. For example, the inclusion of a placebo control group showed that intake of both alcohol and placebo beverages increases post-stressor (e.g. de Wit et al., 2003). Thus, it is not clear whether the effect of stress is specific to alcohol drinking, or non-specifically related to factors such as thirst. We examined this possibility by including both sober and

placebo control groups in our experimental design, revealing that acute stress specifically increases the intake of alcohol. As such, our data present the first evidence, in humans, that stress-induced alcohol consumption is related to the pharmacological effect of alcohol. This finding does not support a role for the expectancy of intoxication or other factors, such as thirst, in increased drinking following a stressor. The discrepancy between our results and findings of non-specific increases in both alcohol and placebo consumption following stress may also relate to other methodological differences. For example, previous studies have employed mixed between- and within-subjects designs (e.g. deWit et al., 2003), in which a significant change between stress and no-stress conditions is determined, at least in part, on an individual level.

The fact that stress increased beverage consumption, selectively, in the alcohol group relates to the theory that small amounts of alcohol prime individuals to seek more of the drug (Field et al., 2010). This may occur through an interaction with cognitive mechanisms controlling impulsivity in that a moderate dose of alcohol impairs inhibitory control, which in turn significantly predicts ad lib intake of alcohol (Weafer and Fillmore, 2008). The finding that this effect is more pronounced in the stress condition may be related to the fact that stress and impulsivity interact to increase alcohol consumption (Hamilton et al., 2013) and alcohol-related problems (Fox et al., 2010) in normal healthy drinkers. This possibility could be addressed in future research examining how trait or behavioural impulsivity moderates the relationship between acute stress and ad lib alcohol consumption.

Despite our finding that acute stress increases alcohol intake, our data did not support the notion that individual differences in anxiety relate to voluntary alcohol consumption. According to self-medication theories, individuals drink to alleviate anxiety but participants in our study who reported greater subjective anxiety, both upon arrival in the laboratory and prior to drinking, did not consume more alcohol. Similarly, trait anxiety did not predict alcohol consumption. Moreover, acute stress did not increase intake of the placebo beverage, again contradicting self medication hypotheses in that the expectancy of alcohol (and the subsequent alleviation of anxiety) should motivate alcohol drinking. The

lack of a relationship between state anxiety and alcohol intake in normal, healthy undergraduates may lend support to the notion that this association is limited to pathological populations. However, it could be that healthy normal participants exhibit insufficient dynamic range in measures of anxiety to produce the robust effects on alcohol consumption seen with pathological populations. Alternatively, it may be the case that individual differences in other factors, such as anxiety sensitivity, contribute to the relationship between subjective anxiety and alcohol intake and this should be explored, further, in non-pathological populations.

Replicating previous studies, our placebo manipulation was robustly effective in terms of perceived intoxication (Balodis et al., 2011; Christiansen et al., 2013; Magrys et al., 2013) in that the placebo group's estimated BAL did not differ significantly from that of the alcohol group. In terms of the positive subjective effects of alcohol, the placebo and alcohol groups demonstrated the same pattern: greater volume of beverage intake was significantly positively correlated with liking the effects and wanting more. To our knowledge, this dose-dependent placebo effect, relating beverage intake to the expected, positive subjective effects of alcohol, is a novel finding. In contrast, neither the alcohol nor the placebo groups showed a significant relationship between alcohol intake and any measures of estimated intoxication. In other words, intoxicated individuals were no better than participants in the placebo group at estimating their level of intoxication relative to the amount of beverage they had consumed. Thus, perceived levels of intoxication do not appear to be aided by physiological cues related to the pharmacological effects of alcohol, at least in this population.

In sum, our study provides evidence for a proximal relationship between acute stress and single-session alcohol intake, but does not support state anxiety as a significant predictor of this behaviour. Past research has examined whether acute stressors directly increase voluntary consumption of alcohol; our study clarifies this relationship by accounting for expectancy and general stress effects through the use of placebo and sober controls, respectively.

Potential limitations of the current study, and directions for future research, should be noted. It is

possible that our study, given its relatively small number of participants, may have failed to detect subtle patterns in the data. Future experiments involving a greater number of participants would help clarify whether additional results, such as placebo- or dose-related effects, would be detected with greater statistical power. Although efforts were made to simulate a normative drinking environment for undergraduates (living room setting, evening time, in the presence of peers), the generalizability of these findings may have been limited by certain factors that do not reflect typical drinking circumstances, such as the duration of the free-drinking period of ~30 min. Some studies have begun to examine ad lib drinking in naturalistic settings (e.g. Bot et al., 2005) and this approach would be useful in further exploring the effect of stress on alcohol intake. Our findings highlight the immediate need for interventions focused on stress-reduction in order to diminish heavy episodic drinking among undergraduate students and, thereby, reduce the risk for future alcohol use disorders.

References

- Abrams, K., Kushner, M.G., Medina, K.L., et al. (2002). Self-administration of alcohol before and after a public speaking challenge by individuals with social phobia. *Psychology of Addictive Behaviors*, 16, 121–8. <http://dx.doi.org/10.1037/0893-164X.16.2.121>
- Balodis, I., Potenza, M., & Olmstead, M. (2009). Binge drinking in undergraduates: relationship with sex, drinking behaviours, impulsivity, and the perceived effects of alcohol. *Behavioral Pharmacology*, 20, 518–26. <http://dx.doi.org/10.1097/FBP.0b013e328330c779>
- Balodis, I.M., Wynne-Edwards, K.E., & Olmstead, M.C. (2011). The stress-response-dampening effects of placebo. *Hormones & Behavior*, 59, 465–72. <http://dx.doi.org/10.1016/j.yhbeh.2011.01.004>
- Boden, J.M., Fergusson, D.M., & Horwood, L.J. (2014). Associations between exposure to stressful life events and alcohol use disorder in longitudinal birth cohort studied to age 30. *Drug & Alcohol Dependence*, 142, 154–60. <http://dx.doi.org/10.1016/j.drugalcdep.2014.06.010>
- Bot, S.M., Engels, R.C., & Knibbe, R.A. (2005). The effects of alcohol expectancies on drinking behaviour in peer groups: observations in a naturalistic setting. *Addiction*, 100, 1270–9. <http://dx.doi.org/10.1111/j.1360-0443.2005.01152.x>
- Boys, A., Marsden, J., & Strang, J. (2001). Understanding reasons for drug use amongst young people: a functional perspective. *Health Education Research*, 16, 457–69. <http://dx.doi.org/10.1093/her/16.4.457>
- Christiansen, P., Rose, A.K., Cole, J.C., et al. (2013). A comparison of the anticipated and pharmacological effects of alcohol on cognitive bias, executive function, craving and ad-lib drinking. *Journal of Psychopharmacology*, 27, 84–92. <http://dx.doi.org/10.1177/0269881112450787>
- de Wit, H., Soderpalm, A.H., Nikolayev, L., et al. (2003). Effects of acute social stress on alcohol consumption in healthy subjects. *Alcoholism: Clinical & Experimental Research*, 27, 1270–7. <http://dx.doi.org/10.1097/01.ALC.0000081617.37539.D6>

- Enoch, M.A. (2011). The role of early life stress as a predictor for alcohol and drug dependence. *Psychopharmacology*, 214, 17–31. <http://dx.doi.org/10.1007/s00213-010-1916-6>
- Field, M., Wiers, R.W., Christiansen, P., et al. (2010). Acute alcohol effects on inhibitory control and implicit cognition: implications for loss of control over drinking. *Alcoholism: Clinical & Experimental Research*, 34, 1346–52. <http://dx.doi.org/10.1111/j.1530-0277.2010.01218.x>
- Fox, H.C., Bergquist, K.L., Gu, P., et al. (2010). Interactive effects of cumulative stress and impulsivity on alcohol consumption. *Alcoholism: Clinical & Experimental Research*, 34, 1376–85.
- Grant, B.F., Stinson, F.S., Dawson, D.A., et al. (2004). Prevalence and co-occurrence of substance use disorders in independent mood and anxiety disorders: results from the national epidemiologic survey on alcohol and related conditions. *Archives of General Psychiatry*, 61, 807–16. <http://dx.doi.org/10.1001/archpsyc.61.8.807>
- Hamilton, K.R., Ansell, E.B., Reynolds, B., et al. (2013). Self-reported impulsivity, but not behavioral choice or response impulsivity, partially mediates the effect of stress on drinking behavior. *Stress* 16, 3–15. <http://dx.doi.org/10.3109/10253890.2012.671397>
- Higgins, R.L., & Marlatt, G.A. (1975). Fear of interpersonal evaluation as a determinant of alcohol consumption in male social drinkers. *Journal of Abnormal Psychology*, 84, 644–51. <http://dx.doi.org/10.1037/0021-843X.84.6.644>
- Hull, J.G., & Young, R.D. (1983). Self-consciousness, self-esteem, and success-failure as determinants of alcohol consumption in male social drinkers. *Journal of Personality & Social Psychology*, 44, 1097–109. <http://dx.doi.org/10.1037/0022-3514.44.6.1097>
- Khantzian, E.J. (1997). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, 4, 231–44. <http://dx.doi.org/10.3109/10673229709030550>
- Kirschbaum, C., Pirke, K.M., & Hellhammer, D.H. (1993). The ‘Trier Social Stress Test’—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 28,

- 76–81. <http://dx.doi.org/10.1159/000119004>
- Kudielka, B.M., Wust, S., Kirschbaum, C., et al. (2007). Trier social stress test. In Fink G (ed).
Encyclopedia of Stress, 2nd ed. Oxford: Academic Press, 776–81.
- Magrys, S.A., Olmstead, M.C., Wynne-Edwards, K.E., et al. (2013). Neuroendocrinological responses to
alcohol intoxication in healthy males: relationship with impulsivity, drinking behavior, and
subjective effects. *Psychophysiology*, 50, 204–9. <http://dx.doi.org/10.1111/psyp.12007>
- Mahoney, M.K., & Olmstead, M.C. (2013). Neurobiology of an endophenotype: modelling the
progression of alcohol addiction in rodents. *Current Opinion in Neurobiology*, 23, 607–14.
<http://dx.doi.org/10.1016/j.conb.2013.03.006>
- Nesic, J., & Duka, T. (2006). Gender specific effects of a mild stressor on alcohol cue reactivity in heavy
social drinkers. *Pharmacology Biochemistry & Behavior*, 83, 239–48.
<http://dx.doi.org/10.1016/j.pbb.2006.02.006>
- O’Hare, T.M. (1990). Drinking in college: consumption patterns, problems, sex differences and legal
drinking age. *Journal of Studies on Alcohol & Drugs*, 51, 536–41.
<http://dx.doi.org/10.15288/jsa.1990.51.536>
- O’Neill, S.E., Parra, G.R., & Sher, K.J. (2001). Clinical relevance of heavy drinking during the college
years: cross-sectional and prospective perspectives. *Psychology of Addictive Behaviors*, 15, 350–
9. <http://dx.doi.org/10.1037/0893-164X.15.4.350>
- Ortner, C.N., MacDonald, T.K., & Olmstead, M.C. (2003). Alcohol intoxication reduces impulsivity in
the delay discounting paradigm. *Alcohol & Alcoholism*, 38, 151–6.
<http://dx.doi.org/10.1093/alcalc/agg041>
- Park, C.L., Armeli, S., & Tennen, H. (2004). The daily stress and coping process and alcohol use among
college students. *Journal of Studies on Alcohol & Drugs*, 65, 126–35.
<http://dx.doi.org/10.15288/jsa.2004.65.126>
- Pelham, W.E., Lang, A.R., Atkeson, B., et al. (1997). Effects of deviant child behavior on parental

- distress and alcohol consumption in laboratory interactions. *Journal of Abnormal Child Psychology*, 25, 413–24. <http://dx.doi.org/10.1023/A:1025789108958>
- Robinson, J.A., Sareen, J., Cox, B.J., et al. (2009). Correlates of self-medication for anxiety disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Nervous & Mental Diseases*, 197, 873–8. <http://dx.doi.org/10.1097/NMD.0b013e3181c299c2>
- Robotham, D., & Julian, C. (2006). Stress and the higher education student: a critical review of the literature. *Journal of Further & Higher Education*, 30, 107–17. <http://dx.doi.org/10.1080/03098770600617513>
- Silberman, Y., Bajo, M., Chappell, A.M., et al. (2009). Neurobiological mechanisms contributing to alcohol stress-anxiety interactions. *Alcohol*, 43, 509–19. <http://dx.doi.org/10.1016/j.alcohol.2009.01.002>
- Soderpalm Gordh, A.H.V., Brkic, S., & Soderpalm, B. (2011). Stress and consumption of alcohol in humans with a Type 1 family history of alcoholism in an experimental laboratory setting. *Pharmacology Biochemistry & Behavior*, 99, 696–703. <http://dx.doi.org/10.1016/j.pbb.2011.05.028>
- Spielberger, C.D. (1983). State-trait Anxiety Inventory (Form Y). Palo Alto, CA: Consulting Psychologists Press.
- Waldrop, A.E., Back, S.E., Verduin, M.L., et al. (2007). Triggers for cocaine and alcohol use in the presence and absence of posttraumatic stress disorder. *Addictive Behavior*, 32, 634–9.
- Weafer, J., & Fillmore, M.T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology*, 201, 315–24. <http://dx.doi.org/10.1007/s00213-008-1284-7>
- Wechsler, H., Nelson, T.F. (2001). Binge drinking and the American college student: what's five drinks? *Psychology of Addictive Behaviors*, 15, 287–91. <http://dx.doi.org/10.1037/0893-164X.15.4.287>

Chapter 4

Neuroendocrinology Responses to Alcohol Intoxication in Health Males: Relationship with Impulsivity, Drinking Behaviour and Subjective Effects

Abstract

Ambiguous biochemical and subjective responses to alcohol may relate to pre-existing individual differences in alcohol expectations, experience, or impulsivity. This study examined cortisol and alpha-amylase responses to alcohol and their association with trait impulsivity, alcohol expectancy, and subjective reports of alcohol's effects. Eighty-seven men assigned to an alcohol, sober, or placebo group provided biochemical and self-report measures. Both cortisol and alpha-amylase increased following alcohol administration. Impulsivity correlated with cortisol changes, and the greatest rise in cortisol correlated with high stimulating effects in the alcohol group. These findings emphasize the importance of individual differences in alcohol responses and support a relationship between hormonal responses and alcohol use.

Introduction

Alcohol produces a variety of physiological effects such as increased heart rate as well as augmented prolactin and adrenal responses (Brunelle, Barrett, & Pihl, 2007; Schuckit, Risch, & Gold, 1988; Soyka, Görig, & Naber, 1991). In contrast, alcohol's effect on cortisol, the primary glucocorticoid hormone in humans, is ambiguous. Some studies report increases in cortisol following alcohol consumption (King, Munisamy, de Wit, & Lin, 2006), whereas others do not (Gianoulakis, Krishnan, & Thavundayil, 1996). Understanding the relationship between alcohol intake and hormonal reactivity is important because dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, whose end product is cortisol, is implicated in the development and maintenance of drug addiction (Koob & Kreek, 2007; Kreek, Nielsen, Butelman, & LaForge, 2005). The HPA axis activity plays an important role in cognition, affective regulation, and stress responding (Blackburn, Whalley, Christie, & Shering, 1987; Heffelfinger & Newcomer, 2001), all of which contribute to maladaptive drug use. The discrepancies in previous findings relating alcohol intake to hormonal responses may be explained by individual differences in alcohol-induced effects. It is well known that the subjective states associated with alcohol intoxication vary widely in humans (Holdstock & de Wit, 1998; Morean, & Corbin, 2010). For example, individual differences reflecting sensitivity to reward and intoxication may contribute to variability in biochemical response to alcohol. Thus, although directionality cannot be confirmed, it is important to examine how individual differences in subjective response to alcohol relate to variability in alcohol-induced hormonal response. Similarly, previous studies have demonstrated that physiological response to alcohol varies depending on trait factors. For example, heightened physiological response to alcohol through increased heart rate is related to disinhibited and aggressive behavior and higher sensitivity to reward (Assaad et al., 2006; Brunelle et al., 2004). These findings therefore suggest that specific physiological responses to alcohol may interact with particular behaviors and personality traits. Specifically, impulsive personality traits are consistently noted in addicted populations and are further linked to relapse (Economidou, Pelloux, Robbins, Dalley, & Everitt, 2009; Mitchell, Fields, D'Esposito, & Boettiger, 2005; Moeller et

al., 2002). In young adults at risk for drug use disorders, higher levels of impulsivity are associated with greater alcohol use and drug experimentation (Balodis, Potenza, & Olmstead, 2009; Balodis, Wynne-Edwards, & Olmstead, 2010). Novelty seeking, a measure of impulsivity, modulates substance-induced dopaminergic activity in the ventral striatum (Leyton et al., 2002), which may contribute to epidemiological findings linking impulsivity to substance abuse. Examining how specific personality traits relate to the physiological and subjective responses to acute alcohol intoxication is therefore important for understanding the development of alcohol use disorders.

The inconsistencies in cortisol responses to alcohol across studies may also reflect methodological differences, such as sampling time or quantitative assessment of hormonal changes. For example, previous work from our group has demonstrated that a recovery period following arrival in the laboratory is necessary to establish an accurate physiological baseline; in addition, reactivity indices of physiological stress (such as the percent change from baseline) are more informative than absolute levels in characterizing biochemical responses (Balodis et al., 2010). A goal of the current experiment was to characterize, more fully, the physiological and subjective responses to alcohol intoxication in a group of young adult men. In line with our previous work, we used several quantitative parameters to analyze biochemical responses. The primary physiological measures were salivary levels of cortisol and alpha-amylase. Salivary alpha-amylase is an enzyme that provides a reliable index of sympathetic adrenal-medullary (SAM) axis responses (Nater et al., 2006). Alpha-amylase activity has been directly associated with various measures of sympathetic activity, elicited by physical and psychological stimuli, which has validated its utility as a useful biomarker of SAM axis response (Nater & Rohleder, 2009). Similar to cortisol, alpha-amylase demonstrates diurnal circadian patterns and is highly sensitive to a variety of stressful conditions in humans (Rohleder, Nater, Wolf, Ehlert, & Kirschbaum, 2004). The role of alcohol expectancy was explored using a placebo condition, in which participants are convinced that they received alcohol. Alcohol expectancies, reflecting an individual's assumptions and beliefs about the drug's effects, have powerful effects on various aspects of cognition and behavior, affecting even higher

order functions such as emotion processing (Walter et al., 2011). Furthermore, alcohol expectancy affects implicit physiological processes such as autonomic regulation (Vaschillo et al., 2008) and, as our previous work indicates, cortisol responses following a psychosocial stressor (Balodis, Wynne-Edwards, & Olmstead, 2011). Given our previous work showing a relationship between impulsivity and drinking patterns in the population under study (Balodis et al., 2009), we included a self-report measure of trait impulsivity and examined whether this measure might modulate alcohol-induced responses. Participants also repeatedly reported subjective states related to alcohol effects after each drink through self-report questionnaires.

We hypothesized that alcohol would increase HPA- and SAM axis activity; however, this is likely moderated by personality measures, such as impulsivity. We also tested whether trait impulsivity might relate to changes in biochemical measures following intoxication and whether this relationship may be moderated in “high-responders.” Finally, we also examined whether the largest changes in biochemical measures would correspond with subjective reports of greater sensitivity to alcohol’s effects.

Methods

Participants.

Eighty-seven full-time male undergraduate students were recruited through a student subject pool, poster advertisements, and class visits at Queen’s University in Kingston, Ontario. Participants were required to be at least 19 years of age (the legal drinking age in Ontario), and had to report consuming alcohol at least once per month. Exclusion criteria included history of a condition that contraindicates the use of alcohol (e.g., migraines, seizures, head injury, or cardiovascular disease), allergic reaction to alcohol, or use of medication that may interact with alcohol. Participants were between 19 and 25 years old with a mean age of 20.7. Participants were asked not to eat 3 h prior to testing and were randomly assigned to experimental condition (placebo, $n = 25$; intoxicated, $n = 31$; sober, $n = 30$) on the day of testing. All participants that completed testing on the same day were assigned to the same experimental group. Four individuals reported smoking on a daily basis: two were in the alcohol group and two in the

sober group. Analyses were conducted with and without these four individuals in the dataset, and inclusion of these individuals did not significantly change the physiological or subjective results. Compensation consisted of course credit or a \$15 honorarium. The experimental protocol was approved by the Queen's University's General Research Ethics Board.

Measures.

Self-Report Measures.

Barratt Impulsiveness Scale.

The Barratt Impulsiveness Scale Version 11 (BIS) was used as a self-report measure of impulsive traits (Patton, Stanford, & Barratt, 1995). The questionnaire consists of 30 items that are rated on a 4-point scale (*rarely/never, occasionally, often, or almost always*), with three subscales: motor, cognitive, and non-planning impulsiveness. Total BIS scores provide a valid measure of impulsivity in undergraduate populations (Patton et al., 1995).

Biphasic Alcohol Effects Scale.

The Biphasic Alcohol Effects Scale (BAES) is a self-report questionnaire examining subjective states associated with intoxication. It has demonstrated good validity and reliability (Martin, Earleywine, Musty, Perrine, & Swift, 1993). The BAES includes both a stimulation and sedation subscale, corresponding with alcohol effects often reported on ascending versus descending limbs of the blood alcohol curve.

Manipulation Check.

A self-report questionnaire was administered after beverage consumption to measure each individual's perceived level of intoxication and to assess the effectiveness of the placebo manipulation. The manipulation check is a modified version of the Drug Effects Questionnaire (Kirk & de Wit, 2000), which includes ratings of the amount of alcohol consumed, level of intoxication, and desire for more alcohol.

Physiological Measures.

Saliva samples were collected at six time points across the experiment; subjects were asked to passively drool 2.0 mL of saliva into a 5-mL tube. No saliva stimulant was used. The samples were labeled and stored in a non-self-defrosting freezer at -20°C. Samples were analyzed for salivary cortisol and alpha-amylase using an expanded range high-sensitivity salivary cortisol enzyme immunoassay kit (Salimetrics, State College, PA) and a salivary alpha-amylase assay kit (Salimetrics) to determine free cortisol and alpha-amylase concentrations. One person's data from the alcohol group constituted an outlier, as their alpha-amylase levels increased by over 500%—this person's data were removed from the analyses.

Procedure.

The details of the experimental timeline are presented in Figure 4-1. All sessions took place between the hours of 17:20 and 21:30. Upon arrival, participants completed a questionnaire to verify eligibility and signed a consent form. They then gave their first saliva sample (S-1). Participants in the sober condition were informed that their beverages would not contain alcohol; participants in both the intoxicated and placebo groups were told that their beverages would contain a moderate amount of alcohol. Over the next 10–15 min, participants completed the self-report questionnaires, after which they provided the baseline saliva sample (S0). Participants who were told they would receive alcohol were weighed. Intoxicated group participants' weight was used to determine alcohol dosage of 0.7 g/kg in order to achieve a target blood alcohol level (BAL) of 0.08%. Blood alcohol concentrations were estimated using the Intoxilyzer 400D, a portable instrument that uses a fuel cell to measure the alcohol concentration in breath expired through a mouthpiece. This is simply an estimate of blood alcohol levels, whereas dosage of alcohol serves as the primary index of intoxication.

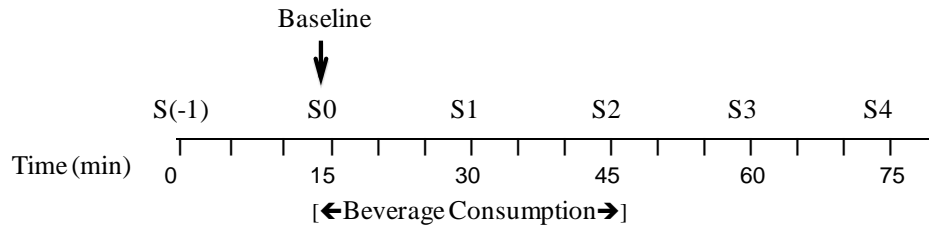


Figure 4-1. Experimental timeline. Diagram shows procedure over time (minutes).

Participants then consumed their beverage, divided into three drinks, one every 15 min, while watching videos of “The Simpsons.” Beverages consisted of two parts calorie-free citrus soda to one part vodka (40% ethanol) for the intoxicated group, and citrus soda plus flattened tonic water for the placebo group. To control for odor and taste cues without increasing BAL, the placebo beverages were rimmed with vodka. Participants in the sober condition consumed calorie-free citrus soda alone. Following each of the three beverages, all participants gave a saliva sample (S1, S2, S3); individuals in the placebo and alcohol condition completed the BAES and gave BAL readings at each time point, although participants were not permitted to see their BAL readings. Following beverage administration, all participants gave a final saliva sample (S4). The intoxicated and placebo groups were administered a manipulation check to assess their perceived level of intoxication.

Finally, participants were debriefed, including revelation of placebo condition, and compensated. BALs of participants in the intoxicated group were measured until their BAL reached 0.06% or below, after which they were sent home in a taxi.

Statistical Analyses.

Data were analyzed using the SPSS version 18 statistical software package. Log transformation was used to normalize data, and outliers were removed when appropriate per Grubbs’ test for statistical outliers. Post hoc analyses were conducted for all analyses of variance (ANOVAs) using Tukey’s *t* tests.

Self-Report Measures.

Only participants who were told they had received alcohol (i.e., alcohol and placebo groups)

completed the BAES. Repeated measures ANOVAs (with Greenhouse-Geisser corrections) were used for BAES scores, with time (three time points) as the within-subjects variable, and group (alcohol vs placebo) as the between-subjects variable.

Relationship between Self-Report Measures.

Collapsing data across groups, Pearson correlation analyses were conducted with the BIS total.

Physiological Responses.

Differences in cortisol and alpha-amylase responses were assessed using percent change from baseline, as previous work from our group has demonstrated that this is more informative than absolute levels in characterizing the biochemical responses (Balodis et al., 2010). To allow for necessary recovery following arrival at the laboratory, the second sample (S0) was used as baseline. A repeated measures ANOVA (with Greenhouse-Geisser corrections) using time point as within subjects variables and group (sober, alcohol, and placebo) as a between-subjects factor were used to examine physiological measures for both cortisol and alpha-amylase over the experimental time course.

Relationship between BAL and Physiological Responses.

Pearson correlation analyses were conducted to explore the relationship between BAL and physiological responses.

Relationship between Self-Report Measures and Physiological Responses.

Collapsing data across groups, Pearson correlation analyses were conducted to explore the relationship between continuous subjective measures (BIS) and both cortisol and alpha-amylase.

Results

Self-Report Measures.

The mean BIS score in our sample was 62.6 ($SD = 9.6$; range = 44–96). A 3 X 2 repeated measures ANOVA examining ratings on the BAES stimulant subscale at the three time points of beverage administration in the alcohol and placebo groups (see Figure 4-2) showed a significant main within-subjects effect of the BAES, $F(2, 100) = 9.27, p < .001$, a main between-groups effect, $F(1, 50) = 28.67, p$

< .001, and a BAES X Condition interaction, $F(2, 100) = 8.64, p < .01$. Specifically, the alcohol group reported higher levels of stimulation following the first drink [$M_{\text{alcohol}} = 20.54 (SD = 13.92)$ vs. $M_{\text{placebo}} = 9.33 (SD = 14.46)$], and continued to report increasing stimulating effects after the second [$M_{\text{alcohol}} = 20.54 (SD = 13.92)$ vs. $M_{\text{placebo}} = 9.71 (SD = 10.34)$] and third [$M_{\text{alcohol}} = 32.86 (SD = 14.85)$ vs. $M_{\text{placebo}} = 9.50 (SD = 10.14)$] drink, whereas the placebo group's ratings of stimulation did not increase over time. For the sedative subscale on the BAES, only the between-groups main effect was significant, $F(1, 50) = 7.89, p < .01$. The alcohol group reported significantly higher levels of sedation relative to the placebo group [$M_{\text{alcohol}} = 10.61 (SD = 10.17)$ vs. $M_{\text{placebo}} = 4.83 (SD = 8.16)$]; however, these ratings did not change significantly over time.

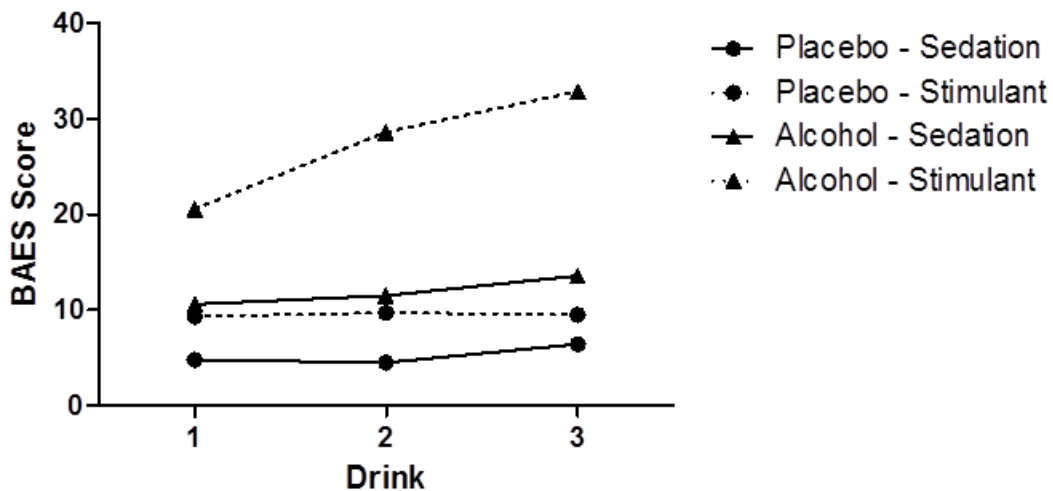


Figure 4-2. BAES scores over time.

Note. Line graph shows BAES scores for the stimulant and sedative subscales in both groups (alcohol and placebo) after each beverage.

Manipulation Check.

The average BAL taken immediately following administration of all beverages was 0.08% ($SD = 0.02$) and 0.00% ($SD = 0.00$) for the alcohol and placebo groups, respectively. When participants were asked to rate their subjective feeling of intoxication on a scale of 1 (*sober*) to 9 (*drunk*), the alcohol group had a mean rating of 5.00 ($SD = 1.37$) and the placebo group a mean rating of 2.50 ($SD = 1.47$). The alcohol group estimated they had consumed the equivalent of 6.21 ($SD = 1.35$) bottles of beer; the

placebo group estimated they had consumed the equivalent of 3.67 ($SD = 1.61$) bottles of beer. The estimations of intoxication and alcohol consumption by the placebo group demonstrate that the placebo manipulation was effective.

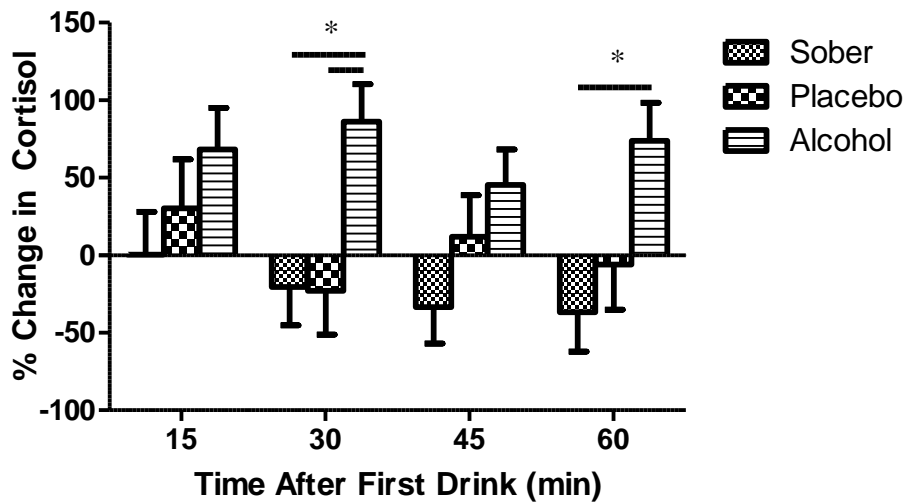
Relationship between Self-Report Measures.

There were no significant correlations between the BIS total score and the BAES ($p > .05$).

Physiological Responses.

Figures 4-3a and 4-3b illustrate the percent change in cortisol and alpha-amylase from baseline over the four subsequent sampling times in the three experimental groups.

(a)



(b)

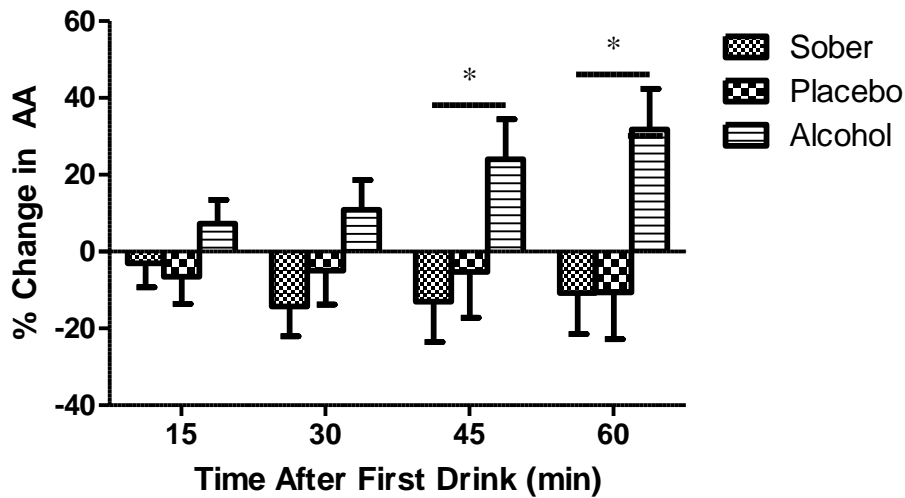


Figure 4-3. Changes in cortisol (a) and alpha-amylase (b) across four sampling points following administration of the first beverage for the 3 beverage groups.

Cortisol.

A repeated measures ANOVA examining time point (4 within-subjects levels) and experimental group (sober, placebo, or intoxicated) for percent change in cortisol from baseline showed a main effect of experimental group, $F(2, 81) = 5.12, p < .01$, whereby the alcohol group had significantly higher cortisol than the sober group ($p < .01$), but not the placebo group ($p > .05$); and these latter two groups did not significantly differ from each other ($p > .05$). There was no significant main effect of time point, $F(3, 243) = 1.50, p > .05$, and no significant experimental group X time point interaction, $F(6, 243) = 1.43, p > .05$.

Alpha-Amylase.

A repeated measures ANOVA examining time point (4 within-subjects levels), and experimental group (sober, placebo, or intoxicated) for percent change in alpha-amylase from baseline showed a main effect of experimental group, $F(2, 81) = 3.43, p < .05$, whereby the alcohol group had marginally higher alpha-amylase than the sober group ($p = .059$), but not the placebo group ($p > .05$); and these latter two groups did not significantly differ from each other ($p > .05$). There was no significant main effect of time point, $F(3, 243) = 0.36, p > .05$, and no significant experimental group X time point interaction, $F(6, 243)$

= 0.75, $p > .05$.

Relationship between BAL and Physiological Responses.

There were no significant correlations between individual BAL collected at four time points and any cortisol samples. BAL levels following the second drink correlated with percent change from baseline in alpha-amylase at S4 ($r = 0.42, p < .05$), S5 ($r = .46, p < .01$), and S6 ($r = .54, p < .01$).

Relationship between Self-Report Measures and Physiological Responses.

There was a significant correlation between percent change from baseline cortisol levels at time 3 and the BIS motor subscale ($r = 0.22, p < .05$). There was also a significant correlation between BIS total scores and percent change in cortisol levels at time 4 ($r = 0.26, p < .05$), which was mostly driven by the motor ($r = 0.29, p < .01$) and the cognitive ($r = 0.21, p = .05$) subscales. The relationship is depicted in a scatter plot in Figure 4-4. A positive correlation between total BIS and cortisol at time 6 ($r = 0.26, p < .05$) was also driven by the motor subscale ($r = 0.28, p = .05$). There were no significant correlations between percent change in alpha-amylase at any time point and BIS scores. Based on percent change in cortisol levels at time 3, we identified a group of 12 high responders, who demonstrated an increase in cortisol levels following alcohol ingestion. These individuals demonstrated a mean increase of 233.13% from baseline ($SD = 200.52$), compared to the other individuals in the intoxicated group who demonstrated a mean *decrease* of 30.72% ($SD = 17.15$). A 2 X 3 repeated measures ANOVA examining the effect of high versus low/average responders on the BAES stimulant subscale after each drink was conducted. There was a significant main within-subjects effect of time point on the BAES, $F(2, 52) = 23.93, p < .01$, and a significant main between-subjects effect of group, $F(1, 26) = 6.53, p < .05$, but no BAES Time Point X Group interaction. Specifically, the high responders reported significantly higher ratings of stimulation relative to the low/average responders, and both groups demonstrated increasing scores of stimulation on the BAES subscale across the three time points. A 2 X 3 repeated measures ANOVA examining high versus low/average responders on the BAES sedation subscale showed no significant differences between the two groups, $F(1, 26) = 0.01, p > .05$.

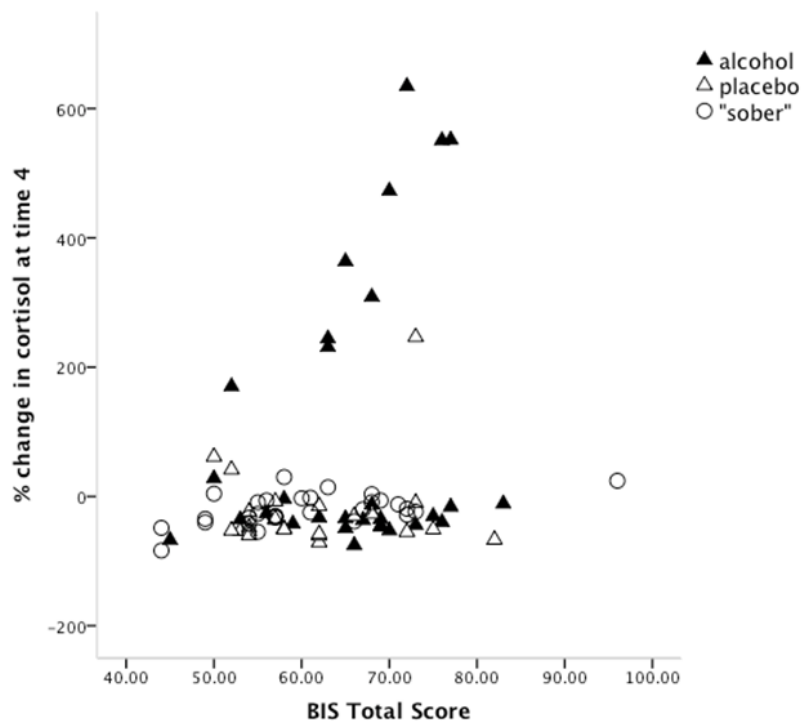


Figure 4-4. Relationship between percent change in cortisol levels and scores on the BIS.

Note. Scatterplot shows a correlation between percent change in cortisol at time 4 and total scores on the BIS ($r = 0.262, p < 0.05$).

Discussion

The primary finding in this study is that alcohol increased both cortisol and alpha-amylase levels in individuals who have prior experience with drinking alcohol while sitting passively watching TV. Our detailed analysis of biochemical changes following alcohol consumption provides a representation of cortisol and alpha-amylase responses over time in healthy young male adults. The profile of alpha-amylase responses in humans is relatively unexplored (Granger, Kivlighan, el-Sheikh, Gordis, & Stroud, 2007), and this is the first description of changes in this enzyme in intoxicated participants with no other experimental manipulation.

Our study did find a weak positive association between impulsivity and cortisol, but not alpha-amylase, responses. Interestingly, trait impulsivity mediates the extent to which cortisol affects dopaminergic activity in frontal circuits (Tops, Wijers, Koch, & Korf, 2006), which helps explain the

relationship between these systems in the context of substance abuse. Important questions for future research are whether trait impulsivity predicts (i.e., predates) subsequent alcohol abuse, and how the interaction between these two variables changes biochemical reactions over time, particularly in adolescents and young adults who consume high amounts of alcohol. The distribution of cortisol responses in the intoxicated sample also highlights that a subset of individuals display very large changes, while others do not. Our exploratory analysis of high responders with BAES scores suggests that these high-responding individuals report more stimulating effects from an intoxicating dose of alcohol. Acute cortisol administration increases subjective vigor among healthy females (Tops, van Peer, Wijers, & Korf, 2006); this suggests that increased cortisol due to alcohol may directly contribute to greater stimulating effects in this male sample. Although our study is restricted by a limited number of these individuals, these results nonetheless highlight that specific subtypes of alcohol responses may underlie discrepancies in the alcohol research regarding alcohol's effects on the stress system. Future studies using larger sample sizes could perform more in-depth analyses examining to what extent differences in impulsivity relate to increased cortisol levels following intoxication and how this might further be associated with the subjective stimulating effects of alcohol. The use of a within-subjects as well as a longitudinal design could further examine directionality of these relationships.

Additionally, characterizing these subtypes may shed more light on why associations between physiological and subjective measures are often ambiguous or not found. In sum, our study demonstrates that alcohol produces marked changes in healthy individuals in cortisol and alpha-amylase, both markers of acute stress responses. Studies that assess alcohol intoxication and stress, therefore, should consider the interactive effect of these two manipulations on both subjective and physiological responses. These findings have implications for both clinical and nonclinical research in that cortisol plays a critical role in cognition in nonpathological populations (Putman, Antypa, Crysovergi, & van der Does, 2010; van den Bos, Harteveld, & Stoop, 2009), and hormonal stress reactivity is linked to a number of psychiatric disorders (Ehlert, Gaab, & Heinrichs, 2001). Our results also highlight the need to explore the relationship

between trait differences and biochemical responses to stress. Understanding the relationship between biochemical responses and alcohol intoxication has direct relevance to addiction research as changes in these measures (at least cortisol) affect prefrontal and limbic brain areas involved in emotion and motivation, and impact both reward-related learning and risk-taking behaviors (Marinelli & Piazza, 2002; Putman et al., 2010; Urry et al., 2006).

References

- Assaad, J. M., Pihl, R. O., Seguin, J. R., Nagin, D. S., Vitaro, F., & Tremblay, R. E. (2006). Intoxicated behavioral disinhibition and the heart rate response to alcohol. *Experimental & Clinical Psychopharmacology, 14*, 377–388. <http://dx.doi.org/10.1037/1064-1297.14.3.377>
- Balodis, I. M., Potenza, M. N., & Olmstead, M. C. (2009). Binge drinking in undergraduates: Relationships with sex, drinking behaviors, impulsivity, and the perceived effects of alcohol. *Behavioural Pharmacology, 20*, 518–526. <http://dx.doi.org/10.1097/FBP.0b013e328330c779>
- Balodis, I. M., Wynne-Edwards, K. E., & Olmstead, M. C. (2010). The other side of the curve: Examining the relationship between pre-stressor physiological responses and stress reactivity. *Psychoneuroendocrinology, 35*, 1363–1373. <http://dx.doi.org/10.1016/j.psyneuen.2010.03.011>
- Balodis, I. M., Wynne-Edwards, K. E., & Olmstead, M. C. (2011). The stress-response-dampening effects of placebo. *Hormones & Behaviour, 59*, 465–472. <http://dx.doi.org/10.1016/j.yhbeh.2011.01.004>
- Blackburn, I. M., Whalley, L. J., Christie, J. E., & Shering, A. (1987). Mood, cognition and cortisol: Their temporal relationships during recovery from depressive illness. *Journal of Affective Disorders, 13*, 31–43. [http://dx.doi.org/10.1016/0165-0327\(87\)90071-1](http://dx.doi.org/10.1016/0165-0327(87)90071-1)
- Brunelle, C., Assaad, J. M., Barrett, S. P., Avila, C., Conrod, P. J., Tremblay, R. E., & Pihl, R. O. (2004). Heightened heart rate response to alcohol intoxication is associated with a reward-seeking personality profile. *Alcoholism: Clinical and Experimental Research, 28*, 394–401. <http://dx.doi.org/10.1097/01.ALC.0000117859.23567.2E>
- Brunelle, C., Barrett, S. P., & Pihl, R. O. (2007). Relationship between the cardiac response to acute intoxication and alcohol-induced subjective effects throughout the blood alcohol concentration curve. *Human Psychopharmacology: Clinical & Experimental, 22*, 437–443. <http://dx.doi.org/10.1002/hup.866>
- Economidou, D., Pelloux, Y., Robbins, T. W., Dalley, J. W., & Everitt, B. J. (2009). High impulsivity predicts relapse to cocaine-seeking after punishment-induced abstinence. *Biological Psychiatry*,

- 65, 851–856. <http://dx.doi.org/10.1016/j.biopsycho.2008.12.008>
- Ehlert, U., Gaab, J., & Heinrichs, M. (2001). Psychoneuroendocrinological contributions to the etiology of depression, posttraumatic stress disorder, and stress-related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. *Biological Psychology*, *57*, 141–152.
[http://dx.doi.org/10.1016/S0301-0511\(01\)00092-8](http://dx.doi.org/10.1016/S0301-0511(01)00092-8)
- Gianoulakis, C., Krishnan, B., & Thavundayil, J. (1996). Enhanced sensitivity of pituitary b-endorphin to ethanol in subjects at high risk of alcoholism. *Archives of General Psychiatry*, *53*, 250–257.
<http://dx.doi.org/10.1001/archpsyc.1996.01830030072011>
- Granger, D., Kivlighan, K., el-Sheikh, M., Gordis, E., & Stroud, L. (2007). Salivary alpha-amylase in biobehavioral research — Recent developments and applications. *Oral-Based Diagnostics*, *1098*, 122–144. <http://dx.doi.org/10.1196/annals.1384.008>
- Heffelfinger, A. K., & Newcomer, J. W. (2001). Glucocorticoid effects on memory function over the human life span. *Developmental Psychopathology*, *13*, 491–513.
<http://dx.doi.org/10.1017/S0954579401003054>
- Holdstock, L., & de Wit, H. (1998). Individual differences in the biphasic effects of ethanol. *Alcoholism: Clinical and Experimental Research*, *22*, 1903–1911. <http://dx.doi.org/10.1111/j.1530-0277.1998.tb05897.x>
- King, A., Munisamy, G., de Wit, H., & Lin, S. (2006). Attenuated cortisol response to alcohol in heavy social drinkers. *International Journal of Psychophysiology*, *59*, 203–209.
<http://dx.doi.org/10.1016/j.ijpsycho.2005.10.008>
- Kirk, J. M., & de Wit, H. (2000). Individual differences in the priming effect of ethanol in social drinkers. *Journal of Studies on Alcohol*, *61*, 64–71. <http://dx.doi.org/10.15288/jsa.2000.61.64>
- Koob, G., & Kreek, M. J. (2007). Stress, dysregulation of drug reward pathways, and the transition to drug dependence. *American Journal of Psychiatry*, *164*, 1149–1159.
<http://dx.doi.org/10.1176/appi.ajp.2007.05030503>

- Kreek, M. J., Nielsen, D. A., Butelman, E. R., & LaForge, K. S. (2005). Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience*, 8, 1450–1457. <http://dx.doi.org/10.1038/nm1583>
- Leyton, M., Boileau, I., Benkelfat, C., Diksic, M., Baker, G., & Dagher, A. (2002). Amphetamine induced increases in extracellular dopamine, drug wanting, and novelty seeking: A PET/ [11C]Raclopride study in healthy men. *Neuropsychopharmacology*, 27, 1027–1035. [http://dx.doi.org/10.1016/S0893-133X\(02\)00366-4](http://dx.doi.org/10.1016/S0893-133X(02)00366-4)
- Marinelli, M., & Piazza, P. V. (2002). Interaction between glucocorticoid hormones, stress and psychostimulant drugs. *European Journal of Neuroscience*, 16, 387–394. <http://dx.doi.org/10.1046/j.1460-9568.2002.02089.x>
- Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M.W., & Swift, R. M. (1993). Development and validation of the Biphasic Alcohol Effects Scale. *Alcoholism: Clinical and Experimental Research*, 17, 140–146. <http://dx.doi.org/10.1111/j.1530-0277.1993.tb00739.x>
- Mitchell, J. M., Fields, H. L., D'Esposito, M., & Boettiger, C. A. (2005). Impulsive responding in alcoholics. *Alcoholism: Clinical and Experimental Research*, 29, 2158–2169. <http://dx.doi.org/10.1097/01.alc.0000191755.63639.4a>
- Moeller, F. G., Dougherty, D. M., Barratt, E. S., Oderinde, V., Mathias, C. W., Harper, A. R., & Swann, A. C. (2002). Increased impulsivity in cocaine dependent subjects in personality disorder and aggression. *Drug and Alcohol Dependence*, 68, 105–111. [http://dx.doi.org/10.1016/S0376-8716\(02\)00106-0](http://dx.doi.org/10.1016/S0376-8716(02)00106-0)
- Morean, M., & Corbin, W. (2010). Subjective response to alcohol: A critical review of the literature. *Alcoholism: Clinical and Experimental Research*, 34, 385–395. <http://dx.doi.org/10.1111/j.1530-0277.2009.01103.x>
- Nater, U. M., La Marca, R., Florin, L., Moses, A., Langhans, W., Koller, M. M., & Ehlert, U. (2006). Stress-induced changes in human salivary alpha-amylase activity—Associations with adrenergic

- activity. *Psychoneuroendocrinology*, *31*, 49–58.
<http://dx.doi.org/10.1016/j.psyneuen.2005.05.010>
- Nater, U. M., & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology*, *34*, 486–496.
<http://dx.doi.org/10.1016/j.psyneuen.2009.01.014>
- Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*, *51*, 768–774. [http://dx.doi.org/10.1002/1097-4679\(199511\)51:6<768::AID-JCLP2270510607>3.0.CO;2-1](http://dx.doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1)
- Putman, P., Antypa, N., Crysovergi, P., & van der Does, W. A. J. (2010). Exogenous cortisol acutely influences motivated decision making in healthy young men. *Psychopharmacology*, *208*, 257–263. <http://dx.doi.org/10.1007/s00213-009-1725-y>
- Rohleder, N., Nater, U. M., Wolf, J. M., Ehlert, U., & Kirschbaum, C. (2004) Psychosocial stress-induced activation of salivary alpha-amylase. *Annals of the New York Academy of Sciences*, *1032*, 258–263. <http://dx.doi.org/10.1196/annals.1314.033>
- Schuckit, M. A., Risch, S. C., & Gold, E. O. (1988). Alcohol consumption, ACTH level, and family history of alcoholism. *American Journal of Psychiatry*, *145*, 1391–1395.
<http://dx.doi.org/10.1176/ajp.145.11.1391>
- Soyka, M., Görig, E., & Naber, D. (1991). Serum prolactin increase induced by ethanol: A dose dependent effect not related to stress. *Psychoneuroendocrinology*, *16*, 441–446.
[http://dx.doi.org/10.1016/0306-4530\(91\)90009-I](http://dx.doi.org/10.1016/0306-4530(91)90009-I)
- Tops, M., van Peer, J. M., Wijers, A. A., & Korf, J. (2006). Acute cortisol administration reduces subjective fatigue in healthy female subjects. *Psychophysiology*, *43*, 653–656.
<http://dx.doi.org/10.1111/j.1469-8986.2006.00458.x>
- Tops, M., Wijers, A. A., Koch, T., & Korf, J. (2006). Modulation of rotational behavior in healthy volunteers by cortisol administration. *Biological Psychology*, *71*, 240–243.

<http://dx.doi.org/10.1016/j.biopsycho.2005.06.007>

- Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thurow, M. E., Schaefer, H. S., & Davidson, R. J. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. *Journal of Neuroscience*, *26*, 4415–4425. <http://dx.doi.org/10.1523/jneurosci.3215-05.2006>
- van den Bos, R., Harteveld, M., & Stoop, H. (2009). Stress and decision-making in humans: Performance is related to cortisol reactivity, albeit differently in men and women. *Psychoneuroendocrinology*, *34*, 1449–1458. <http://dx.doi.org/10.1016/j.psyneuen.2009.04.016>
- Vaschillo, E. G., Bates, M. E., Vaschillo, B., Lehrer, P., Udo, T., Mun, E.Y., & Ray, S. (2008). Heart rate variability response to alcohol, placebo, and emotional cue challenges: Effects of 0.1-Hz stimulation. *Psychophysiology*, *45*, 847–858. <http://dx.doi.org/10.1111/j.1469-8986.2008.00673.x>
- Walter, N. T., Mutic, S., Markett, S., Montag, C., Klein, A. M., & Reuter, M. (2011). The influence of alcohol intake and alcohol expectations on the recognition of emotions. *Alcohol & Alcoholism*, *46*, 680–685. <http://dx.doi.org/10.1093/alcalc/agr082>

Chapter 5

General Discussion

Summary of Findings

In light of the far-reaching consequences and high economic costs associated with excessive drinking (Mohapatra, Patra, Popova, Duhig, & Rehm, 2010; Rehm et al., 1996; Rehm et al., 2009; Roerecke & Rehm, 2013), an important research focus is the examination of vulnerable populations and risk factors related to this behaviour. Thus, the goal of this dissertation was to explore individual differences in intrapersonal factors that confer risk for excessive drinking, particularly disinhibited behaviour and stress. This research focused on undergraduate students, a group that is at high risk for hazardous drinking. The first study (Chapter 2) examined whether trait factors related to disinhibited behaviour (impulsivity, sensation seeking) and stress (anxiety sensitivity) were related to escalated drinking behaviour over the course of the first year of undergraduate studies. Our findings demonstrated that individual differences in trait impulsivity prospectively predicted increases in hazardous drinking and, at a trend level, the amount of alcohol consumed per drinking session. Contrary to expectations, sensation seeking was not a significant predictor of change in drinking behaviour or drinking-related problems during the first year of university; however, this trait variable was significantly correlated with reported drinking onset (first recreational drinking session). In contrast to impulsivity and sensation seeking, anxiety sensitivity was not associated with undergraduates' drinking behaviour (cross-sectionally or prospectively) or with self-reported drinking onset.

The objective of the second experiment (Chapter 3; Magrys & Olmstead, 2015) was, first, to establish whether acute stress increases voluntary alcohol intake in a healthy undergraduate population and, further, to assess whether intake of alcohol is related to individual differences in anxiety. In keeping with our predictions, psychosocial stress increased voluntary intake of alcohol, but not placebo or non-alcoholic beverages. The use of an *ad libitum* laboratory design extends the literature on the stress-

drinking relationship, which is largely comprised of studies that employ retrospective self-reports. To our knowledge, this is the first study to use appropriate controls (i.e., both sober and placebo) to assess the effects of stress on drinking in a non-clinical sample, which allowed us to separate the pharmacological and expectancy effects of alcohol. Using this methodology, we were able to demonstrate that stress-induced beverage consumption is selective to alcohol, suggesting that a pharmacological mechanism underlies this process. In contrast to our hypotheses, individual differences in state and trait anxiety did not predict voluntary alcohol consumption.

Finally, the third study (Chapter 4; Magrys, Olmstead, Wynne-Edwards, & Balodis, 2013) sought to elucidate whether alcohol induces biochemical stress responses in a non-clinical population and, moreover, whether this phenomenon relates to individual differences in impulsivity and subjective alcohol responses. Our data demonstrated that alcohol intoxication was associated with increases in biochemical markers of stress, namely cortisol and alpha amylase, in male university students. These results suggest that alcohol activates endogenous stress systems and, as such, can be viewed as a systemic stressor. Additionally, some biochemical responses to alcohol were related to individual differences in behavioural disinhibition. Specifically, trait impulsivity was positively correlated with cortisol, but not alpha amylase, responses.

Individual Differences in Risk Factors

According to the individual-centered model of AUD, inter-individual differences in trait, behavioural, physiological, or genetic factors underlie variation in risk for development of the disorder (Swendsen & Le Moal, 2011). Among undergraduates, stress and behavioural disinhibition appear to be two of the most important individual difference factors related to alcohol use. With respect to stress, the relationship between stressful life events and increased drinking is well-supported; however, there is a paucity of research examining the effects of acute stress on drinking behaviour. The current dissertation supports a proximal relationship between acute stress and single-session alcohol intake among undergraduates (Chapter 3). The fact that the effect of stress was limited to the alcohol condition suggests

that stress-induced increases in drinking are mediated by the pharmacological effects of the drug, most likely through an action on brain reward systems. In animal models, activation of the stress system (by exogenous or endogenous stressors) stimulates reward circuits via glucocorticoid receptors (for review see Piazza & Le Moal, 1998). Our data support this model in that alcohol increased cortisol and alpha amylase responses (Chapter 4), which are both downstream effects of glucocorticoid activation. Repeated alcohol intoxication, therefore, may contribute to dysregulation of the stress response system, leading to a phenotype that has been consistently associated with AUD.

Moreover, individual differences in subjective responses to stress may moderate the relationship between stressful events and alcohol consumption. Consistent with the Self Medication and Tension Reduction models of alcohol use (Greeley & Oei, 1999; Khantzian, 1999), people may drink to alleviate the negative affective state associated with anxiety. Taken together, the studies in the current dissertation did not provide evidence to support this hypothesis among healthy undergraduates. Instead, our findings demonstrated that, although exposure to stress increased voluntary intake of alcohol, alcohol intake was not significantly associated with individual differences in either state or trait anxiety (Chapter 3). An alternative possibility is that alcohol consumption is motivated by *sensitivity* to the symptoms associated with anxiety, as opposed to anxiety symptoms per se, (Dixon, Stevens, & Viana, 2014; Schmidt, Buckner, & Keough, 2007; Sherry H. Stewart, Zvolensky, & Eifert, 2001). Yet, contrary to this hypothesis, our data showed that anxiety sensitivity was not associated with drinking behaviour among first-year undergraduates and did not predict progression in drinking behaviour during the first several months of university (Chapter 2). These findings suggest that, in non-clinical populations, the relationship between subjective stress response and alcohol use is not straightforward.

Behavioural disinhibition is another factor that has been closely linked to the development of AUD (for review see Verdejo-García, Lawrence, & Clark, 2008), and individual differences in disinhibited trait factors may help explain pre-clinical patterns of drinking behaviour. Indeed, in the current dissertation, trait factors related to behavioural disinhibition were associated with drinking

behaviour in first-year undergraduate students, in that impulsivity and sensation seeking related to all measures of drinking behaviour and drinking-related problems cross-sectionally. In combining retrospective and prospective measures, our study extended this area of research by showing an interesting temporal dissociation between impulsivity and sensation seeking. Specifically, sensation seeking was related to age of drinking onset, whereas impulsivity prospectively predicted escalation in drinking behaviour during the first year of university. Taken together, these data reinforce the importance of distinguishing between disinhibition-related constructs (e.g., impulsivity vs. sensation seeking; Magid, MacLean, & Colder, 2007), suggesting that these factors may have dissociable effects on initial drinking patterns and escalation in drinking, respectively.

The observed dissociation between impulsivity and sensation seeking is consistent with animal literature, which demonstrates that novelty seeking (i.e., sensation seeking) predicts initiation of drug self-administration, whereas trait impulsivity is associated with escalation in drug intake (for review see Bari, Robbins, & Dalley, 2011). Personality theorists posit that individual differences in intrapersonal trait factors are amplified during life transitions, which are “characterized by novelty, ambiguity, and uncertainty” (p. 256; Caspi & Moffitt, 1993); it is plausible that different traits exert different effects on behavior depending on the specific transition event (Gotham, Sher, & Wood, 1997). In keeping with this notion, the effect of sensation seeking may be accentuated during the transition into adolescence (the typical age of drinking onset, as was replicated in the current research), whereas individual differences in impulsivity are more pronounced in the transition to university.

As a whole, the current dissertation lends support to the individual differences model of alcohol use, in that variability in trait factors accounted for some drinking-related behaviours. At face value, our data suggest that behavioural disinhibition is more strongly associated with drinking behaviour than is regulation of negative affect (i.e., anxiety) among healthy undergraduates. It could be the case that the relationship between tension-reduction and drinking behaviour is more pronounced in pathological populations. This relates to the hypothesis that positive reinforcement is most important during the initial

phase of alcohol use, whereas negative reinforcement (e.g., alleviating anxiety) becomes increasingly important as individuals progress to the compulsive drinking that typifies AUD (for overview see Koob & Volkow, 2010). Indeed, a positive relationship between self-reported symptoms of anxiety and alcohol use is not consistently supported in non-clinical populations, including undergraduates, with some studies even demonstrating an inverse relationship (e.g., Morris, Stewart, & Ham, 2005). The state of the animal literature is similar, with current researchers stating that “while there is a substantial amount of high-quality work on stress and its relation to drug abuse, more studies are needed on the role of stress in acquisition of drug self-administration” (p. 255; Carroll & Meisch, 2011). Additionally, recent findings suggest that more proximal factors, such as coping style, drinking motives, and alcohol expectancies, may moderate drinking behaviour (Armeli, Conner, Cullum, & Tennen, 2010; Fitzgerald & Long, 2012; Morris et al., 2005).

Interaction between Risk Factors

Models of risk for alcoholism are not mutually exclusive (Sher & Trull, 1994), because different individuals may be influenced to drink by different factors. Similarly, for any given individual, multiple risk factors may be at play and, moreover, these factors may interact. More specifically, animal and human research has demonstrated that drinking behaviour is influenced by interactions between impulsivity and stress (Carroll et al., 2011; De Wit, 2009; Piazza & Le Moal, 1996). In the current dissertation, stress selectively increased the consumption of alcohol (Chapter 3); we proposed that this effect was likely related to pharmacological ‘priming’ by the initial dosage of alcohol (Chutuape, Mitchell, & de Wit, 1994; Field, Wiers, Christiansen, Fillmore, & Verster, 2010; Rose, Hobbs, & Drummond, 2013), which suppresses inhibitory control and, in turn, leads to increased drinking (Christiansen, Rose, Cole, & Field, 2013; Weafer & Fillmore, 2008). Consistently, an initial dose of alcohol increases craving, attentional bias for alcohol cues, and *ad libitum* alcohol intake among moderate social drinkers (Christiansen et al., 2013; Fernie, Christiansen, Cole, Rose, & Field, 2012; Schoenmakers,

Wiers, & Field, 2008). The fact that alcohol intake was more pronounced in the stress condition (Chapter 3) highlights a possible interaction between stress and impulsivity in predicting alcohol consumption.

Additionally, stress may exert its influence on drinking behaviour by altering subjective responses to alcohol (Corbin, Gearhardt, & Fromme, 2008; Söderpalm & de Wit, 2002), thereby enhancing the reinforcing properties of the drug. For example, individuals who report greater stimulant-like responses to alcohol report reduced stimulation, increased sedation, and a greater desire to drink when alcohol intake is preceded by exposure to acute stress (Childs et al., 2011). In our study, individuals with more pronounced neuroendocrine responses to alcohol reported a greater stimulant response (Chapter 4); it stands to reason that these individuals may be motivated to drink more alcohol following a stressor because their typical subjective experience (i.e., stimulation) is diminished. In this way, our data support an interaction between the endogenous stress system and subjective alcohol responses. Consistent with this interpretation, stimulant effects after a priming dose of alcohol predict within-session drinking (Corbin et al., 2008). Thus, our finding of increased *ad libitum* consumption of alcohol following stress (Chapter 3) may also relate to an interaction between stress and the subjective effects of alcohol.

Interactions between multiple factors may also influence alcohol consumption. In fact, given the complex and multifactorial nature of drinking and AUD, these types of interactions are most likely at play. Consistent with this notion, our findings demonstrated that more pronounced neuroendocrine responses to alcohol were associated, not only with greater subjective effects (specifically stimulation), but also with trait impulsivity (Chapter 4). Additionally, as previously mentioned, the ‘priming’ effect of alcohol has been shown to relate to both the stimulating effects of alcohol (Corbin et al., 2008) and diminished executive control (Christiansen et al., 2013), thus both factors likely interacted with stress to promote *ad libitum* drinking in our study (Chapter 3).

Revisiting the Concept of Alcoholic Personality

Historically, society held the view that alcoholism is caused by (a) individuals' moral and/or religious deficiencies, and their *desire* to drink, or (b) alcohol itself, in the sense that any exposure to alcohol is sufficient to lead to excessive drinking (White, 2000). However, the beginning of the 19th century marked an important paradigm shift, with the emergence of the notion that alcoholism is a progressive medical disease (Levine, 1978). The Disease Theory of Alcoholism was markedly different from past viewpoints as it introduced alcoholism as an addiction, which inferred the loss of voluntary control, and moved away from the idea of alcoholism reflecting a lack of will power or deficient moral character (Shaffer, 1985). Subsequent iterations of the Disease Theory of Alcoholism moved away from the assertion that alcohol per se was the root cause of the disorder and towards the view that something within individuals leads to a loss of control over drinking (Jellinek, 1960). A corollary of the updated Disease Model was the notion that, as with other chronic diseases, individuals exhibit differential risk for developing alcoholism.

Over the subsequent decades, significant efforts were aimed at identifying an 'alcoholic' or 'addictive' personality – a particular group of traits that typify individuals who develop substance use disorder. After hundreds of studies, researchers failed to identify a premorbid personality facet or cluster of traits that fully accounted for the development or maintenance of alcoholism. As a result, beginning in the 1970's, the scientific community began to discard the idea of a singular 'addictive personality' (Kerr, 1996; Ogborne, 2004).

Despite the fact that the construct of an 'addictive personality' was not borne out in the literature, efforts aimed at validating this construct were not in vain. In particular, a large body of evidence has amassed to support the general notion that personality is an important facet in understanding alcohol use (for reviews see McGue, Slutske, & Iacono, 1999; Mulder, 2002). Thus, we have essentially refined our idea of how personality predicts AUD; whereas earlier 'addictive personality' research focused on distinctions in traits between two groups (i.e., addicted vs. non-addicted individuals), more recent

research has shifted toward an individual differences approach. Current literature indicates that, across personality traits, variation in behavioural disinhibition is the most robustly associated with AUD (for review see Littlefield & Sher, 2010). Moreover, behavioural undercontrol is also a significant predictor of addiction to other substances of abuse, as well other mental health disorders, such as conduct disorder (McGue et al., 1999; Slutske et al., 2002). The findings of the current dissertation generally align with these findings, in that individual differences in self-reported behavioural disinhibition were associated with increased neuroendocrine response to alcohol and increased hazardous drinking during the first year of university, whereas other trait factors that were examined (e.g., anxiety sensitivity) showed no relationship with drinking behaviour.

As the study of trait factors in the context of alcoholism has evolved over time, the complexity of related hypotheses and methodological approaches has also expanded. An important question that has arisen is whether trait differences associated with alcoholism are premorbid, or develop as a result of the addiction (Kerr, 1996). Studies have attempted to address this issue by examining pre-clinical populations and by using longitudinal design, both of which were employed in the current dissertation. Additionally, the notion of stability in trait factors has become more flexible (Kerr, 1996), particularly in the context of state-trait theory, which reasons that stable psychological constructs (i.e., traits), such as behavioural disinhibition, are affected by dynamic situational factors, or states (Steyer et al., 1999). The current dissertation addressed this distinction by including both dynamic state measures (e.g., state anxiety, neuroendocrine responsivity), as well as trait measures (e.g., trait anxiety, anxiety sensitivity).

The study of trait factors and drinking behaviour has also expanded to include genetic factors. In 1972, a consensus committee involving the National Council on Alcoholism and Drug Dependence and American Society of Addiction Medicine put forward a revised definition of alcoholism (Kaim et al., 1972), which improved on prior conceptualizations of alcoholism by describing the heterogeneous and multifactorial nature of the disease, and recognizing the role of genetic vulnerability (Morse & Flavin, 1992). Since then, genetic analysis of alcoholism has grown exponentially, and studies have consistently

implicated genetic factors in the development of the disorder. Alcoholism exhibits moderate to high *heritability*, which refers to the estimated proportion phenotype variation that is attributable to genetic variation, as opposed to environmental (i.e., non-genetic) factors (Goldman, Oroszi, & Ducci, 2005; Kreek, Nielsen, & LaForge, 2004). Notably, the heritability rates of alcoholism (40-70%) are comparable to other medical diseases such as diabetes (McLellan, Lewis, O'Brien, & Kleber, 2000), which further supports the Disease Model of alcoholism.

Many genetic polymorphisms that have been associated with AUD also confer genetic risk for abuse of other substances, suggesting the existence of common genetic diathesis for addiction (Kreek, Nielsen, Butelman, & LaForge, 2005; Kreek et al., 2004). However, genotyping has also been helpful in distinguishing between pathways to alcohol abuse, by providing yet another level of individual-differences analysis. Genetic polymorphisms related to disinhibited behaviour and stress have been put forward as particularly promising avenues of genetic research (Goldman et al., 2005; Kreek, Nielsen, Butelman, & LaForge, 2005). In fact, genetic variance related to these personality dimensions account for a significant portion of the genetic risk for alcoholism (Slutske et al., 2002). Further genetic studies would be useful in following up the findings of the current dissertation. For example, genetic polymorphisms related to stress reactivity (Koob, 2008; Kreek et al., 2005), such as the alleles of the catechol-o-methyltransferase (COMT) gene, may moderate stress-induced voluntary consumption of alcohol.

Role of Alcohol Expectancy

In addition to increasing understanding of risk factors for excessive drinking among undergraduates, the current dissertation added to our knowledge regarding the effects of alcohol expectancy, or the *belief* that one has consumed alcohol. According to the Alcohol Expectancy Theory (Goldman, Brown, & Christiansen, 1987), observable drinking-related behaviour is a combination of the pharmacological effects of alcohol and expectancy regarding its anticipated effects. Unfortunately, much of the laboratory research involving alcohol administration fails to adequately parse these effects. Many alcohol administration studies explicitly inform participants about the possibility of receiving a *placebo*

beverage, which arguably has the potential to attenuate the ‘power of suggestion’ (Testa et al., 2006). Additionally, studies often do not assess participants’ belief that they consumed alcohol (for meta-analysis regarding use of placebo manipulations in alcohol challenge studies see Schlauch et al., 2010), so there is no verification that the placebo protocol was effective. Perhaps most critically, alcohol administration studies often include only a placebo comparison group in addition to the experimental alcohol group (e.g., Abrams & Kushner, 2004; Abrams, Kushner, Medina, & Voight, 2002; Chutuape & de Wit, 1995; Corbin et al., 2008; King, de Wit, McNamara, & Cao, 2011; Söderpalm, Nikolayev, & De Wit, 2003; Zimmermann et al., 2004). In alcohol-versus-placebo designs, any significant differences between the two groups can be attributed to pharmacological effects; however, when no difference is observed, the findings become difficult to interpret, as several possibilities can be put forth to explain the pattern of results (e.g., the placebo and alcohol effects are commensurate, there is a generalized effect owing to a third variable, etc.). In contrast, some research groups include both sober and placebo groups (Christiansen et al., 2013; MacDonald, Fong, Zanna, & Martineau, 2000), which is currently considered to be the methodological ideal in alcohol challenge studies (Schlauch et al., 2010). In keeping with this best-practice design, the laboratory studies in the current dissertation (i.e., Chapters 3 and 4) were able to distinguish between the pharmacological and expectancy effects of alcohol. In the sober condition, participants are aware they have consumed an inactive substance and do not anticipate alcohol-related changes; thus, when contrasted with the placebo condition, the sober control group allows the separation of anticipated effects.

Our placebo manipulation was robustly effective, as verified by self-report measures of intoxication. Importantly, our findings demonstrated that stress increased *ad libitum* consumption of alcohol, but not placebo beverage. These findings suggest that the observed results are due to pharmacological, as opposed to anticipated, effects of alcohol, which lends support to the Pharmacological Vulnerability Model of alcohol use (Sher et al., 2005), rather than the Medication Hypotheses (Khantzian, 1999). Previous research has found that both placebo and alcohol increase

alcohol craving and automatic approach to alcohol-related cues (Christiansen et al., 2013; Schoenmakers et al., 2008), whereas only an initial dose of alcohol promotes *ad libitum* drinking (Christiansen et al., 2013; Fernie et al., 2012). Thus, alcohol approach behaviour appears to be mediated by the anticipated and pharmacological effects of alcohol, whereas increased voluntary intake is limited to the pharmacological effects, as was demonstrated in our study. This distinction may not be surprising when considering the fact that antecedent behaviours related to alcohol use are separable. For example, attentional bias for alcohol-related cues does not predict subsequent alcohol-approach behaviour (Fernie et al., 2012).

Additionally, the present research showed that, in both the placebo and alcohol groups, volume of voluntary beverage intake positively correlated with liking the effects and wanting more; this is the first study, to our knowledge, to demonstrate a dose-dependent placebo effect on positive subjective responses to alcohol. Additionally, among participants who believed that they had received alcohol (i.e., the alcohol and placebo groups), there was a lack of association between how much beverage they consumed and their estimated BAL. This finding suggests that the physiological cues secondary to alcohol intoxication do not provide an appreciable advantage in helping individuals estimate their level of intoxication. However, this finding could also be an artefact of the metric employed, in the sense that BAL may be a difficult concept for individuals to estimate.

Our data also demonstrated that, while alcohol increased neuroendocrine stress responses, the role of anticipated effects of alcohol was nuanced. Specifically, in terms of neuroendocrine responses, the placebo group did not differ significantly from alcohol or sober groups. In the absence of a sober control group – as is the case with most alcohol administration studies – we may have concluded that the expectancy and pharmacological effects are commensurate. However, the fact that the placebo group did not differ significantly from the sober control, whereas the alcohol group did, allowed us to determine that neuroendocrine stress reactivity interacts primarily with the pharmacological effects of alcohol.

Limitations and Future Research

The current studies are limited by several factors, such as the relatively small sample size. With greater power, we may have been able to detect additional effects, such as those related to expectancy, alcohol dosage, and gender. For example, it is possible that the low number of men in the online study dampened our ability to detect a relationship between drinking behaviour and anxiety sensitivity, which has been shown to differ between men and women (Stewart, Taylor, & Baker, 1997). Additionally, all three studies in the current dissertation focused on healthy, undergraduate samples, and one study was limited to men, which limits the generalizability of our findings. That being said, we were particularly motivated to study undergraduate students given their heavy drinking patterns and related risk for AUD; thus, the current findings have value to the extent that they are unique to this population. Nonetheless, these studies should be replicated in the general population, and within a variety of clinical samples, in order to examine the findings more broadly.

Related to the concept of generalizability, ecological validity should also be considered when interpreting the findings of the current studies. Although significant efforts were made to employ ecologically valid protocols and measurement tools (e.g., laboratory designed to simulate a living room environment, drinking with confederates, etc.), there are aspects of the research that fell short in this regard. For example, some of the questionnaires employed are not particular to undergraduate samples (e.g., BIS), and the drinking protocols we employ do not accurately simulate a typical duration or style of drinking among undergraduates. Additionally, participants may have behaved differently in the laboratory or responded to the questionnaires in a biased manner because they knew their drinking behaviour was being studied (i.e., demand characteristics). This type of bias, either conscious or unconscious, may attenuate extremes in behavioural and/ or reported drinking behaviour. These shortcomings can be addressed by future studies employing *in vivo* or naturalistic designs, which have greater external validity.

Another potential limitation of the current work relates to the fact that advertisements and letters of information for our behavioural and survey studies explicitly stated that the research being conducted

was related to alcohol. Thus, recruitment bias may have affected our participant samples in a systematic way. For example, individuals who engage in frequent or heavy alcohol use may be more likely to volunteer for laboratory studies involving alcohol administration. Recently, studies have begun using the *alternative substance paradigm* (Conrad, McNamara, & King, 2012), wherein participants are informed that the beverage they receive may contain one of many substances, such as a stimulant, sedative, alcohol, placebo, or a combination of these substances. This is a potential approach to circumventing recruitment bias.

Conclusions and Practical Implications

Despite the noted limitations, the findings of the current dissertation have practical implications. Taken together, the current studies underscore the influence of disinhibited behaviour on subjective effects of alcohol (i.e., stimulation) and drinking behaviour among undergraduates. Behavioural disinhibition would be an appropriate screening measure for hazardous drinking, which could be used to identify students at risk for this behavior. Moreover, interventions aimed at reducing behavioural disinhibition may help to manage drinking behaviour in this population. Encouragingly, the literature regarding the efficacy of targeted interventions focused on intrapersonal risk factors is promising. For example, brief cognitive-behavioural interventions targeting ‘disinhibition-sensation seeking’ effectively reduces risky drinking and alcohol-related problems among students, compared to a no-intervention control (Conrod, Stewart, Comeau, & Maclean, 2006).

Given our finding that impulsivity and sensation seeking had dissociable effects on drinking behaviour, it is advisable that screening and intervention programs assess these traits as distinct constructs, as opposed to conceptualizing disinhibited behaviour more generally. Notably, these separable constructs influence drinking behaviour in measurably distinct ways, in that sensation seeking appears to exert its effects on alcohol intake primarily via enhancement motives (i.e., to enhance an already-positive emotional state), whereas impulsivity relates more to alcohol-related problems (as opposed to alcohol use per se) and this effect is mediated, primarily, by coping motives (i.e., to alleviate an aversive emotional

state; Magid et al., 2007). These findings map well onto our data: sensation seeking related to initial alcohol involvement, whereas impulsivity was associated with progression in hazardous drinking during a transitional period characterized by novelty and increased social and academic demands (Robotham & Julian, 2006).

Our data suggest that early intervention efforts at the undergraduate level should focus on impulsivity, as sensation seeking may be more closely related to establishment of early drinking patterns that predate university. Such considerations are necessary in public sector settings, such as universities, where resources, funding, and time are often limited. Given that the construct of impulsivity includes rapid, unplanned responding and acting without consideration of consequences, it is likely the case that students who are high on this trait factor opt for immediate-reward activities, such as drinking, instead of choosing more adaptive alternatives for coping. As such, early interventions should be aimed at providing healthy coping strategies and encouraging their use as an alternative to drinking. Additionally, interventions should ideally incorporate a certain degree of harm reduction by educating students on appropriate contexts for drinking. This is especially important for impulsive individuals, who may be apt to drink in situations that are unsafe (e.g., prior to operating a motor vehicle) or inadvisable (e.g., the night before an early exam).

Our data also demonstrated that undergraduate students increase their voluntary alcohol intake in response to acute stress, irrespective of their level of subjective anxiety or anxiety sensitivity. Thus, interventions aimed at reducing drinking among undergraduates should incorporate education about the effect of stress on single-session drinking, regardless of whether or not one actually feels subjectively stressed. Students should also be educated about the fact that placebo beverages are as effective as alcohol in reducing physiological and psychological stress responses (Balodis, Wynne-Edwards, & Olmstead, 2011), which will help to challenge preconceptions about alcohol's ability to allay tension. Universities might also provide information and/or on-campus opportunities to engage in stress-relieving activities that are healthier alternatives to drinking, such as yoga, particularly around high-stress periods like exam

week. Universal (broad, education-based) interventions have historically been less effective than targeted interventions (Foxcroft, Ireland, Lowe, & Breen, 2011), so if this approach is used for educational campaigns, administrators should make use of factors that have been shown to moderate the effectiveness of on-campus drinking interventions. For example, peer influences have considerable bearing on undergraduate students' patterns of alcohol use (Borsari & Carey, 2001; Perkins, 2007) as well as their willingness to initiate and maintain reductions in drinking following brief interventions (Reid, Carey, Merrill, & Carey, 2015). Thus, universities might benefit from seeking buy-in for interventions at a peer-group level, through residency advisors or grassroots outreach approaches (i.e., providing education in naturalistic settings where peer groups socialize). Some research suggests that counsellor-delivered brief interventions are more effective than those disseminated via computer (Monahan et al., 2013). Additionally, it may be helpful for universities to implement mandated means of discouraging drinking during high-stress periods, such as the closure of campus bars or increased monitoring of alcohol use in student on-campus residences during exam time.

In conclusion, the current dissertation contributes to knowledge about inter-individuals differences that confer risk for excessive drinking among undergraduates, a vulnerable population in the context of alcohol consumption. Given the persistent, treatment-refractory nature of alcoholism, there is great benefit to identifying risk factors and implementing early interventions before drinking behavior escalates to clinical thresholds.

References

- Aalto, M., Alho, H., Halme, J. T., & Seppä, K. (2009). AUDIT and its abbreviated versions in detecting heavy and binge drinking in a general population survey. *Drug and Alcohol Dependence, 103*(1-2), 25–29. <http://doi.org/10.1016/j.drugalcdep.2009.02.013>
- Abrams, K., & Kushner, M. G. (2004). The moderating effects of tension-reduction alcohol outcome expectancies on placebo responding in individuals with social phobia. *Addictive Behaviors, 29*(6), 1221–1224. <http://doi.org/10.1016/j.addbeh.2004.03.020>
- Abrams, K., Kushner, M. G., Medina, K. L., & Voight, A. (2002). Self-administration of alcohol before and after a public speaking challenge by individuals with social phobia. *Psychology of Addictive Behaviors, 16*(2), 121–128. <http://doi.org/10.1037/h0092445>
- Armeli, S., Conner, T. S., Cullum, J., & Tennen, H. (2010). A longitudinal analysis of drinking motives moderating the negative affect-drinking association among college students. *Psychology of Addictive Behaviors, 24*(1), 38–47. <http://doi.org/10.1037/a0017530>
- Baer, J. S. (2002). Student factors: understanding individual variation in college drinking. *Journal of Studies on Alcohol, S1986*(14), 40–53.
- Balodis, I. M., Potenza, M. N., & Olmstead, M. C. (2009). Binge drinking in undergraduates: Relationship with gender, drinking behaviors, impulsivity and the perceived effects of alcohol. *Behavioral Pharmacology, 20*(5-6), 518–526. <http://doi.org/10.1097/FBP.0b013e328330c779>.Binge
- Balodis, I. M., Wynne-Edwards, K. E., & Olmstead, M. C. (2011). The stress-response-dampening effects of placebo. *Hormones and Behavior, 59*(4), 465–472. <http://doi.org/10.1016/j.yhbeh.2011.01.004>

- Bari, A., Robbins, T., & Dalley, J. (2011). Impulsivity. In M. C. Olmstead (Ed.), *Animal models of drug - addiction* (pp. 379–401). New York, NY: Springer Science.
- Barr, C. S., & Goldman, D. (2006). Non-human primate models of inheritance vulnerability to alcohol use disorders. *Addiction*, 374–385. <http://doi.org/10.1111/j.1369-1600.2006.00034.x>
- Blanchard, M. M., Mendelsohn, D., & Stamp, J. A. (2009). The HR/LR model: Further evidence as an animal model of sensation seeking. *Neuroscience and Biobehavioral Reviews*, 33(7), 1145–1154. <http://doi.org/10.1016/j.neubiorev.2009.05.009>
- Boden, J. M., Fergusson, D. M., & Horwood, L. J. (2014). Associations between exposure to stressful life events and alcohol use disorder in a longitudinal birth cohort studied to age 30. *Drug and Alcohol Dependence*, 142, 154–160. <http://doi.org/10.1016/j.drugalcdep.2014.06.010>
- Borsari, B., Boyle, K. E., Hustad, J. T. P., Barnett, N. P., O’Leary Tevyaw, T., & Kahler, C. W. (2007). Drinking before drinking: Pregaming and drinking games in mandated students. *Addictive Behaviors*, 32(11), 2694–2705. <http://doi.org/10.1016/j.addbeh.2007.05.003>
- Borsari, B., & Carey, K. B. (2001). Peer influences on college drinking: A review of the research. *Journal of Substance Abuse*, 13(4), 391–424. [http://doi.org/10.1016/S0899-3289\(01\)00098-0](http://doi.org/10.1016/S0899-3289(01)00098-0)
- Borsari, B., Murphy, J. G., & Barnett, N. P. (2007). Predictors of alcohol use during the first year of college: Implications for prevention. *Addictive Behaviors*, 32(10), 2062–2086. <http://doi.org/10.1016/j.addbeh.2007.01.017>
- Bouchery, E. E., Harwood, H. J., Sacks, J. J., Simon, C. J., & Brewer, R. D. (2011). Economic costs of excessive alcohol consumption in the U.S., 2006. *American Journal of Preventive Medicine*, 41(5), 516–524. <http://doi.org/10.1016/j.amepre.2011.06.045>

- Brache, K., & Stockwell, T. (2011). Drinking patterns and risk behaviors associated with combined alcohol and energy drink consumption in college drinkers. *Addictive Behaviors*, *36*(12), 1133–1140. <http://doi.org/10.1016/j.addbeh.2011.07.003>
- Brunelle, C., & Pihl, R. O. (2007). Effects of conditioned reward and nonreward cues on the heart rate response to alcohol intoxication in male social drinkers. *Alcoholism: Clinical and Experimental Research*, *31*(3), 383–389. <http://doi.org/10.1111/j.1530-0277.2006.00318.x>
- Butt, P., Beirness, D., Stockwell, T., Gliksman, L., & Paradis, C. (2011). *Alcohol and Health in Canada : A Summary of Evidence and Guidelines for Low-Risk Drinking*.
- Carroll, M. E., & Meisch, R. (2011). Acquisition of drug self-administration. In M. C. Olmstead (Ed.), *Animal models of drug addiction* (pp. 237–265). New York, NY: Springer Science.
- Caspi, A., & Moffitt, T. E. (1993). When do individual differences matter? A paradoxical theory of personality coherence. *Psychological Inquiry*, *4*(4), 247–271. http://doi.org/10.1207/s15327965pli0404_1
- Chassin, L., Forthofer, D. B., & King, K. M. (2004). Trajectories of alcohol and drug use and dependence from adolescence to adulthood: the effects of familial alcoholism and personality. *Journal of Abnormal Psychology*, *113*(4), 483–498. <http://doi.org/10.1037/0021-843X.113.4.483>
- Childs, E., O'Connor, S., & de Wit, H. (2011). Bidirectional interactions between acute psychosocial stress and acute intravenous alcohol in healthy men. *Alcoholism: Clinical and Experimental Research*, *35*(10), 1794–1803. <http://doi.org/10.1111/j.1530-0277.2011.01522.x>
- Christiansen, P., Rose, A. K., Cole, J. C., & Field, M. (2013). A comparison of the anticipated and pharmacological effects of alcohol on cognitive bias, executive function, craving and ad-lib drinking. *Journal of Psychopharmacology*. <http://doi.org/10.1177/0269881112450787>

- Chrousos, G. P. (1998). Stressors, stress and neuroendocrine integration of the adaptative response. *Annals of the New York Academy of Sciences*, 851, 331-335. <http://doi.org/10.1111/j.1749-6632.1998.tb09006.x>
- Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *The Journal of the American Medical Association*, 267(9), 1244–1252. <http://doi.org/10.1001/jama.267.9.1244>
- Chutuape, M. A. D., & de Wit, H. (1995). Preferences for ethanol and diazepam in anxious individuals: an evaluation of the self-medication hypothesis. *Psychopharmacology*, 121(1), 91–103. <http://doi.org/10.1007/BF02245595>
- Chutuape, M. A. D., Mitchell, S. H., & de Wit, H. (1994). Ethanol preloads increase ethanol preference under concurrent random-ratio schedules in social drinkers. *Experimental and Clinical Psychopharmacology*, 2(4), 310–318. <http://doi.org/10.1037/1064-1297.2.4.310>
- Cippitelli, A., Damadzic, R., Singley, E., Thorsell, A., Ciccocioppo, R., Eskay, R. L., & Heilig, M. (2012). Pharmacological blockade of corticotropin-releasing hormone receptor 1 (CRH1R) reduces voluntary consumption of high alcohol concentrations in non-dependent Wistar rats. *Pharmacology Biochemistry and Behavior*, 100(3), 522–529. <http://doi.org/10.1016/j.pbb.2011.10.016>
- Collins, R. L., Kashdan, T. B., Koutsky, J. R., Morsheimer, E. T., & Vetter, C. J. (2008). A self-administered Timeline Followback to measure variations in underage drinkers' alcohol intake and binge drinking. *Addictive Behaviors*, 33(1), 196–200. <http://doi.org/10.1016/j.addbeh.2007.07.001>
- Conrad, M., McNamara, P., & King, A. (2012). Alternative substance paradigm: Effectiveness of beverage blinding and effects on acute alcohol responses. *Experimental and Clinical Psychopharmacology*, 20(5), 382–389. <http://doi.org/10.1037/a0029261>

- Conrod, P. J., Stewart, S. H., Comeau, N., & Maclean, a M. (2006). Efficacy of cognitive-behavioral interventions targeting personality risk factors for youth alcohol misuse. *Journal of Clinical Child and Adolescent Psychology*, 35(4), 550–563. http://doi.org/10.1207/s15374424jccp3504_6
- Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: a motivational model of alcohol use. *Journal of Personality and Social Psychology*, 69(5), 990–1005. <http://doi.org/10.1037/0022-3514.69.5.990>
- Corbin, W. R., Gearhardt, A., & Fromme, K. (2008). Stimulant alcohol effects prime within session drinking behavior. *Psychopharmacology*, 197(2), 327–337. <http://doi.org/10.1007/s00213-007-1039-x>
- Corbin, W. R., Scott, C., Leeman, R. F., Fucito, L. M., Toll, B. a., & O'Malley, S. S. (2013). Early subjective response and acquired tolerance as predictors of alcohol use and related problems in a clinical sample. *Alcoholism: Clinical and Experimental Research*, 37(3), 490–497. <http://doi.org/10.1111/j.1530-0277.2012.01956.x>
- Courtney, K. E., & Polich, J. (2009). Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin*, 135(1), 142–156. <http://doi.org/10.1037/a0014414>.Binge
- Crews, F. T., & Boettiger, C. A. (2009). Impulsivity, frontal lobes and risk for addiction. *Pharmacology Biochemistry and Behavior*, 93(3), 237–247. <http://doi.org/10.1016/j.pbb.2009.04.018>
- Dawson, D. A., Grant, B. F., & Ruan, W. J. (2005). The association between stress and drinking: Modifying effects of gende and vulnerability. *Alcohol and Alcoholism*, 40(5), 453–460. <http://doi.org/10.1093/alcalc/agh176>

Dawson, D. A., Grant, B. F., Stinson, F. S., Chou, P. S., Huang, B., & Ruan, W. J. (2005). Recovery from DSM-IV alcohol dependence: United States, 2001-2002. *Addiction*, *100*(3), 281–292.

<http://doi.org/10.1111/j.1360-0443.2004.00964.x>

de Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, *14*(1), 22–31. <http://doi.org/10.1111/j.1369-1600.2008.00129.x>

Del Boca, F. K., Darkes, J., Greenbaum, P. E., & Goldman, M. S. (2004). Up close and personal: temporal variability in the drinking of individual college students during their first year. *Journal of Consulting and Clinical Psychology*, *72*(2), 155–164. <http://doi.org/10.1037/0022-006X.72.2.155>

Dick, D. M., Smith, G., Olausson, P., Mitchell, S. H., Leeman, R. F., O'Malley, S. S., & Sher, K. (2010). Understanding the construct of impulsivity and its relationship to alcohol use disorders. *Addiction Biology*, *15*(2), 217–226. <http://doi.org/10.1111/j.1369-1600.2009.00190.x>

Dixon, L. J., Stevens, E. N., & Viana, A. G. (2014). Anxiety sensitivity as a moderator of the relationship between trait anxiety and illicit substance use. *Psychology of Addictive Behaviors*, *28*(4), 1284–1289.

Esser, M. B., Kanny, D., Brewer, R. D., & Naimi, T. S. (2012). Binge drinking intensity: A comparison of two measures. *American Journal of Preventive Medicine*, *42*(6), 625–629.

<http://doi.org/10.1016/j.amepre.2012.03.001>

Fernie, G., Christiansen, P., Cole, J. C., Rose, A. K., & Field, M. (2012). Effects of 0.4g/kg alcohol on attentional bias and alcohol-seeking behaviour in heavy and moderate social drinkers. *Journal of Psychopharmacology*, *26*(7), 1017–1025. <http://doi.org/10.1177/02698811111434621>

Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking.

Alcoholism: Clinical and Experimental Research, 34(8), 1346–1352. <http://doi.org/10.1111/j.1530-0277.2010.01218.x>

Fitzgerald, K.-A., & Long, C. G. (2012). The influence of drinking motives and anxiety on low-, moderate- and high-risk drinkers. *Journal of Substance Use*, 17(3), 240–248. <http://doi.org/10.3109/14659891.2011.565109>

Fromme, K., Corbin, W. R., & Kruse, M. I. (2008). Behavioral risks during the transition from high school to college. *Developmental Psychology*, 44(5), 1497–1504. <http://doi.org/10.1037/a0012614>

Gianoulakis, C. (1998). Alcohol-seeking behavior. *Alcohol Health & Research World*, 22(3), 202–210.

Gmel, G., Kuntsche, E., & Rehm, J. (2011). Risky single-occasion drinking: Bingeing is not bingeing. *Addiction*, 106(6), 1037–1045. <http://doi.org/10.1111/j.1360-0443.2010.03167.x>

Goldman, D., Oroszi, G., & Ducci, F. (2005). The genetics of addictions: Uncovering the genes. *Nature Reviews Genetics*, 6(7), 521–532. <http://doi.org/10.1038/nrg1635>

Goldman, M. S., Brown, S. A., & Christiansen, B. A. (1987) In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (pp. 181–226). New York: Guilford Press.

Gotham, H. J., Sher, K. J., & Wood, P. K. (1997). Predicting stability and change in frequency of intoxication from the college years to beyond: individual-difference and role transition variables. *Journal of Abnormal Psychology*, 106(4), 619–629. <http://doi.org/10.1037/0021-843X.106.4.619>

Grant, V. V., Stewart, S. H., & Mohr, C. D. (2009). Coping-anxiety and coping-depression motives predict different daily mood-drinking relationships. *Psychology of Addictive Behaviors*, 23(2), 226–237. <http://doi.org/10.1037/a0015006>

- Greeley, J. D., & Oei, T. P. S. (1999). Alcohol and tension reduction. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (pp. 14–53). New York: The Guilford Press.
- Grekin, E. R., & Sher, K. J. (2006). Alcohol dependence symptoms among college freshmen: Prevalence, stability, and person-environment interactions. *Experimental and Clinical Psychopharmacology*, *14*(3), 329–338. <http://doi.org/10.1037/1064-1297.14.3.329>
- Habib, K. E., Gold, P. W., & Chrousos, G. P. (2001). Neuroendocrinology of stress. *Endocrinology and Metabolism Clinics of North America*, *30*(3), 695–728. [http://doi.org/10.1016/S0889-8529\(05\)70208-5](http://doi.org/10.1016/S0889-8529(05)70208-5)
- Ham, L. S., & Hope, D. A. (2003). College students and problematic drinking: A review of the literature. *Clinical Psychology Review*, *23*(5), 719–759. [http://doi.org/10.1016/S0272-7358\(03\)00071-0](http://doi.org/10.1016/S0272-7358(03)00071-0)
- Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV narcissistic personality disorder: Results from the wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, *64*(7), 830–842. <http://doi.org/10.1001/archpsyc.64.7.830>
- Henson, J. M., Pearson, M. R., & Carey, K. B. (2015). Defining and characterizing differences in college alcohol intervention efficacy : A growth mixture modeling application, *Journal of Consulting and Clinical Psychology*, *83*(2), 370–381. <http://doi.org/10.1037/a0038897>
- Hingson, R. W., Zha, W., & Weitzman, E. R. (2009). Magnitude of and trends in alcohol-related mortality and morbidity among U.S. college students ages 18-24, 1998-2005. *Journal of Studies on Alcohol and Drugs. Supplement*, (16), 12–20.
<http://doi.org/10.1146/annurev.publhealth.26.021304.144652>

- Hittner, J. B., & Swickert, R. (2006). Sensation seeking and alcohol use: A meta-analytic review. *Addictive Behaviors, 31*(8), 1383–1401. <http://doi.org/10.1016/j.addbeh.2005.11.004>
- Jackson, K. M., Sher, K. J., & Park, A. (2005). Drinking among college students. Consumption and consequences. *Recent Developments in Alcoholism, 17*, 85–117.
- Johnson, E. O., Kamilaris, T. C., Chrousos, G. P., & Gold, P. W. (1992). Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neuroscience and Biobehavioral Reviews, 16*(2), 115–130. [http://doi.org/10.1016/S0149-7634\(05\)80175-7](http://doi.org/10.1016/S0149-7634(05)80175-7)
- Kaim, S. C., Brill, H., Cloud, L. A., Knott, D. H., Lieber, C. S., McIsaac, W. M., Seixas, F. A. (1972). Rationale for the diagnosis. *American Journal of Psychiatry, 129*(2), 127–134.
- Kerr, J. S. (1996). Two myths of addiction: The addictive personality and the issue of free choice. *Human Psychopharmacology, 11*(SUPPL. 1). [http://doi.org/10.1002/\(SICI\)1099-1077\(199602\)11:1+<S9::AID-HUP747>3.0.CO;2-6](http://doi.org/10.1002/(SICI)1099-1077(199602)11:1+<S9::AID-HUP747>3.0.CO;2-6)
- Khantzian, E. J. (1999). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry, 4*(5), 231–244. <http://doi.org/10.3109/10673229709030550>
- King, A. C., de Wit, H., McNamara, P. J., & Cao, D. (2011). Rewarding, stimulant, and sedative alcohol responses and relationship to future binge drinking. *Archives of General Psychiatry, 68*(4), 389–399. <http://doi.org/10.1001/archgenpsychiatry.2011.26>
- King, A., Munisamy, G., De Wit, H., & Lin, S. (2006). Attenuated cortisol response to alcohol in heavy social drinkers. *International Journal of Psychophysiology, 59*(3), 203–209. <http://doi.org/10.1016/j.ijpsycho.2005.10.008>

- King, K. M., Patock-Peckham, J. A., Dager, A. D., Thimm, K., & Gates, J. R. (2014). On the mismeasurement of impulsivity: Trait, behavioral, and neural models in alcohol research among adolescents and young adults. *Current Addiction Reports, 1*(1), 19–32.
<http://doi.org/10.1007/s40429-013-0005-4>
- Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology, 19*(4), 313–333.
[http://doi.org/10.1016/0306-4530\(94\)90013-2](http://doi.org/10.1016/0306-4530(94)90013-2)
- Koob, G. F. (2008). A role for brain stress systems in addiction. *Neuron, 59*(1), 11–34.
<http://doi.org/10.1016/j.neuron.2008.06.012>
- Koob, G. F. (2009). Brain stress systems in the amygdala and addiction. *Brain Research, 1293*, 61–75.
<http://doi.org/10.1016/j.brainres.2009.03.038>
- Koob, G. F., & Volkow, N. D. (2010). Neurocircuitry of addiction. *Neuropsychopharmacology, 35*(1), 217–238. <http://doi.org/10.1038/npp.2010.4>
- Kreek, M. J., Nielsen, D. A., Butelman, E. R., & LaForge, K. S. (2005). Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience, 8*(11), 1450–1457. <http://doi.org/10.1038/nn1583>
- Kreek, M. J., Nielsen, D. A., & LaForge, K. S. (2004). Genes associated with addiction: alcoholism, opiate, and cocaine addiction. *Neuromolecular Medicine, 5*(1), 85–108.
<http://doi.org/10.1385/NMM:5:1:085>
- Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*(1), 2–18. <http://doi.org/10.1016/j.psyneuen.2008.10.004>

- Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2005). Why do young people drink? A review of drinking motives. *Clinical Psychology Review*, 25(7), 841–861.
<http://doi.org/10.1016/j.cpr.2005.06.002>
- Kushner, M. G., Abrams, K., & Borchardt, C. (2000). The relationship between anxiety disorders and alcohol use disorders: a review of major perspectives and findings. *Clinical Psychology Review*, 20(2), 149–171. [http://doi.org/10.1016/S0272-7358\(99\)00027-6](http://doi.org/10.1016/S0272-7358(99)00027-6)
- LaBrie, J. W., & Pedersen, E. R. (2008). Prepartying promotes heightened risk in the college environment: An event-level report. *Addictive Behaviors*, 33(7), 955–959.
<http://doi.org/10.1016/j.addbeh.2008.02.011>
- Lee, M. R., Chassin, L., & Villalta, I. K. (2013). Maturing out of alcohol involvement: transitions in latent drinking statuses from late adolescence to adulthood. *Development and Psychopathology*, 25(4 Pt 1), 1137–53. <http://doi.org/10.1017/S0954579413000424>
- Leshner, A. I. (1999). Science-based views of drug addiction and its treatment. *The Journal of the American Medical Association*, 282(14), 1314–1316. <http://doi.org/10.1001/jama.282.14.1314>
- Leshner, A. I. (1997). Addiction is a brain disease, and it matters. *Science*, 278(5335), 45–47.
<http://doi.org/10.1126/science.278.5335.45>
- Levine, H. G. (1978). The discovery of addiction. Changing conceptions of habitual drunkenness in America. *Journal of Studies on Alcohol*, 39(1), 143–174. [http://doi.org/10.1016/0740-5472\(85\)90022-4](http://doi.org/10.1016/0740-5472(85)90022-4)
- Littlefield, A. K., & Sher, K. J. (2010). The multiple, distinct ways that personality contributes to alcohol use disorders. *Social and Personality Psychology Compass*, 4(9), 767–782.
<http://doi.org/10.1111/j.1751-9004.2010.00296.x>

- Littlefield, A. K., Stevens, A. K., & Sher, K. J. (2013). Impulsivity and alcohol involvement: Multiple, distinct constructs and processes. *Current Addiction Reports, 1*(1), 33–40.
<http://doi.org/10.1007/s40429-013-0004-5>
- Lovallo, W. R. (2006). Cortisol secretion patterns in addiction and addiction risk. *International Journal of Psychophysiology, 59*(3), 195–202. <http://doi.org/10.1016/j.ijpsycho.2005.10.007>
- MacDonald, T. K., Fong, G. T., Zanna, M. P., & Martineau, A. M. (2000). Alcohol myopia and condom use: Can alcohol intoxication be associated with more prudent behavior? *Journal of Personality and Social Psychology, 78*(4), 605–619. <http://doi.org/10.1037/0022-3514.78.4.605>
- Magid, V., MacLean, M. G., & Colder, C. R. (2007). Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors, 32*(10), 2046–2061. <http://doi.org/10.1016/j.addbeh.2007.01.015>
- Magrys, S. A., & Olmstead, M. C. (2015). Acute stress increases voluntary consumption of alcohol in undergraduates. *Alcohol and Alcoholism, 50*(2), 213–218. <http://doi.org/10.1093/alcalc/agu101>
- Magrys, S. A., Olmstead, M. C., Wynne-Edwards, K. E., & Balodis, I. M. (2013). Neuroendocrinological responses to alcohol intoxication in healthy males: Relationship with impulsivity, drinking behavior, and subjective effects. *Psychophysiology, 50*(2), 204–209. <http://doi.org/10.1111/psyp.12007>
- Mallett, K. A., Varvil-Weld, L., Borsari, B., Read, J. P., Neighbors, C., & White, H. R. (2013). An update of research examining college student alcohol-related consequences: New perspectives and implications for interventions. *Alcoholism: Clinical and Experimental Research, 37*(5), 709–716.
<http://doi.org/10.1111/acer.12031>

- Martinez, J. A., Sher, K. J., & Wood, P. K. (2008). Is heavy drinking really associated with attrition from college? The alcohol-attrition paradox. *Psychology of Addictive Behaviors*, 22(3), 450–456.
<http://doi.org/10.1037/0893-164X.22.3.450>
- McGue, M., Slutske, W., & Iacono, W. G. (1999). Personality and substance use disorders: II. Alcoholism versus drug use disorders. *Journal of Consulting and Clinical Psychology*, 67(3), 394–404. <http://doi.org/10.1037/0022-006X.67.3.394>
- McLellan, A. T., Lewis, D. C., O'Brien, C. P., & Kleber, H. D. (2000). Drug dependence, a chronic medical illness. *Journal of the American Medical Association*, 284(13), 1689.
<http://doi.org/10.1001/jama.284.13.1689>
- Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, 158(11), 1783–1793.
<http://doi.org/10.1176/appi.ajp.158.11.1783>
- Mohapatra, S., Patra, J., Popova, S., Duhig, A., & Rehm, J. (2010). Social cost of heavy drinking and alcohol dependence in high-income countries. *International Journal of Public Health*, 55(3), 149–157. <http://doi.org/10.1007/s00038-009-0108-9>
- Monahan, C. J., McDevitt-Murphy, M. E., Dennhardt, A. A., Skidmore, J. R., Martens, M. P., & Murphy, J. G. (2013). The impact of elevated posttraumatic stress on the efficacy of brief alcohol interventions for heavy drinking college students. *Addictive Behaviors*, 38(3), 1719–1725.
<http://doi.org/10.1016/j.addbeh.2012.09.004>
- Moos, R. H., & Moos, B. S. (2006). Rates and predictors of relapse after natural and treated remission from alcohol use disorders. *Addiction*, 101(2), 212–222. <http://doi.org/10.1111/j.1360-0443.2006.01310.x>

- Morean, M. E., & Corbin, W. R. (2010). Subjective response to alcohol: A critical review of the literature. *Alcoholism: Clinical and Experimental Research*, 34(3), 385–395. <http://doi.org/10.1111/j.1530-0277.2009.01103.x>
- Morris, E. P., Stewart, S. H., & Ham, L. S. (2005). The relationship between social anxiety disorder and alcohol use disorders: A critical review. *Clinical Psychology Review*, 25(6), 734–760. <http://doi.org/10.1016/j.cpr.2005.05.004>
- Morse, R. M., & Flavin, D. K. (1992). The definition of alcoholism. *Alcohol and Alcoholism*, 28(6), 617–620. <http://doi.org/10.1001/jama.269.5.586>
- Mulder, R. T. (2002). Alcoholism and personality, *Australian and New Zealand Journal of Psychiatry*, 36, 44–52. <http://doi.org/10.1046/j.1440-1614.2002.00958.x>
- Neal, D. J., & Fromme, K. (2007). Event-level covariation of alcohol intoxication and behavioral risks during the first year of college. *Journal of Consulting and Clinical Psychology*, 75(2), 294–306. <http://doi.org/10.1037/0022-006X.75.2.294>
- Newlin, D. B., & Thomson, J. B. (1990). Alcohol challenge with sons of alcoholics: A critical review and analysis. *Psychological Bulletin*, 108(3), 383–402. <http://doi.org/10.1037/0033-2909.108.3.383>
- O'Brien, M. C., McCoy, T. P., Rhodes, S. D., Wagoner, A., & Wolfson, M. (2008). Caffeinated cocktails: Energy drink consumption, high-risk drinking, and alcohol-related consequences among college students. *Academic Emergency Medicine*, 15(5), 453–460. <http://doi.org/10.1111/j.1553-2712.2008.00085.x>
- O'Neill, S. E., Parra, G. R., & Sher, K. J. (2001). Clinical relevance of heavy drinking during the college years: cross-sectional and prospective perspectives. *Psychology of Addictive Behaviors*, 15(4), 350–359. <http://doi.org/10.1037/0893-164X.15.4.350>

- Ogborne, A. C. (2004). Theories of addiction and implications for counselling. In *Alcohol & Drug Problems: A Practical Guide for Counsellors* (pp. 3–21). Toronto: Centre for Addictions & Mental Health.
- Pedersen, E. R., & LaBrie, J. (2006). Drinking game participation among college students: Gender and ethnic implications. *Addictive Behaviors, 31*(11), 2105–2115.
<http://doi.org/10.1016/j.addbeh.2006.02.003>
- Perkins, H. W. (2002). Surveying the damage: a review of research on consequences of alcohol misuse in college populations. *Journal of Studies on Alcohol. Supplement, (14)*, 91–100.
- Perkins, H. W. (2007). Misperceptions of peer drinking norms in Canada: Another look at the “reign of error” and its consequences among college students. *Addictive Behaviors, 32*(11), 2645–2656.
<http://doi.org/10.1016/j.addbeh.2007.07.007>
- Perry, J. L., & Carroll, M. E. (2008). The role of impulsive behavior in drug abuse. *Psychopharmacology, 200*(1), 1–26. <http://doi.org/10.1007/s00213-008-1173-0>
- Piazza, P. V., & Le Moal, M. L. (1996). Pathophysiological basis of vulnerability to drug abuse: role of an interaction between stress, glucocorticoids, and dopaminergic neurons. *Annual Review of Pharmacology and Toxicology, 36*(1), 359–378.
<http://doi.org/10.1146/annurev.pa.36.040196.002043>
- Piazza, P. V., & Le Moal, M. (1998). The role of stress in drug self-administration. *Trends in Pharmacological Sciences, 19*(2), 67–71. [http://doi.org/10.1016/S0165-6147\(97\)01115-2](http://doi.org/10.1016/S0165-6147(97)01115-2)
- Pope, J., Ionescu-Pioggia, M., & Pope, K. W. (2001). Drug use and life style among college undergraduates: A 30-year longitudinal study. *American Journal of Psychiatry, 158*(9), 1519–1521.
<http://doi.org/10.1176/appi.ajp.158.9.1519>

- Price, S. R., Hilchey, C. A., Darredeau, C., Fulton, H. G., & Barrett, S. P. (2010). Energy drink co-administration is associated with increased reported alcohol ingestion. *Drug and Alcohol Review*, 29(3), 331–333. <http://doi.org/10.1111/j.1465-3362.2009.00163.x>
- Quinn, P. D., & Fromme, K. (2011a). Predictors and outcomes of variability in subjective alcohol intoxication among college students: An event-level analysis across 4 years. *Alcoholism: Clinical and Experimental Research*, 35(3), 484–495. <http://doi.org/10.1111/j.1530-0277.2010.01365.x>
- Quinn, P. D., & Fromme, K. (2011b). Subjective response to alcohol challenge: A quantitative review. *Alcoholism: Clinical and Experimental Research*, 35(10), 1759–1770. <http://doi.org/10.1111/j.1530-0277.2011.01521.x>
- Rehm, J., Ashley, M. J., Room, R., Single, E., Bondy, S., Ferrence, R., & Giesbrecht, N. (1996). On the emerging paradigm of drinking patterns and their social and health consequences. *Addiction*, 91(11), 1615–1621. <http://doi.org/10.1080/09652149639178>
- Rehm, J., Mathers, C., Popova, S., Thavorncharoensap, M., Teerawattananon, Y., & Patra, J. (2009). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. *The Lancet*, 373(9682), 2223–2233. [http://doi.org/10.1016/S0140-6736\(09\)60746-7](http://doi.org/10.1016/S0140-6736(09)60746-7)
- Rehm, J., Rehn, N., Room, R., Monteiro, M., Gmel, G., Jernigan, D., & Frick, U. (2003). The global distribution of average volume of alcohol consumption and patterns of drinking. *European Addiction Research*, 9(4), 147–156. <http://doi.org/10.1159/000072221>
- Reid, A. E., Carey, K. B., Merrill, J. E., & Carey, M. P. (2015). Social network influences on initiation and maintenance of reduced drinking among college students. *Journal of Consulting and Clinical Psychology*, 83(1), 36–44. <http://doi.org/10.1037/a0037634>

- Robotham, D., & Julian, C. (2006). Stress and the higher education student: A critical review of the literature, *Journal of Further & Higher Education*, *30*(2), 107–117.
<http://doi.org/10.1080/03098770600617513>
- Roerecke, M., & Rehm, J. (2013). Alcohol use disorders and mortality: A systematic review and meta-analysis. *Addiction*, *108*(9), 1562–1578. <http://doi.org/10.1111/add.12231>
- Roh, S., Matsushita, S., Hara, S., Maesato, H., Matsui, T., Suzuki, G., Higuchi, S. (2011). Role of GABRA2 in moderating subjective responses to alcohol. *Alcoholism: Clinical and Experimental Research*, *35*(3), 400–407. <http://doi.org/10.1111/j.1530-0277.2010.01357.x>
- Rose, A. K., Hobbs, M., & Drummond, C. (2013). Differentiating the contribution of pharmacological from alcohol expectancy effects to changes in subjective response and priming over successive drinks. *Alcoholism: Clinical and Experimental Research*, *37*(4), 687–695.
<http://doi.org/10.1111/acer.12022>
- Rossow, I., & Romelsjö, A. (2006). The extent of the “prevention paradox” in alcohol problems as a function of population drinking patterns. *Addiction*, *101*(1), 84–90. <http://doi.org/10.1111/j.1360-0443.2005.01294.x>
- Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption--II. *Addiction*, *88*(6), 791–804.
<http://doi.org/10.1111/j.1360-0443.1993.tb02093.x>
- Schlauch, R. C., Waesche, M. C., Riccardi, C. J., Donohue, K. F., Blagg, C. O., Christensen, R. L., & Lang, A. R. (2010). A meta-analysis of the effectiveness of placebo manipulations in alcohol-

challenge studies. *Psychology of Addictive Behaviors*, 24(2), 239–253.

<http://doi.org/10.1037/a0017709>

Schlotz, W., Kumsta, R., Layes, I., Entringer, S., Jones, A., & Wüst, S. (2008). Covariance between psychological and endocrine responses to pharmacological challenge and psychosocial stress: A question of timing. *Psychosomatic Medicine*, 70(7), 787–796.

<http://doi.org/10.1097/PSY.0b013e3181810658>

Schmidt, N. B., Buckner, J. D., & Keough, M. E. (2007). Anxiety sensitivity as a prospective predictor of alcohol use disorders. *Behavior Modification*, 31(2), 202–219.

<http://doi.org/10.1177/0145445506297019>

Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology*, 197(1), 169–178.

<http://doi.org/10.1007/s00213-007-1023-5>

Schuckit, M. A. (1987). Biological vulnerability to alcoholism. *Journal of Consulting and Clinical Psychology*, 55(3), 301–309. <http://doi.org/10.1037/0022-006X.55.3.301>

Schuckit, M. A. (2002). Vulnerability factors for alcoholism. *Neuropsychopharmacology*, 1399–1411.

Schuckit, M.A., Smith, T.L., Kalmijn, J., Trim, R.S., Cesario, E., Saunders, G., Sanchez, N., & Campbell, N. (2012). Comparison across two generations of prospective models of how the low level of response to alcohol. *Journal of Studies on Alcohol and Drugs*, 73, 195–204.

<http://dx.doi.org/10.15288/jsad.2012.73.195>

Schuckit, M. A. (2006). Comorbidity between substance use disorders and psychiatric conditions , *Addiction*, 101, 76–88. <http://doi.org/10.1111/j.1360-0443.2006.01592.x>

- Schuckit, M. A., Smith, T. L., Anderson, K. G., & Brown, S. A. (2004). Testing the level of response to alcohol: Social information processing model of alcoholism risk: a 20-year prospective study. *Alcoholism: Clinical & Experimental Research*, 28(12), 1890–1897.
<http://doi.org/10.1097/01.ALC.0000148111.43332.A5>
- Shaffer, H. J. (1985). The discovery of addiction: Levine and the philosophical foundations of drug abuse treatment. *Journal of Substance Abuse Treatment*, 2(1), 41–57. [http://doi.org/10.1016/0740-5472\(85\)90021-2](http://doi.org/10.1016/0740-5472(85)90021-2)
- Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology*, 1, 493–523. <http://doi.org/10.1146/annurev.clinpsy.1.102803.144107>
- Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and anti-social personality disorder. *Journal of Abnormal Psychology*, 103(1), 92–102.
<http://dx.doi.org/10.1037/0021-843X.103.1.92>
- Sinha, R., Fox, H. C., Hong, K.-I. A., Hansen, J., Tuit, K., & Kreek, M. J. (2011). Effects of adrenal sensitivity, stress- and cue-induced craving, and anxiety on subsequent alcohol relapse and treatment outcomes. *Archives of General Psychiatry*, 68(9), 942–952.
<http://doi.org/10.1001/archgenpsychiatry.2011.49>
- Slutske, W. S. (2005). Alcohol use disorders among US college students and their non-college-attending peers. *Archives of General Psychiatry*, 62(3), 321–327. [http://doi.org/10.1016/S0084-3717\(08\)70186-7](http://doi.org/10.1016/S0084-3717(08)70186-7)
- Slutske, W. S., Heath, A. C., Madden, P.A.F., Bucholz, K. K., Statham, D. J., & Martin, N. G. (2002). Personality and the genetic risk for alcohol dependence. *Journal of Abnormal Psychology*, 111(1), 124–133. <http://doi.org/10.1037/0021-843X.111.1.124>

- Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D. M. (2007). On the validity and utility of discriminating among impulsivity-like traits. *Assessment, 14*(2), 155–170. <http://doi.org/10.1177/1073191106295527>
- Sobell, L. C., Brown, J., Leo, G. I., & Sobell, M. B. (1996). The reliability of the Alcohol Timeline Followback when administered by telephone and by computer. *Drug and Alcohol Dependence, 42*(1), 49–54. [http://doi.org/10.1016/0376-8716\(96\)01263-X](http://doi.org/10.1016/0376-8716(96)01263-X)
- Söderpalm, A., Nikolayev, L., & de Wit, H. (2003). Effects of stress on responses to methamphetamine in humans. *Psychopharmacology, 170*(2), 188–199. <http://doi.org/10.1007/s00213-003-1536-5>
- Söderpalm, A. H. V., & de Wit, H. (2002). Effects of stress and alcohol on subjective state in humans. *Alcoholism, Clinical and Experimental Research, 26*(6), 818–826. <http://doi.org/http://dx.doi.org/10.1111/j.1530-0277.2002.tb02610.x>
- Sorocco, K. H., Lovallo, W. R., Vincent, A. S., & Collins, F. L. (2006). Blunted hypothalamic-pituitary-adrenocortical axis responsivity to stress in persons with a family history of alcoholism. *International Journal of Psychophysiology, 59*(3), 210–217. <http://doi.org/10.1016/j.ijpsycho.2005.10.009>
- Soyka, M., Gorig, E., & Naber, D. (1992). Serum prolactin increase induced by ethanol: a dose-dependent effect not related to stress. *Psychoneuroendocrinology, 16*(5), 441–446. [http://dx.doi.org/10.1016/0306-4530\(91\)90009-I](http://dx.doi.org/10.1016/0306-4530(91)90009-I)
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews, 24*, 417–463. [http://doi.org/10.1016/S0149-7634\(00\)00014-2](http://doi.org/10.1016/S0149-7634(00)00014-2)

- Stewart, S. H., Taylor, S., & Baker, J. M. (1997). Gender differences in dimensions of anxiety sensitivity. *Journal of Anxiety Disorders, 11*(2), 179–200. [http://doi.org/10.1016/S0887-6185\(97\)00005-4](http://doi.org/10.1016/S0887-6185(97)00005-4)
- Stewart, S. H., Zvolensky, M. J., & Eifert, G. H. (2001). Negative-reinforcement drinking motives mediate the relation between anxiety sensitivity and increased drinking behavior. *Personality and Individual Differences, 31*(2), 157–171. [http://doi.org/10.1016/S0191-8869\(00\)00213-0](http://doi.org/10.1016/S0191-8869(00)00213-0)
- Steyer, R., Schmitt, M., & Eid, M. (1999). Latent state - trait theory and research in personality and individual differences. *European Journal of Personality, 13*, 389–408.
[http://doi.org/10.1002/\(SICI\)1099-0984\(199909/10\)13:5<389::AID-PER361>3.0.CO;2-A](http://doi.org/10.1002/(SICI)1099-0984(199909/10)13:5<389::AID-PER361>3.0.CO;2-A)
- Swendsen, J., & Le Moal, M. (2011). Individual vulnerability to addiction. *Annals of the New York Academy of Sciences, 1216*(1), 73–85. <http://doi.org/10.1111/j.1749-6632.2010.05894.x>
- Testa, M., Fillmore, M. T., Norris, J., Abbey, A., Curtin, J. J., Leonard, K. E., Hayman, L. W. (2006). Understanding alcohol expectancy effects: Revisiting the placebo condition. *Alcoholism: Clinical and Experimental Research, 30*(2), 339–348. <http://doi.org/10.1111/j.1530-0277.2006.00039.x>
- Thomas, G. (2012). Levels and patterns of Alcohol use in Canada: Alcohol Price Policy series, Report 1 of 3, (November). Ottawa: Canadian Centre on Substance Abuse.
- Timberlake, D. S., Hopfer, C. J., Rhee, S. H., Friedman, N. P., Haberstick, B. C., Lessem, J. M., & Hewitt, J. K. (2007). College attendance and its effect on drinking behaviors in a longitudinal study of adolescents. *Alcoholism: Clinical and Experimental Research, 31*(6), 1020–1030.
<http://doi.org/10.1111/j.1530-0277.2007.00383.x>
- Uhart, M., & Wand, G. S. (2009). Stress, alcohol and drug interaction: An update of human research. *Addiction Biology, 14*(1), 43–64. <http://doi.org/10.1111/j.1369-1600.2008.00131.x>

- Uhart, M., Weerts, E. M., McCaul, M. E., Guo, X., Yan, X., Kranzler, H. R., Li, N., & Wand, G. S. (2012). GABRA2 markers moderate the subjective effects of alcohol. *Addiction Biology, 18*(2), 357–369. <http://doi.org/10.1111/j.1369-1600.2012.00457.x>
- Vaughan, E. L., Corbin, W. R., & Fromme, K. (2009). Academic and social motives and drinking behavior. *Psychology of Addictive Behaviors, 23*(4), 564–576. <http://doi.org/10.1037/a0017331>
- Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience and Biobehavioral Reviews, 32*(4), 777–810. <http://doi.org/10.1016/j.neubiorev.2007.11.003>
- Vincent, K.B., Kasperski, S.J., Caldeira, K.M., Garnier, L.M., Pinchevsky, G.M., Grady, K.E.O., & Arria, A.M. (2013). Experiences from the college life study, *International Journal of Multiple Research Approaches, 6*(1), 1–21. <http://doi.org/10.5172/mra.2012.6.1.56>
- Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology, 201*(3), 315–324. <http://doi.org/10.1007/s00213-008-1284-7>
- Wechsler, H., Davenport, A., Dowdall, G., Moeykens, B., & Castillo, S. (1994). Health and behavioral consequences of binge drinking in college. *Journal of the American Medical Association, 272*(21), 1672–1677. <http://doi.org/10.1001/jama.1994.03520210056032>
- Wechsler, H., Dowdall, G.W., Davenport, A., & Castillo, S. (1995). Correlates of college student binge drinking. *American Journal of Public Health, 85*(7), 921–926. <http://doi.org/10.2105/AJPH.85.7.921>

Wechsler, H., Dowdall, G.W., Davenport, A.A., & Rimm, E.B. (1995). A gender-specific measure of binge drinking among college students. *American Journal of Public Health, 85*(7), 982–985.
<http://doi.org/10.2105/AJPH.85.7.982>

Wechsler, H., Lee, J. E., Kuo, M., Seibring, M., Nelson, T. F., & Lee, H. (2002). Trends in college binge drinking during a period of increased prevention efforts. Findings from 4 Harvard School of Public Health College Alcohol Study surveys: 1993-2001. *Journal of American College Health, 50*(5), 203–217. <http://doi.org/10.1080/07448480209595713>

Wechsler, H., Molnar, B. E., Davenport, A. E., & Baer, J. S. (1999). College alcohol use: A full or empty glass? *Journal of American College Health, 47*(6), 247–252.
<http://doi.org/10.1080/07448489909595655>

White, H. R., Labouvie, E. W., & Papadaratsakis, V. (2005). Changes in substance use during the transition to adulthood: A comparison of college students and their noncollege age peers. *Journal of Drug Issues, 35*(2), 281–306. <http://doi.org/10.1177/002204260503500204>

White, H. R., McMorris, B. J., Catalano, R. F., Fleming, C. B., Haggerty, K. P., & Abbott, R. D. (2006). Increases in alcohol and marijuana use during the transition out of high school into emerging adulthood: The effects of leaving home, going to college, and high school protective factors. *Journal of Studies on Alcohol, 67*(6), 810–822. <http://dx.doi.org/10.15288/jsa.2006.67.810>

White, W. L. (2000). Addiction as a disease: Birth of a concept. *Counselor, 1*(1), 46–51.

Wood, P. K., Sher, K. J., & Rutledge, P. C. (2007). College student alcohol consumption, day of the week, and class schedule. *Alcoholism: Clinical and Experimental Research, 31*(7), 1195–1207.
<http://doi.org/10.1111/j.1530-0277.2007.00402.x>

Zamboanga, B. L., Schwartz, S. J., Ham, L. S., Borsari, B., & Van Tyne, K. (2010). Alcohol expectancies, pregaming, drinking games, and hazardous alcohol use in a multiethnic sample of college students. *Cognitive Therapy and Research*, *34*(2), 124–133. <http://doi.org/10.1007/s10608-009-9234-1>

Zimmermann, U., Spring, K., Kunz-Ebrecht, S. R., Uhr, M., Wittchen, H.U., & Holsboer, F. (2004). Effect of ethanol on hypothalamic-pituitary-adrenal system response to psychosocial stress in sons of alcohol-dependent fathers. *Neuropsychopharmacology*, *29*(6), 1156–1165. <http://doi.org/10.1038/sj.npp.1300395>

Appendix A

Appendix A. Demographic information for participant sample at Time 1 and Time 2.

Demographic Variable	Time 1 [n (%)]	Time 2 [n (%)]
<i>Living Arrangement</i>		
Alone	1 (0.6)	76 (44.7)
With Friends	1 (0.6)	20 (11.8)
With Roommates	0 (0)	64 (37.6)
With Parents	165 (97.6)	7 (4.1)
With Spouse	0 (0)	1 (0.6)
With Spouse and Younger children (< 13 years)	0 (0)	0 (0)
With Spouse and Older Children (> 13 yrs)	1 (0.6)	0 (0)
Alone with Younger children (< 13 years)	0 (0)	0 (0)
Alone Older Children (> 13 yrs)	0 (0)	0 (0)
Other	1 (0.6)	2 (1.2)
<i>Total</i>	<i>169 (100)</i>	<i>170 (100)</i>
<i>Relationship Status</i>		
Single	116 (69.9)	117 (68.8)
Dating	18 (10.8)	16 (9.4)
Serious Relationship	27 (16.3)	31 (18.2)
Recently broke up	5 (2.7)	6 (3.5)
Living with Partner	0 (0)	0 (0)
Engaged	0 (0)	0 (0)
Married	0 (0)	0 (0)
Separated/ Divorced	0 (0)	0 (0)
Widow/ed	0 (0)	0 (0)
<i>Total</i>	<i>166 (100)</i>	<i>170 (100)</i>

Note. Due to missing data, some demographic variables include fewer participants than the total sample ($n = 183$).