

**IMPULSIVITY: A LINK BETWEEN
BIPOLAR AND ALCOHOL USE DISORDERS**

by

Season C. Tompkins

B.A., The University of British Columbia, 2007

M.A., The University of British Columbia, 2009

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

in

THE FACULTY OF GRADUATE AND POSTDOCTORAL STUDIES

(Psychology)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

June 2015

© Season C. Tompkins, 2015

Abstract

There are many commonalities between bipolar and alcohol use disorders which suggest a common etiology, or vulnerability, for these disorders. Impulsivity is a shared feature of both disorders which may help to identify an underlying link between the disorders. The presented studies used an undergraduate sample to examine personality, behavioural, and electroencephalogram (EEG) measures which have previously been linked to either bipolar or alcohol use disorders, or to both disorders. The first study examined self-report impulsivity as a mediator of the relationship between hypomanic personality and alcohol use. Individuals from the first study were then invited to participate in a second study using delay discounting, a behavioural measure of impulsivity, and EEG measures, which have been related to bipolar and alcohol use disorders. In the first study, higher hypomanic personality was related to higher impulsivity and alcohol use. Impulsivity was also positively related to alcohol use. In this study, impulsivity mediated the relationship between hypomanic personality and various measures of alcohol use. Specifically, Sensation Seeking and Negative Urgency were the UPPS-P scales found to most often mediate the relationships between hypomanic personality and alcohol use. In the second study, hypomanic personality, Positive Urgency, and alcohol use were all positively correlated with delay discounting rates. However, Positive Urgency did not significantly mediate the relationship between hypomanic personality or alcohol use and delay discounting. With regard to EEG measures, there were some findings showing impulsivity, hypomanic personality, and alcohol use related to longer latencies of the P300 event-related potential, but not smaller amplitudes. This lack of EEG findings may be explained by the relatively

healthy sample of undergraduate participants that did not endorse severe enough psychopathology to show the associations previously seen in clinical samples.

Preface

With guidance from my faculty supervisor, Dr. Colleen Brenner, I was fully involved in the identification and design of both studies included in this dissertation. With the help of undergraduate research assistants, I was fully responsible for data collection and entry required by both studies. I solely conducted the analysis and writing of this dissertation, under the supervision of Dr. Brenner.

UBC Research Ethics Board approval was required and obtained to conduct all stages of this research:

H10 – 02135 Alcohol Use, Personality, and Expectancies

H11 – 00126 Alcohol Use, Personality, and Expectancies – Lab Measures

Table of Contents

Abstract.....	ii
Preface.....	iv
Table of Contents	v
List of Tables	ix
List of Figures.....	x
List of Abbreviations	xii
Acknowledgements	xiii
Dedication	xiv
Chapter 1: Introduction	1
Bipolar Disorder.....	2
Hypomanic personality	3
Alcohol Use	3
Alcohol use and bipolar disorder	5
Alcohol use and hypomanic personality	7
Impulsivity	8
UPPS-P Impulsive Behaviour Scale	9
Delay discounting	10
Delay discounting and the UPPS-P.....	11
Impulsivity and Bipolar Disorder	12
UPPS-P and bipolar disorder	13
Delay discounting and bipolar disorder	14
Impulsivity and Alcohol Use	15
UPPS-P and alcohol use.....	15
Delay discounting and alcohol use	18
Event-Related Potential Measures	20
P300 ERP and bipolar disorder.....	21
The auditory oddball task and bipolar disorder	21
The visual oddball task and bipolar disorder	22

P300 ERP and alcohol use	23
The auditory oddball task and alcohol use.....	23
The visual oddball task and alcohol use	24
P300 ERP and impulsivity	26
The auditory oddball task and impulsivity.....	26
The visual oddball task and impulsivity	27
Impulsivity as a Link Between Bipolar Disorder and Alcohol Use Disorders	28
Overview of Studies.....	29
Chapter 2: Study 1	30
Introduction and Hypotheses	30
Method	31
Participants.....	31
Procedure	32
Measures	33
Personality questionnaires	33
Hypomanic Personality Scale.....	33
UPPS-P Impulsive Behaviour Scale.....	34
Alcohol use questionnaires	35
Short Michigan Alcoholism Screening Test	36
Statistical analyses	36
Results.....	37
Hypomanic personality	41
Impulsivity	44
Mediation models using the HPS total score	45
Mediation models using the HPS subscales.....	58
Discussion	65
Hypomanic personality related to impulsivity	65
Hypomanic personality related to alcohol use	67
Impulsivity related to alcohol use	68
Mediation	69
Chapter 3: Study 2.....	71

Introduction and Hypotheses	71
Method	73
Participants.....	73
Procedure	74
Measures	74
Questionnaires.....	74
Delay discounting	75
Event-related potential measures	76
Electroencephalogram (EEG) procedure.....	76
The auditory P300 oddball task.....	77
The visual P300 oddball task.....	77
Statistical analyses	80
Results.....	80
Hypomanic personality related to delay discounting.....	89
Impulsivity related to delay discounting.....	90
Mediation models using the HPS scales	90
Alcohol use related to delay discounting	90
Mediation models using alcohol use.....	91
Hypomanic personality related to EEG measures	92
Impulsivity related to EEG measures	94
Mediation	95
Alcohol use related to EEG measures.....	96
Male only subsample	98
Discussion.....	98
Hypomanic personality related to delay discounting.....	99
Impulsivity related to delay discounting.....	99
Alcohol use related to delay discounting	100
Hypomanic personality related to EEG measures	100
Impulsivity related to EEG measures	101
Alcohol use related to EEG measures.....	102
Male only subsample	103

Chapter 4: General Discussion	104
Contributions to the Literature.....	115
Limitations	120
Directions for Future Research.....	121
Conclusion	123
References.....	124
Appendix 1: Ethnicity Differences.....	154

List of Tables

Table 1: Measures Used in Study 1	31
Table 2: Means and Standard Deviations for all Study 1 Variables	38
Table 3: First Order Correlations for Study 1	39
Table 4: Summary of Regression Results for Study 1 Predicting Alcohol Use	43
Table 5: Summary of Regression Results for Study 1 Predicting Impulsivity	43
Table 6: Means and Standard Deviations for all Study 1 Variables for the Study 2 Sample .	82
Table 7: Comparing Means for Study 1 and Study 2 Samples	83
Table 8: Means and Standard Deviations for Study 2 Delay Discounting and the Auditory Oddball EEG Task Variables.....	84
Table 9: Means and Standard Deviations for Study 2 Visual Oddball EEG Task Variables .	85
Table 10: First Order Correlations for Study 2	86
Table 11: Summary of Variables Involved in Mediation Models for Study 2 Predicting Delay Discounting.....	89
Table 12: Summary of Variables Involved in Mediation Models for Study 2 Predicting EEG Measures	96

List of Figures

Figure 1: Mediation model predicting lifetime maximum number of drinks within a 24-hour period from the Hypomanic Personality Scale total score with Negative Urgency, Sensation Seeking, and Lack of Premeditation as mediators	47
Figure 2: Mediation model predicting alcohol use quantity from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.....	49
Figure 3: Mediation model predicting alcohol use frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.....	51
Figure 4: Mediation model predicting alcohol use quantity x frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators...53	
Figure 5: Mediation model predicting binge drinking frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators... 55	
Figure 6: Mediation model predicting alcohol related problems from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators... 57	
Figure 7: Mediation model predicting lifetime maximum number of drinks within a 24-hour period from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator	59
Figure 8: Mediation model predicting alcohol use quantity from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator	60
Figure 9: Mediation model predicting alcohol use frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator	61
Figure 10: Mediation model predicting alcohol use quantity x frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.....	62

Figure 11: Mediation model predicting binge drinking frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator	63
Figure 12: Mediation model predicting alcohol related problems from the Hypomanic Personality Scale Mood Volatility subscale with Negative Urgency and Positive Urgency as mediators.....	64
Figure 13: Rotated Heads task stimuli	79
Figure 14: Typical P300 waveform obtained from the Rotated Heads task	79

List of Abbreviations

APA: American Psychiatric Association

BIS-11: Barratt Impulsiveness Scale-11

BS: Boredom Susceptibility scale of the SSS-V

DASS-21: Depression Anxiety Stress Scales

DIS: Disinhibition scale of the SSS-V

DSM 5: Diagnostic and Statistical Manual of Mental Disorders (5th ed.)

EEG: Electroencephalogram

ERP: Event-related potential

FFM: Five Factor Model

HPS: Hypomanic Personality Scale

I-7: I-7 Impulsiveness Questionnaires

MAST: Michigan Alcoholism Screening Test

MPQ: Multidimensional Personality Questionnaire

NEO-PI-R: Revised NEO Personality Inventory

PRF: Personality Research Form

SMAST: Short Michigan Alcoholism Screening Test

SSS-V: Sensation Seeking Scale Form V

TCI: Temperament and Character Inventory

UBC: University of British Columbia

UPPS-P: UPPS-P Impulsive Behaviour Scale

Acknowledgements

I offer my gratitude to the faculty and my fellow students at the University of British Columbia, who have supported, guided, and encouraged me over the years. I also want to thank the research assistants, volunteers, and participants without whom this would not have been possible. I would like to thank my committee members, Dr. Wolfgang Linden and Dr. Stan Floresco for sharing their wisdom with me and helping me to think about the details. I owe particular thanks to Dr. Colleen Brenner for making time for me, reading countless revisions, providing endless insightful feedback, and continuing to challenge me and guide me through the graduate training experience.

I would also like to thank my family and friends. I owe special thanks to my mother, the strongest woman I know, who constantly motivates me to work harder and be better. I would like to thank my father for always believing in me. I am grateful for my siblings, each of whom have unique talents and inspire me different ways. I would also like to thank Dave, for his unfaltering patience and encouragement throughout this process.

I am extremely grateful for the funding that has helped to support this research: a UBC Four Year Doctoral Fellowship and a Pacific Century Graduate Scholarship.

For Stella

Chapter 1: Introduction

Bipolar and alcohol related disorders have strong genetic bases and are highly heritable (e.g., Goldberg, 2001; Heath et al., 1997; Hill, Steinhauer, Locke-Wellman, & Ulrich, 2009; Preuss & Wong, 2000). These disorders often co-occur (Goldberg, 2001; Preuss & Wong, 2000; Regier, et al., 1990; Swann, Dougherty, Pazzaglia, Pham, & Moeller, 2004; Tohen & Zarate, 1999) and individuals with both disorders generally have worse outcomes than those with only one (Goldberg, 2001; Preuss & Wong, 2000; Swann et al., 2004). Impulsivity is a shared feature of bipolar and alcohol use disorders (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; Swann et al., 2004) which has been shown to be highest in individuals with both disorders (Swann et al., 2004) and appears to share a common heritability with both disorders (Goldberg, 2001; Preuss & Wong, 2000; Swann, 2010). In addition to personality commonalities such as impulsivity, bipolar and alcohol use disorders also share behavioural (Ahn et al., 2011; Bobova, Finn, Rickert, & Lucas, 2009; Kollins, 2003; Petry, 2001; Vuchinich & Simpson, 1998; Yi, Mitchell, & Bickel, 2010) and electrophysiological (Bestelmeyer, Phillips, Crombie, Benson, & St.Clair, 2009; Hall et al., 2007; Polich, Pollock, & Bloom, 1994; Ramachandran, Porjesk, Begleiter, & Litke, 1996) correlates. With so much in common, it seems plausible that there would be a common etiology linking these two disorders.

While the study of clinical samples is essential, subclinical correlates of these disorders have also been identified and may present an opportunity for early intervention. Hypomanic personality traits, as measured by the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986) have been related longitudinally to bipolar disorder, major depressive episodes, and substance use disorders, as well as other features related to mental

illness (Kwapil et al., 2000). Individuals high on hypomanic traits who were also high on impulsivity had even worse outcomes, with higher rates of bipolar disorder, arrests, alcohol use, and borderline personality traits (Kwapil et al., 2000).

The strong relationship of impulsivity to bipolar and alcohol use disorders suggests that it might be important in finding a common etiology. The presented studies examined whether self-report impulsivity mediates the relationship between hypomanic personality and alcohol use. In addition, self-report impulsivity was also considered as a potential mediator of the relationships between hypomanic personality and alcohol use with behavioural and electroencephalogram (EEG) measures that have previously been linked to bipolar and alcohol use disorders.

Bipolar Disorder

Bipolar disorder is characterized by the presence or history of one or more manic, hypomanic, or mixed episodes with or without a history of major depressive episodes (American Psychiatric Association [APA], 2013). Features of mania and hypomania include abnormally elevated or irritable mood and at least three symptoms including: inflated self-esteem, decreased need for sleep, increased talkativeness, flight of ideas, distractibility, psychomotor agitation, and increased involvement in risky activities (APA, 2013). Lifetime prevalence rates of bipolar disorder are estimated between 2.1-3.3% (Grant et al., 2005; Merikangas et al., 2007) and heritability has been estimated between 60-85% (McGuffin et al., 2003; Smoller & Finn, 2003). Bipolar disorder is a debilitating disorder which is often comorbid with substance use disorders, and this comorbidity has been related to greater severity of the disorder (APA, 2013; Swann, 2010).

Hypomanic personality.

Hypomanic personality has been used to describe the premorbid personality style of some individuals who develop bipolar disorder. Individuals with this type of personality are likely to be energetic, upbeat, gregarious, and social (Eckblad & Chapman, 1986). The Hypomanic Personality Scale (HPS) was developed to identify individuals at risk for developing hypomanic or manic episodes and bipolar disorder. During validation of the HPS, high scorers (at least 1.67 SD above the mean for their sex) had significantly higher hypomanic personality characteristics, depressive symptoms, alcohol and drug use, and schizotypal and psychotic-like symptoms, compared to control subjects. High scorers also indicated poorer social adjustment than controls and had experienced more hypomanic episodes (Eckblad & Chapman, 1986). The same validation sample was re-evaluated thirteen years later and, compared to the control group, high HPS scorers were significantly higher in rates of hypomanic episodes, bipolar disorder, major depressive episodes, alcohol abuse and dependence, marijuana abuse and dependence, and psychotic symptoms (Kwapil et al., 2000). Other studies have similarly found high HPS scores to be related to mania, depression, and substance use problems (Krueger, 1999; Meyer, 2002; Vollebergh, et al., 2001). Determining how this personality style is related to other measures previously associated with bipolar and alcohol use disorders would be an important step towards early identification and intervention for individuals at high risk of developing these disorders.

Alcohol Use

Alcohol use continues to be high among young Canadians. The 2004 Canadian Addiction Survey (Adlaf, Begin, & Sawka, 2005) reported that approximately 90 percent of

18 to 24 year olds surveyed had consumed alcohol in the past year and 17 percent of current drinkers, age 15 and over, engaged in high-risk drinking behaviour (i.e., behaviour that may lead to alcohol-related problems). Sixteen percent of past year drinkers endorsed regular heavy drinking (five or more drinks for men, four or more drinks for women), which has been associated with more alcohol-related problems. This heavy drinking pattern, or binge drinking, is most prominent in young drinkers (15-24 year olds), peaking at 42.5% for 18-19 year olds. A recent study at the University of British Columbia found that 80.1% of the student drinkers in that sample reported at least one binge in the previous year, with 16.8% reporting at least one binge per week during the previous year (Carlson, Johnson, & Jacobs, 2010). In addition, early substance use may lead to increased (Andersen, Due, Holstein, & Iversen, 2003; York, Welte, Hirsch, Hoffman, & Barnes, 2004), or diversified, later substance use (Guy, Smith, & Bentler, 1994).

Alcohol related disorders have long been understood to be highly heritable. This has been shown with higher rates of alcohol abuse and dependence in children of alcohol abusers, compared to non-abusers, even when those children are raised by non-alcohol abusing adoptive parents (Bohman, Cloninger, Sagvardsson, & Knorrning, 1987; Cloninger, Bohman, Sigvardsson, 1981; Iacono, McGue, & Krueger, 2006). Twin, adoption, and family studies have shown the heritability of alcohol use to be between 50-60% (Heath et al., 1997; McGue, 1999). For such a prevalent problem with such high rates of heritability, there is still a great deal of uncertainty about what is the best combination of predictors for identifying individuals at high risk of developing alcohol related disorders.

Alcohol use and bipolar disorder.

Bipolar and alcohol use disorders often co-occur (Di Florio, Craddock, & van den Bree, 2014; Goldberg, 2001; Preuss & Wong, 2000; Regier, et al., 1990; Swann et al., 2004; Swann, 2010; Tohen & Zarate, 1999), with rates of alcohol use disorders as high as 60% in individuals with bipolar disorder (Cassidy, Ahearn, & Carroll, 2001; Grant et al., 2005; Regier et al, 1990). Compared to healthy controls, bipolar individuals have significantly higher rates of lifetime alcohol abuse or dependence (Strakowski et al., 2009) and, in a national epidemiologic survey in the United States, approximately 25% of respondents with mania or hypomania reported a comorbid alcohol use disorder in the past year (Grant et al., 2004).

In addition to evidence of their respective heritability, bipolar and alcohol use disorders appear to share a common genetic influence (e.g., Goldberg, 2001; Hill et al., 2009; Preuss & Wong, 2000; Swann, 2010). Relatives of individuals with bipolar disorder are four times more likely to develop an alcohol related disorder than those with no relation with the disorder (Preuss & Wong, 2000). Likewise, family studies of bipolar individuals with and without alcohol use disorders found no difference in their family histories of alcohol related disorders. This suggests that there may be an overlap in the inheritance of the two disorders and that they may instead be two manifestations of the same underlying disease process; that is, a genetic predisposition to alcohol use disorders may be common to many bipolar patients (Winokur, 1999). In fact, one study found that both disorders were genetically correlated, meaning that the authors found evidence for shared genetic effects between bipolar and alcohol use disorders. The authors estimated that between 47-57% of the genetic variance in bipolar disorder is common to alcohol use disorders (Carmioli et al., 2014). The development

of bipolar and alcohol related disorders is not linear; neither disorder necessarily precedes the other. Studies have found that many bipolar individuals have substance related problems prior to the onset of the disorder, but it is also clear that many develop a substance abuse problem during the course of the disorder (Goldberg, 2001; Swann et al., 2004). Researchers concluded that when a substance related disorder precedes bipolar disorder, it does not bring on the disorder, but instead represents a common predisposition to both disorders (Goldberg, 2001).

Individuals with both bipolar and alcohol use disorders generally have worse outcomes (Goldberg, 2001; Preuss & Wong, 2000; Sonne, Brady, & Morton, 1994; Swann et al., 2004; Swann, 2010), including earlier onset (Sonne et al., 1994) and more severe course of bipolar disorder, more hospitalizations (Cassidy et al., 2001; Sonne et al., 1994), and increased risk for suicide (Maser et al., 2002). Alcohol use disorders are considered to be part of a highly heritable externalizing spectrum which also consists of antisocial behaviour and a disinhibited personality style, such as impulsiveness (Krueger et al., 2002). Bipolar disorder can also be linked to this externalizing factor of psychopathology through impulsivity, a prominent feature of the disorder (Widiger & Clark, 2000). Since impulsivity is a common feature of both bipolar and alcohol related disorders (Moeller et al., 2001; Swann et al., 2004; Swann, 2010) and tends to be highest in individuals with both disorders (Swann et al., 2004), it may be important in identifying common risk factors for the two disorders.

Strakowski and DelBello (2000) proposed four possible reasons why bipolar and substance use disorders co-occur, with some empirical support for each: 1) substance abuse may be a symptom of bipolar disorder; 2) bipolar individuals use substances to self-medicate

symptoms; 3) substance abuse causes bipolar disorder; and 4) substance use and bipolar disorders share a common risk factor. Due to mixed support for all four explanations, the authors suggested that all may contribute to the comorbidity between bipolar and substance use disorders, but that more evidence is required to help clarify this relationship. The studies presented here focus on the last hypothesis and examine impulsivity as a link to identifying a common etiology or vulnerability to these disorders.

Alcohol use and hypomanic personality.

While it is widely accepted that bipolar and alcohol use disorders co-occur, there has been less focus on subclinical levels of hypomanic personality related to alcohol use. However, studies have found support for a positive relationship between hypomanic personality traits, as measured by the HPS, and substance use ($d = 0.36-0.57$; Eckblad & Chapman, 1986; Klein, Lewinsohn, & Seeley, 1996; Krumm-Merabet & Meyer, 2005; Kwapil et al., 2000; Meyer, Rahman, & Shepard, 2007), and HPS scores predicted substance use disorders longitudinally (Kwapil et al., 2000). Hypomania, as defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), has also been found to be associated with a greater likelihood of substance use disorders (Do & Mezuk, 2013). A study of male undergraduate students found that HPS scores uniquely predicted binge drinking and variability of drinking quantity within individuals (i.e., the standard deviation across time for each individual), even when controlling for alcohol related disorders. However, overall alcohol consumption (quantity x frequency), for the three months preceding the study, was not uniquely predicted by HPS scores. This suggested that hypomanic personality may be related to a pattern of alcohol use characterized by heavy drinking and variability. The authors suggested that this instability reflects a core vulnerability to bipolar disorder (Meyer

& Wolkenstein, 2010). Clearly, the relationship between subclinical levels of hypomanic personality traits and alcohol use needs further examination.

Impulsivity

Impulsivity, a common feature of both bipolar and alcohol use disorders, has been shown to be highly heritable and stable over time, even when measured as early as age 3 (Caspi and Silva, 1995). This early impulsivity has been shown to predict alcohol problems later in life (Caspi, Moffitt, Newman, & Silva, 1996; Granö, Virtanen, Vahtera, Elovainio, & Kivimäki, 2004; Jones, 1968, 1971), suggesting that individuals higher on impulsivity may be at high risk for developing alcohol use problems. Higher levels of impulsivity have also been found to be stable in bipolar patients across mood episodes, that is, during manic, depressed, and euthymic states (Peluso et al., 2007), suggesting that impulsivity may represent an early personality trait marker for this disorder.

Impulsivity is a complex term that has been defined in several different ways, including behavioural impulsivity (acting without thinking; e.g., Moeller et al., 2001; Patton, Stanford, & Barratt, 1995), cognitive impulsivity (being distractible, or lacking focus; Patton et al., 1995) and novelty seeking (e.g., Zuckerman, 1994). A comprehensive definition of impulsivity suggests that it is “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” (Moeller et al., 2001, p.1784). There are many well established measures covering these various aspects of impulsivity. However, research has recently indicated that impulsivity is a multidimensional concept which represents heterogeneous clusters of traits and behaviours reflecting behavioural disinhibition, sensation seeking, reward sensitivity, lack of consideration for negative consequences, and devaluation

of future events (de Wit, Flory, Acheson, McCloskey, & Manuck, 2007; Flory et al., 2006; Moeller et al., 2001; Reynolds, Ortengren, Richards, & de Wit, 2006; Whiteside and Lynam, 2001, 2003). Impulsivity has been measured with a wide variety of self-report and behavioural measures.

UPPS-P Impulsive Behaviour Scale.

The UPPS-P Impulsive Behaviour Scale (UPPS-P) is a newer self-report questionnaire designed to measure the unique facets of impulsivity. Whiteside and Lynam (2001) examined several commonly used self-report measures of impulsivity in order to identify non-overlapping aspects which map onto the Five Factor Model of personality (FFM; McCrae & Costa, 1990). The five factors of the FFM are neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. Impulsivity measures examined in this study included: the EASI-III (Buss & Plomin, 1975), Dickman's (1990) Functional and Dysfunctional Impulsivity Scales, the Barratt Impulsiveness Scale-11 (BIS-11; Patton et al., 1995), the I-7 Impulsiveness Questionnaires (I-7; Eysenck, Pearson, Easting, & Allsopp, 1985), the Personality Research Form Impulsivity Scale (PRF; Jackson, 1984), the Multidimensional Personality Questionnaire Control Scale (MPQ; Tellegen, 1982), the Temperament and Character Inventory (TCI, Cloninger, Przybeck, & Svrakic, 1991), the Sensation Seeking Scale's (SSS-V; Zuckerman, 1994) Disinhibition (DIS) and Boredom Susceptibility (BS) scales, fourteen additional questions of impulsiveness created by the authors, and the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992). Principal components analysis revealed a four factor solution: Urgency (i.e., the impulse to act rashly to alleviate negative emotions), (lack of) Premeditation (i.e., acting without regard to the consequences), (lack of) Perseverance (i.e., inability to remain focused

on a task), and Sensation Seeking (i.e., excitement and adventure seeking; UPPS). Lack of Premeditation and lack of Perseverance were related to the NEO-PI-R's conscientiousness, Sensation Seeking to extraversion, and Urgency to neuroticism (Whiteside & Lynam, 2001). Cyders et al. (2007) added a fifth scale to the UPPS which reflects the tendency to act rashly when in a positive mood state (Positive Urgency; UPPS-P). Together, these five factors make up the UPPS-P Impulsive Behaviour Scale.

Delay discounting.

A common behavioural measure of impulsivity is the delay discounting task which asks participants to make a series of choices between smaller immediate rewards or larger rewards after various time delays (e.g., “would you rather have \$100 now or \$200 a week from now”). Delay discounting is a tendency for delayed rewards to be discounted, or devalued, compared to immediate rewards. That is, some individuals exhibit a preference for smaller immediate rewards over larger rewards after some period of time. When individuals place greater subjective value on an immediate reward even though the objective value of the delayed reward is greater, this inability to delay gratification is considered impulsive. The greater the difference in objective values and the shorter the delay to the future reward, the more impulsive one has to be to choose the smaller immediate reward (Green, Fry, & Myerson, 1994; Green & Myerson, 2010; Madden & Johnson, 2010).

A hyperbolic function has been found to describe temporal discounting more accurately than other functions (e.g., exponential), especially in psychological research with humans (Green et al., 1994; Rachlin, Raineri, & Cross, 1991; Simpson & Vuchinich, 2000; Vuchinich & Simpson, 1998). The hyperbolic function uses the equation $V = A/(1+kD)$, where V is the indifference point (or the subjective equivalent immediate value of the

delayed reward), A is the delay amount, k is the discounting rate, and D is the delay in time. The discounting rate is a constant which measures the weight an individual places on future rewards. Higher, or steeper, discounting rates imply that the individual places less value on the future reward as delay time increases, and is more likely to choose a smaller immediate reward (Mazur, 1987).

Behavioural measures of delay discounting have been found to be related to self-report measures of impulsivity (e.g., Bobova et al., 2009; Cherek, Moeller, Dougherty, & Rhoades, 1997; de Wit et al., 2007; Kirby & Petry, 2004; Richards, Zhang, Mitchell, & de Wit, 1999; Vuchinich & Simpson, 1998). However, results have been mixed with several other studies finding no significant relationship between delay discounting performance and self-report impulsivity (e.g., Crean, de Wit, & Richards, 2000; Dom, De Wilde, Hulstijn, & Sabbe, 2007; Reynolds et al., 2006; Strakowski et al., 2009). This suggests that behavioural and self-report measures likely tap different aspects of impulsivity and in studies where they are related, individuals may be high on both aspects.

Delay discounting and the UPPS-P.

While there are no known studies to date examining the relationship between delay discounting and the UPPS-P, measures that were involved in the development of the UPPS-P have been related to discounting rates. Various measures of impulsivity, and the Barratt Impulsiveness Scale (BIS) in particular, have been related to discounting rates, with higher impulsivity being related to steeper discounting rates ($d = 0.41-1.40$; Cherek et al., 1997; Kirby & Petry, 2004; Mitchell, Fields, D'Esposito, & Boettiger, 2005; Petry, 2001). BIS-11 total score and all three subscales have been positively related to discounting rates (Mitchell et al., 2005). However, a study using an older version of the BIS (10-R) found no

relationship with the Motor and Cognitive subscales (de Wit et al., 2007). All three BIS-11 subscales are reflected in the lack of Premeditation scale of the UPPS-P and the Attentional Impulsiveness scale is additionally reflected in the Negative Urgency scale (Whiteside & Lynam, 2001). This suggests that the lack of Premeditation and Negative Urgency scales will be positively related to delay discounting rates.

Discounting rates were correlated with Eysenck's I-7 Impulsiveness scale ($d = 0.41-1.09$; Kirby & Petry, 2004; Petry, 2001), which is reflected in the UPPS-P's lack of Premeditation scale (Whiteside & Lynam, 2001). There is mixed support for a relationship between discounting and UPPS-P's Sensation Seeking scale, with some studies supporting the relationship between discounting and one of Zuckerman's Sensation Seeking Scales (SSS-V Disinhibition; $d = 0.70-1.01$; Richards et al., 1999; Vuchinich & Simpson, 1998) and another study finding no relationship with any SSS-V subscale (Kirby & Petry, 2004). Based on these past findings, delay discounting rates would be expected to be positively related to the UPPS-P's lack of Premeditation scale, and to a lesser extent, the Negative Urgency and Sensation Seeking scales. There is no current evidence examining the relationship between discounting and either Positive Urgency or lack of Perseveration. However, it seems likely that higher scores on Positive Urgency would be related to steeper discounting, since it reflects the tendency to act rashly in a positive mood state and discounting appears to be state dependent, as discussed below. Clearly more examination is needed to clarify these relationships.

Impulsivity and Bipolar Disorder

Impulsivity is a common feature of many disorders in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; APA, 2013), including mania in bipolar

disorder, substance-related and addictive disorders, antisocial personality disorder, attention-deficit/hyperactivity disorder, borderline personality disorder, and disruptive, impulse-control, and conduct disorders. That impulsivity is highest in individuals with both bipolar and substance use disorders, compared to either disorder alone, ($d = 0.12-0.66$; Swann et al., 2004), suggests that there is an additive effect of comorbid disorders. Alternatively, higher impulsivity may be a risk factor for developing multiple or comorbid disorders.

UPPS-P and bipolar disorder.

Self-report impulsivity has been shown to be a stable trait across all phases of bipolar disorder; manic, euthymic, and depressed (Najt et al., 2007). Peluso et al. (2007) found that BIS-11 subscale and total scores were significantly lower in healthy controls than depressed bipolar, euthymic bipolar, and depressed unipolar participants ($d = 0.94-2.33$). The idea that impulsivity transcends mood states suggests that it is more than just a symptom of mania, and may also reflect a risk factor for bipolar disorder or an endophenotype related to bipolar disorder or a common underlying psychopathology (Peluso et al., 2007). Other studies have found that bipolar participants reported significantly more impulsivity than healthy controls on the self-report BIS-11. This pattern held for the total score ($d = 1.55$; Strakowski et al., 2010) as well as each of the subscales; Motor, Non-planning, and Attentional impulsiveness ($d = 0.65-1.77$; Ahn et al., 2011; Holmes, et al., 2009; Strakowski et al., 2009, 2010). However, there is also evidence that self-report impulsivity, as measured by the BIS-11, might be related to severity of current symptoms, with some asymptomatic bipolar participants scoring lower than syndromal participants ($d = 0.86-1.35$; Lewis, Scott, & Frangou, 2009). Using a well supported measure of subclinical traits, such as the HPS, would help clarify these discrepant findings.

There is little research on the relationship between the UPPS-P and the HSP. However, all subscales of the BIS-11 are reflected in the lack of Premeditation scale of the UPPS-P and BIS-11 Attentional impulsiveness is also reflected in the Negative Urgency scale (Whiteside & Lynam, 2001). This suggests that bipolar participants, or those higher on hypomanic personality traits, would likely score higher on the UPPS-P scales of lack of Premeditation and Negative Urgency. In addition, one study found a positive relationship between the German version of the HPS and the extraversion and openness scales of the NEO-PI (Meyer, 2002), which would suggest that the Sensation Seeking scale of the UPPS-P may also be related to HPS scores. Bipolar participants with comorbid substance use disorders reported higher levels of Positive Urgency than bipolar participants without this comorbidity ($d = 0.58$; Victor, Johnson, & Gotlib, 2011). While there was no control group in this study to confirm that higher levels of Positive Urgency are related to bipolar disorder, it seems logical that they would be highly correlated since Positive Urgency refers to rash action in response to positive affect (Cyders et al., 2007), such as mania. One study found that the HPS was most strongly positively correlated with a factor of impulsivity centered on positive emotions triggering impulsive actions ($d = 1.12$), like the Positive Urgency scale. In fact, this factor consisted of some items from the Positive Urgency scale (Johnson, Carver, Mulé, & Joormann, 2012). Therefore, it is anticipated that the HPS will be positively correlated with the UPPS-P scales lack of Premeditation, Negative Urgency, Sensation Seeking, and Positive Urgency.

Delay discounting and bipolar disorder.

Few studies have examined delay discounting in relation to bipolar disorder and hypomanic personality. A recent study, using a task similar to the current study, found that

individuals with bipolar disorder discounted hypothetical rewards more steeply than healthy controls ($d = 1.15$), indicating that they valued smaller immediate rewards more than larger delayed rewards. This was true even after controlling for age, sex, and current substance use (Ahn et al., 2011). Another study compared high- and low-HPS scorers on another measure of smaller immediate versus larger delayed rewards and found that the high-HPS group was more likely to choose the smaller immediate rewards ($d = 0.85$; Mason, O'Sullivan, Blackburn, Bentall, & El-Dereby, 2012). Both bipolar participants and high HPS scorers have been found to exhibit steeper discounting rates on various other measures of delayed reward ($d = 0.42-0.53$; Mason, El-Dereby, & Bentall, 2011; Strakowski et al., 2009, 2010). Discounting rates have not consistently been related to self-report impulsivity (Ahn et al., 2011; Crean et al., 2000) and there is evidence that this kind of behavioural impulsiveness is state-dependent, that is, subjects in manic states perform more impulsively than those in mixed or euthymic states (Najt et al., 2007; Strakowski et al., 2009, 2010). This is consistent with the idea that self-report measures tap trait impulsivity, which is different than the more state-dependent behavioural impulsivity measured by this kind of laboratory task (Dick et al., 2010). While there appears to be support for a relationship between bipolar disorder, and hypomanic personality, and steeper delay discounting rates, further evidence is needed to clarify this relationship and examine the role of self-report impulsivity as a potential mediator.

Impulsivity and Alcohol Use

UPPS-P and alcohol use.

Several studies have examined the relationships of the UPPS-P scales to alcohol use and related disorders, with some mixed results. There is support for zero order correlations

between all five UPPS-P scales and alcohol use ($d = 0.26-0.95$; Cyders et al., 2007; Miller, Flory, Lynam, & Leukefeld, 2003; Smith et al., 2007). However, scales uniquely predict different aspects of alcohol use, when controlling for the effects of the other scales. For example, lack of Premeditation and Sensation Seeking have been found to uniquely predict alcohol consumption (quantity x frequency; $d = 0.43-0.63$; Magid & Colder, 2007; Miller et al., 2003), while Negative Urgency and lack of Perseverance uniquely predicted alcohol related problems (Magid & Colder, 2007). Longitudinal studies have shown that Positive and Negative Urgency were both related to increased quantity of use and alcohol problems after eight months, whereas Sensation Seeking was related to higher frequency of consumption ($d = 0.26-0.72$; Cyders, Flory, Rainer, & Smith, 2009; Smith et al., 2007). The authors suggested that frequency of drinking may be related to the desire to seek new and exciting experiences, whereas drinking quantity and problems may be related to the tendency to act rashly while in a positive or negative mood state (Cyders et al., 2009).

A meta-analysis (Coskunpinar, Dir, & Cyders, 2013) examining how the UPPS-P scales were differentially related to different measures of alcohol use, found that lack of Perseverance was most strongly positively related to quantity of alcohol use, all scales were equally positively related to frequency of alcohol use, Negative and Positive Urgency were most strongly positively related to alcohol related problems, and Sensation Seeking was most strongly positively related to binge drinking ($d = 0.43-0.77$). A second meta-analysis found that measures related to Sensation Seeking and Positive Urgency were most strongly associated with alcohol consumption, while Positive and Negative Urgency were most strongly associated with alcohol related problems, and Sensation Seeking was most strongly associated with binge drinking ($d = 0.54-0.68$; Stautz & Cooper, 2013). In addition, Shin,

Hong, and Jeon (2012) found that Sensation Seeking and Negative Urgency, but not lack of Perseverance or lack of Premeditation, positively predicted frequency of alcohol use, alcohol related problems, binge drinking, and alcohol use disorders. Positive Urgency was not measured in this study.

Cyders and Smith (2007) found that affective states were differentially related to alcohol use. Drinking to enhance an existing positive mood (e.g., at a party) was uniquely predicted by Positive Urgency, controlling for the effects of Negative Urgency. Likewise, drinking to cope with negative affect (e.g., when depressed) was uniquely predicted by Negative Urgency, controlling for Positive Urgency. This shows that these constructs predict drinking behaviour under different circumstances. Positive Urgency may be related to alcohol use during manic episodes, or for individuals high on hypomanic traits, whereas Negative Urgency may be related to alcohol use during depressive episodes.

Alcohol use in different situations and different patterns of use appear to be differentially related to scales of the UPPS-P. Based on past research, it was expected that all scales of the UPPS-P would be positively correlated with all measures of alcohol use. However, after controlling for the relationship between scales, it was expected that Sensation Seeking would be uniquely related to frequency of use and binge drinking, (lack of) Premeditation, Positive Urgency, and Negative Urgency would each be uniquely related to quantity x frequency of alcohol use (i.e., overall alcohol consumption), and Positive Urgency, Negative Urgency, and (lack of) Perseverance would each be uniquely related to binge drinking and alcohol related problems.

Delay discounting and alcohol use.

Numerous studies have found support for a relationship between higher alcohol use and steeper discounting rates ($d = 0.19-0.72$; e.g., Bobova et al., 2009; Kollins, 2003; MacKillop et al., 2011; Petry, 2001; Vuchinich & Simpson, 1998; Yi et al., 2010). Conceptually, it makes sense that individuals who abuse alcohol have difficulty resisting immediate rewards in favour of long-term payoffs. This is seen in their drinking behaviour when they choose the immediate benefits of alcohol use over the delayed rewards of avoiding the negative consequences associated with intoxication. This same behaviour can be seen in delay discounting tasks where substance abusing individuals tend to choose the smaller immediate rewards over the larger delayed rewards (Yi et al., 2010). There is also evidence for the heritability of delay discounting and it has been suggested as a possible endophenotype for substance use and externalizing disorders (Anokhin, Golosheykin, Grant, & Heath, 2011). Further research has suggested that not only is delay discounting heritable, but there may be a genetic relationship between delay discounting and alcohol use disorders, although evidence for this is tenuous (Mitchell, 2011). Consistent with this hypothesis, one study found that individuals with a family history of substance use disorders discounted delayed rewards more steeply than those with no family history of substance use disorders. However, this relationship was mediated by antisocial traits, which uniquely predicted delay discounting rates (Acheson, Vincent, Sorocco, & Lovallo, 2011).

Research suggests that discounting is steeper with increased consumption or severity of alcohol related problems. For example, a meta-analysis supporting the relationship between higher alcohol use and steeper discounting rates found significantly greater effect sizes in clinical samples ($d = 0.50$), compared to non-clinical samples ($d = 0.26$; MacKillop

et al., 2011). College students defined as light social drinkers discounted less steeply than either heavy social drinkers or problem drinkers, with the biggest difference between light and problem drinkers, indicating steeper discounting with increased alcohol use and problems (Vuchinich & Simpson, 1998). In addition, early onset alcoholics, considered to have a more severe form of the disorder, discounted more steeply than late onset alcoholics (Dom, D'haene, Hulstijn, & Sabbe, 2006). Further support for this idea was found by Petry (2001), who reported that currently drinking alcoholics discounted more steeply than abstinent alcoholics. In addition, abstinent alcoholics had steeper discounting rates than controls (Mitchell et al., 2005), suggesting that current alcohol use may not be the primary factor in this relationship. It may be an underlying feature of individuals who develop alcohol problems that is related to delay discounting. Additional measures of problematic substance use which have been related to steeper discounting rates include an earlier age of first use for alcohol, cigarettes, and marijuana, total number of illicit drugs tried in a lifetime, and the number of times the individual passed out from alcohol use, related to heavy drinking (i.e., bingeing; Kollins, 2003).

Past studies have consistently shown a relationship between higher alcohol use and steeper delay discounting rates (Bobova et al., 2009; Kollins, 2003; Petry, 2001; Vuchinich & Simpson, 1998; Yi et al., 2010). There is also support for impaired discounting related to bipolar disorder or hypomanic personality (Ahn et al., 2011; Crean et al., 2000). Although there have been mixed results regarding the relationship of self-report impulsivity to behavioural impulsivity as measured by the delay discounting tasks, some support remains for this relationship as well (Cherek et al., 1997; Kirby & Petry, 2004; Mitchell et al., 2005). It is possible that performance on discounting tasks may be an endophenotype for both

bipolar and alcohol use disorders and that self-report impulsivity, may be the feature that links these two disorders.

Event-Related Potential Measures

In addition to personality and behavioural commonalities between bipolar and alcohol use disorders, there are also common EEG markers. EEG information is collected by recording participants' brain waves using electrodes on the scalp. The P300 event-related potential (ERP) is a positive waveform which peaks approximately 300-500ms after stimulus presentation. The P300 component is measured by evaluating its amplitude, latency, and scalp distribution. Amplitude is the voltage difference from the mean pre-stimulus baseline to the component peak. It is thought to reflect attention allocation and working memory activation; specifically updating (Donchin, 1981; Polich, 2007). P300 latency, the post-stimulus time at which the component peaks, is considered to be a measure of information processing speed (Polich, 2007; Polich & Kok, 1995). More difficult or demanding tasks are related to reduced P300 amplitudes and longer latencies (Polich, 2007). The P300 ERP is typically elicited using an oddball paradigm which requires participants to differentiate between frequently and infrequently presented stimuli. The amplitude of the P300 ERP has been found to be larger when participants are presented with the infrequent target stimuli, compared to the frequent stimuli (Donchin, 1981). A commonly used auditory oddball task requires that participants discriminate between an infrequent (target) tone and a frequent (non-target) tone. Participants are asked to press a button each time they hear the less frequently presented target tone (Polich & Kok, 1995). A commonly used visual oddball task, called the Rotated Heads task, requires that participants press a button to indicate on which side of the head (an oval) an ear (a half circle) appears (Begleiter, Porjesz, Bihari, &

Kassin, 1984). The P300 peak tends to be largest at parietal sites in both visual (Johnson, 1993) and auditory oddball tasks (Degabriele & Lagopoulos, 2009; O'Donnell, Vohs, Hetrick, Carroll, & Shekhar, 2004).

P300 amplitude reduction is the putative endophenotype that has most often been found to be related to a latent externalizing trait predictive of alcohol use (Iacono et al., 2006). However, reduced P300 amplitude may not be specific to this externalizing dimension. This reduction has also been seen in disorders such as bipolar disorder (Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009), major depression (Bruder et al., 2009; Gangadhar, Ancy, Janakiramaiah, & Umaphy, 1993), and schizophrenia (Bestelmeyer et al., 2009; Doege et al., 2009; Ford, White, Lim, & Pfefferbaum, 1994; Shin et al., 2010). Both visual and auditory paradigms have frequently been associated with alcohol use disorders (Euser et al., 2012), while the auditory paradigm had more commonly been associated with bipolar disorder (Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009). The current study examined correlates of both disorders using both auditory and visual paradigms. If the P300 ERP were negatively related to both hypomanic personality and alcohol use in both paradigms, this could lend support to the idea that there is a common genetic basis to both bipolar and alcohol use disorders.

P300 ERP and bipolar disorder.

The auditory oddball task and bipolar disorder.

A study comparing concordant and discordant monozygotic bipolar twins to control twin pairs using an auditory oddball paradigm found that P300 amplitude was significantly smaller ($d = 1.77$), and P300 latency significantly longer ($d = 0.86$), in concordant bipolar twins compared to controls. Concordant bipolar twins also had significantly smaller

amplitudes ($d = 1.01$) and longer latencies ($d = 0.71$) compared to discordant bipolar twins. Discordant bipolar twins evidenced a non-significant trend toward smaller amplitudes and longer latencies compared to controls. Heritability of P300 amplitude in this paradigm was estimated at between 75-77%. However, heritability of P300 latency was only about 21%. A significant phenotypic correlation was found between bipolar disorder and P300 amplitude ($d = 0.70$), with smaller amplitudes in bipolar individuals. However, there was no significant correlation between bipolar disorder and P300 latency, nor was there evidence of shared environmental effects. This suggests that bipolar disorder is significantly associated with smaller P300 amplitudes, but not prolonged latencies, at a genetic level and that P300 amplitude may be an endophenotype for bipolar disorder (Hall et al., 2007).

Several other studies have found support for a smaller P300 component amplitude in bipolar participants compared to controls ($d = 0.18-1.77$; Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009; Hall et al., 2007; O'Donnell et al., 2004). However, evidence regarding longer P300 latencies in bipolar individuals compared to controls has been mixed (Degabriele & Lagopoulos, 2009). Some studies have found an association ($d = 0.64-0.89$; e.g., Hall et al., 2007; O'Donnell et al., 2004; Schulze et al., 2008) and some have not (e.g., Bestelmeyer et al., 2009).

The visual oddball task and bipolar disorder.

There are fewer studies examining the visual oddball task in relation to bipolar disorder. One study found that manic participants, compared to controls, had reduced P300 amplitudes on a visual oddball task using facial emotions as the target stimuli ($d = 0.24$; Ryu, An, Jo, & Cho, 2010). A study using both auditory and visual oddball tasks (Bestelmeyer et al., 2009) found that bipolar participants had significantly lower P300

amplitudes on the auditory task compared to controls, and there was a non-significant trend for an amplitude difference on the visual task. However, this study used a simple (Xs and Os) visual paradigm and there is evidence that more complicated (e.g., rotated heads) paradigms better elicit a significant reduction in P300 amplitude (Polich, 2007). No latency differences were found in this study (Bestelmeyer et al., 2009). Another study noted that bipolar participants in a depressive episode had longer P300 latencies ($d = 1.90-2.19$), in a visual oddball paradigm, compared to controls, but no difference was found on amplitude. Reaction time and accuracy for the bipolar participants were also significantly worse (i.e., slower) than the control group (Bange & Bathien, 1998). With so many other commonalities between bipolar and alcohol use disorders, it seems surprising that a measure commonly examined with relation to alcohol use has been largely unexamined with regard to bipolar disorder.

P300 ERP and alcohol use.

The auditory oddball task and alcohol use.

Compared to bipolar disorder, there is much more research to support the relationship of P300 amplitude to alcohol use disorders. This is true for both visual and auditory paradigms and for individuals with, or at risk for, alcohol related disorders ($d = 0.28-0.60$; Euser et al., 2012). Using an auditory oddball task, significant P300 amplitude reductions were found in a group of non-alcoholic young adult males at high-risk of developing alcohol related disorders (i.e., their fathers were alcohol dependent), compared to low-risk controls (i.e., no familial history of alcohol dependence). The reduction was greatest in frontal and occipital regions, but reductions were also found at some central and parietal sites. There were no significant differences in P300 latency at any site (Ramachandran, et al., 1996).

In a longitudinal study, smaller P300 amplitudes in an auditory oddball task, measured in early adolescence, predicted substance use disorders in early adulthood ($d = 0.59$; Hill et al., 2009). In addition, recently abstinent male alcoholics, compared to controls, had P300 amplitude reductions on an auditory oddball task in frontal, central, parietal, occipital, and temporal areas ($d = 0.74-1.17$) but there were no group differences for P300 latency in any region. Genetic support for the P300 amplitude reduction was found when the abstinent alcoholics were split into two groups, those with and without alcoholic relatives. Alcoholics with alcoholic relatives had significantly reduced P300 amplitudes in all five areas compared to controls, while no significant difference was found between controls and alcoholics without alcoholic relatives (Cohen, Wang, Porjesz, & Begleiter, 1995). These studies suggest that P300 amplitude reductions in an auditory paradigm may be an endophenotype for alcohol disorders where heritability is high.

The visual oddball task and alcohol use.

Substance use has been significantly associated with P300 amplitude reductions in visual oddball tasks ($d = 0.33-0.60$; e.g., Chen et al., 2007; Euser et al., 2012; Iacono, Malone, & McGue, 2003; Polich, Pollock, & Bloom, 1994; Porjesz & Begleiter, 1990). Severity of the disorder may also be a factor; smaller P300 amplitude in a visual oddball task was related to earlier age of onset of substance use problems, compared to later-onset, in a male community sample (Carlson, McLarnon, & Iacono, 2007). Reduced P300 amplitudes have also been found in youth at risk of developing alcohol related disorders (e.g., children of alcoholics) using both visual (Polich et al., 1994) and auditory tasks (Ramachandran et al., 1996). However, the effect has been more consistent with visual than auditory tasks (Polich et al., 1994; Ramachandran et al., 1996). Reduced P300 amplitude in a visual oddball task

was found in alcoholics, compared to non-alcoholics, in unaffected relatives of alcoholics, compared to relatives of controls, and in unaffected offspring of alcoholic fathers, compared to offspring of controls (Hesselbrock, Begleiter, Porjesz, O'Connor, & Bauer, 2001). Risk for alcoholism, that is, being the non-alcoholic child of an alcoholic, even if raised by adoptive parents, has been shown to be related to reduced P300 amplitude in a visual Rotated Heads oddball task (Begleiter et al., 1984), especially in male children of fathers with alcohol related problems (Polich et al., 1994). Using both auditory and visual oddball tasks, recently abstinent alcoholic men with a positive family history of alcohol problems, demonstrated smaller P300 amplitudes than alcoholics with no family history of alcohol problems, whose amplitudes were not significantly different than controls (Pfefferbaum, Ford, White, & Mathalon, 1991). Therefore, reduced P300 amplitude appears to be related to the genetic risk for alcohol related disorders, and externalizing more generally (Gilmore, Malone, & Iacono, 2010; Hicks et al., 2007), rather than level of past alcohol consumption.

A genetic influence on P300 amplitude in a visual rotated heads task was found in a study comparing male and female twins who were either discordant or concordant for alcohol use disorders (i.e., abuse or dependence), and control twins with no alcohol use disorder or other related externalizing disorder (i.e., drug abuse/dependence, nicotine dependence, Conduct Disorder, Attention Deficit/Hyperactivity Disorder, or Oppositional Defiant Disorder). Compared to unaffected twin pairs (no disorder in either twin) P300 amplitude was reduced in both discordant (one twin had an alcohol use disorder) and concordant (both twins had an alcohol use disorder) affected twin pairs. Affected and unaffected members of discordant twin pairs did not differ from each other and had comparable correlations in P300 reductions as concordant affected twins. There were no significant within-pair or between-

subjects findings for latency or reaction time (Carlson, Iacono, & McGue, 2002). This suggests that P300 amplitude is a good candidate biological marker for alcohol use disorders (Carlson et al., 2002; Perlman, Johnson, & Iacono, 2009; Porjesz et al., 1998, 2005).

Consistent with the idea that P300 amplitude reductions may be a risk marker for alcohol related disorders, longitudinal studies using the visual rotated heads task, have found that reduced P300 amplitude in adolescence predicted substance use disorders by late adolescence or early adulthood (Hill, Shen, Lowers, & Locke, 2000; Hill et al., 2009; Iacono, Carlson, Malone, & McGue, 2002). Additionally, a meta-analysis examining twin studies of EEG measures determined that the heritability of the P300 ERP was 60% for amplitude and 51% for latency (van Beijsterveldt & van Baal, 2002). Further support for the heritability of the P300 amplitude has been found in several other twin and family studies (e.g., Euser et al., 2012; Katsanis, Iacono, McGue, & Carlson, 1997; Perlman et al., 2009; Polich et al., 1994).

P300 ERP and impulsivity.

The auditory oddball task and impulsivity.

Findings from studies investigating the relationship between impulsivity and P300 amplitude have been mixed (e.g., Martin & Potts, 2004), although some support has been found for a negative relationship between BIS-11 scores and P300 amplitude on visual ($d = 0.43-0.98$; Barratt, Stanford, Kent, & Felthous, 1997; Chen et al., 2007; Harmon-Jones, Barratt, & Wigg, 1997) and auditory ($d = 0.90$; Moeller et al., 2004) oddball tasks. In a sample of cocaine-dependent individuals and controls, P300 amplitude in an auditory oddball task was negatively correlated with BIS-11 total score, even after controlling for age, gender, education, and the number of conduct disorder symptoms experienced in childhood (Moeller et al., 2004). Since the BIS-11 subscales are reflected in the UPPS-P scales of lack of

Premeditation and, to a lesser extent, Negative Urgency (Whiteside & Lynam, 2001), it was hypothesized that these scales will be most strongly related to P300 amplitude reduction in the auditory oddball task.

The visual oddball task and impulsivity.

In a visual oddball task, BIS-11 total score and the Motor Impulsiveness subscale were significantly negatively related to P300 amplitude at the Pz and Fz sites ($d = 0.68-0.90$). Motor Impulsiveness was also significantly negatively related to P300 amplitude at Cz ($d = 0.85$). Higher levels of impulsivity were related to smaller P300 amplitude. However, after controlling for full scale intelligence scores, the relationships between total score and amplitude were no longer significant. The authors suggested that reduced P300 amplitude in individuals high on impulsivity may represent less efficient inhibition of task-irrelevant information which may also reduce cognitive performance (Russo, De Pascalis, Varriale, Barratt, 2008). BIS-11 total score was also found to be negatively related to P300 amplitude in a visual oddball task in a sample of prison inmates and controls ($d = 0.43-0.98$; Barratt et al., 1997). In a visual oddball paradigm, impulsivity, as measured by a composite of the Eysenck Impulsivity/Venturesomeness scale and the Control subscale of the Multidimensional Personality Questionnaire, was significantly negatively related to P300 amplitude, at the Pz, Cz, and Fz sites, in men but not women ($d = 0.49-0.98$; Justus, Finn, & Steinmetz, 2001). In a study of adolescents using a visual oddball task, P300 amplitude was negatively correlated with BIS-11 Attentional Impulsiveness and Non-planning Impulsiveness in parietal regions ($d = 0.52-0.80$; Harmon-Jones et al., 1997). These findings together suggest that the UPPS-P lack of Premeditation scale will be most strongly related to

P300 amplitude reductions in the visual paradigm, with some support also for relationships with Negative Urgency and lack of Perseverance.

Impulsivity as a Link Between Bipolar Disorder and Alcohol Use Disorders

While toxicological effects of alcohol use can lead to increased impulsivity (Haugen Light, 1986) and possibly resemble some of the symptoms of bipolar disorder, there is evidence that these features can also precede the onset of alcohol use (Caspi et al., 1996; Granö et al., 2004; Jones, 1968, 1971). This suggests that alcohol use does not necessarily lead to the development of impulsivity or bipolar disorder. Therefore, the high comorbidity between the two disorders would suggest a common etiology or vulnerability to both disorders (Goldberg, 2001; Preuss & Wong, 2000; Swann, 2010), or a common mechanism influenced by different risk factors. More than half of individuals with bipolar disorder have a lifetime history of substance abuse (Goldberg, 2001; Preuss & Wong, 2000; Regier, et al., 1990; Swann et al., 2004; Tohen & Zarate, 1999). For many individuals, alcohol use disorders precede the onset of bipolar disorder and can even mask symptoms, making diagnosis difficult or delayed (Goldberg, 2001). However, in a large number of individuals, bipolar disorder precedes the onset of the substance related disorder (Tohen & Zarate, 1999). This non-linear relationship of the disorders suggests that there may be a shared vulnerability affecting both disorders.

Bipolar and alcohol use disorders have evidenced similar relationships with measures of hypomanic and impulsive personality, delay discounting, and P300 ERP amplitude reductions. In addition, self-report impulsivity has been related to delay discounting and P300 amplitude reductions. These findings, together with the common heritability of bipolar and alcohol use disorders and impulsivity (Goldberg, 2001; Preuss & Wong, 2000; Swann,

2010) suggest that impulsivity may be an important key to finding the underlying link between bipolar and alcohol use disorders. This is not to say that everyone with bipolar disorder will develop an alcohol-related disorder, or vice versa. However, it appears that there are overlapping traits common to both disorders that lead to worse outcomes. People with alcohol-related disorders may have traits related to bipolar disorder, such as affective instability and reward sensitivity, which can lead to problem levels of drinking. Identifying these common traits may allow us to target interventions to the specific areas of risk. For example, individuals high on Negative Urgency, but not Sensation Seeking could be taught effective ways to deal with negative emotions rather than using alcohol as a coping mechanism, and individuals high on Positive Urgency, or Sensation Seeking, but not Negative Urgency, may benefit from education about less risky ways to enhance their positive experiences or feel excitement (e.g., running instead of drinking).

Overview of Studies

The current studies aimed to examine measures which have commonly been related to both bipolar and alcohol use disorders in undergraduate student drinkers at various levels of hypomanic personality. Participants completed self-report measures of hypomanic personality, impulsivity, and alcohol use and problems, a behavioural measure of impulsivity, and ERP measures that have shown sensitivity to bipolar or alcohol disordered individuals. If there is a common etiology for bipolar and alcohol use disorders, it would stand to reason that results previously found in either disorder should replicate in the other, or in individuals at risk for either disorder. In addition, self-report impulsivity was examined as a potential mediator and link between the two disorders.

Chapter 2: Study 1

Introduction and Hypotheses

The first study was an online questionnaire study asking undergraduate students to complete measures of hypomanic and impulsive personality, and alcohol use and problems. It was hypothesized that HPS scores would predict all measures of alcohol use and the lack of Premeditation, Negative Urgency, Sensation Seeking, and Positive Urgency scales of the UPPS-P. It was also predicted that UPPS-P scales would predict alcohol use, with higher impulsivity related to greater alcohol use and problems. Specifically, based on past research, it was expected that Sensation Seeking would be uniquely related to frequency of use and binge drinking frequency; lack of Premeditation, Positive Urgency, and Negative Urgency would each be uniquely related to quantity x frequency of alcohol use; and Positive Urgency, Negative Urgency, and lack of Perseverance would each be uniquely related to binge drinking and alcohol related problems. It was anticipated that the relationships between the HPS and alcohol use would be mediated by the UPPS-P scales related to the different patterns of alcohol use. That is to say that the relationships between hypomanic traits and alcohol use measures would be mediated by different aspects of impulsivity. Table 1 outlines the measures used in Study 1.

Table 1

Measures Used in Study 1

Hypomanic Personality Scale (HPS)	UPPS-P Impulsive Behaviour Scale	Alcohol Use measures
HPS Total Score	Negative Urgency	Lifetime Maximum Number of Drinks Within a 24-hour Period
Social Vitality	Lack of Premeditation	Quantity of Alcohol Use
Mood Volatility	Lack of Perseverance	Frequency of Alcohol Use
Excitement	Sensation Seeking	Quantity x Frequency
	Positive Urgency	Binge Drinking Frequency
		Alcohol Related Problems (SMAST)

Method

Participants.

One thousand one hundred and ninety four undergraduate student drinkers participated in this online questionnaire study. Participants (67% female) aged 19 to 59 ($M = 21.11$; $SD = 3.23$) had consumed at least one alcoholic beverage in the past 12 months. Fifty-four percent described themselves as European or of European descent, while another 36% described themselves as East Asian or of East Asian descent (see Appendix 1 for a brief discussion of ethnicity differences). Other ethnicities represented included: African, First Nations, Indian-South Asian, Latin American-Hispanic, Middle Eastern, or a combination of more than one. Participants were recruited using the University of British Columbia Psychology Department's human subject pool system, and participants received course credit for their participation in the study. Participants provided informed consent and all procedures were approved by the appropriate institutional ethics review board. Participants

were excluded from analysis if their survey was incomplete, they failed to get debriefed, the participant had not consumed at least one alcoholic beverage in the 12 months prior to the study, the survey had obviously been completed inaccurately (e.g., multiple questionnaires were answered with only one response, such as all “true”), or if the participant completed the survey twice, only their first responses were used. Additional participants were excluded from analysis if they reported speaking English for less than five years or if they failed to endorse two or more items on the HPS validity scale, suggesting that they did not accurately read and interpret the scale items and may not have completed the full survey appropriately.

Procedure.

Participants completed questionnaires using an online survey administration method on the website www.SurveyMonkey.com, and then attended a debriefing session in person. The first page of the online survey was a consent form, and participants had to agree to participate in the study before being granted access to the survey. The following questionnaires were completed by participants: a demographic questionnaire, the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986), the UPPS-P Impulsive Behaviour Scale (Whiteside & Lynam, 2001; Cyders et al., 2007), an alcohol use questionnaire developed from the questions recommended by the National Council on Alcohol Abuse and Alcoholism’s Task Force on Recommended Alcohol Questions (National Institute of Health, 2003), and the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975).

Measures.

Personality questionnaires.

Hypomanic Personality Scale.

The HPS (Eckblad & Chapman, 1986) is a self-report measure of hypomanic personality features and experiences consisting of 53 true or false items such as “I often feel excited and happy for no apparent reason” and “I often get into excited moods where it’s almost impossible for me to stop talking”. The original 48 items, some reverse scored, are summed to form a total score. The remaining 5 items are validity items to confirm accurate responding. Schalet, Durbin, and Revelle (2011) identified three factors in the HPS: Social Vitality (22 items), Mood Volatility (15 items), and Excitement (8 items) and suggested that these subscales be employed in future analyses. Social Vitality refers to social potency and vivaciousness. Mood Volatility is a measure of negative and unpredictable moods and hypomanic cognitions. Excitement refers to an energetic and cheerful mood. Three items, 12, 24, and 28, were excluded from these subscales due to lack of fit.

Internal consistency of the HPS total score was found to be (coefficient-alpha reliability) .87 (Eckblad & Chapman, 1986; Schalet et al., 2011) and 15 week test-retest reliability was .81 (Eckblad & Chapman, 1986). Internal consistency of the subscales was .79 (Social Vitality), .79 (Mood Volatility), and .87 (Excitement; Schalet et al., 2011).

The HPS total score has been found to correlate with hypomanic episodes, alcohol and drug use, poorer social functioning and schizotypal traits (Eckblad & Chapman, 1986). It has also been shown to predict bipolar and alcohol use disorders longitudinally (Kwapil et al., 2000). When subscales were examined individually, the Mood Volatility subscale was the scale most strongly related to measures of negative emotionality and internalizing

symptoms, such as anxiety and depression. It was the only scale significantly related to poorer social functioning, particularly in the areas of work, family, school, and finances. The Social Vitality subscale was the scale most strongly related to measures of positive emotionality (e.g., extraversion and social closeness), openness, sexual activity and substance use. The Excitement subscale was also significantly related to positive emotionality measures (Schalet et al., 2011).

UPPS-P Impulsive Behaviour Scale.

The UPPS-P Impulsive Behaviour Scale (Whiteside & Lynam, 2001; Cyders et al., 2007) is 59-item self-report measure of impulsive personality. Participants respond to questions such as “I have trouble controlling my impulses” and “when I am upset I often act without thinking” using a four point Likert-type scale ranging from “Strongly Agree” to “Strongly Disagree”. The UPPS-P measures five facets of impulsivity, four of which were derived from principal components analysis of commonly used self-report measures of impulsivity and correspond to facets of the Five Factor Model of Personality. Negative Urgency (12 items) refers to a tendency to act rashly when in a negative, or distressed, affective state. (Lack of) Premeditation (11 items) is the propensity to act without regard to the consequences. (Lack of) Perseverance (10 items) measures an inability to remain focused on a task. Sensation Seeking (12) refers to an inclination to seek out novel and exciting experiences (Whiteside & Lynam, 2001). The last factor, Positive Urgency (14 items), was derived by Cyders et al. (2007) and measures the tendency to act rashly when in a positive affective state.

Internal consistency for the scales was found to be .86 (Negative Urgency), .91 (lack of Premeditation), .82 (lack of Perseverance), .90 (Sensation Seeking; Whiteside & Lynam,

2001), and .94 (Positive Urgency; Cyders et al., 2007). Test-retest reliability over an eight month period ranged from .50 (lack of Perseverance) to .64 (Sensation Seeking; Cyders et al., 2009).

Alcohol use questionnaires.

The alcohol use questionnaire in this study was developed from questions recommended by the National Institute on Alcohol Abuse and Alcoholism's Task Force on Recommended Alcohol Questions (National Institute of Health, 2003). Participants answered eight self-report questions about their average frequency and quantity of alcohol consumption in the past 12 months, the maximum number of drinks consumed within a 24-hour period in the past 12 months, the frequency of this maximum number of drinks, the frequency of binge drinking in the past 12 months (defined as 4 or more drinks in a 2-hour period for women and 5 or more drinks in a 2-hour period for men), lifetime maximum number of drinks in a 24-hour period, age at first drink, and age at first intoxication. A drink is defined as half an ounce (30 ml) of absolute alcohol (i.e., a 12 ounce/355 ml can or glass of beer or cooler, a 5 ounce/150 ml glass of wine, or a drink containing 1 shot of liquor).

The average number of occasions per week or month when the participant consumed alcohol was used to determine a yearly average number of occasions on which the participant consumed alcohol. This number was then multiplied by the average number of drinks consumed by the participant per occasion in the past 12 months, in order to get a total consumption value for the year (e.g., Conrad, Petersen, & Pihl, 1997; Finn, Bobava, Wehner, Fargo, & Rickert, 2005; Finn & Hall, 2004; Hittner & Swickert, 2006). Previous studies have used a measure of quantity x frequency to determine alcohol consumption levels in undergraduate samples (e.g., Carlson et al., 2010; Henderson, Goldman, Coovert, &

Carnevalla, 1994; Schwarz, Burkhart, & Green, 1978). In addition, a longitudinal twin study found that the heritability of the mean alcohol consumption was 70% and that test-retest reliability for a similar quantity x frequency measure ranged from .56 over a 13 year retest interval to .69 over a four year retest interval (Whitfield, et al., 2004).

Short Michigan Alcoholism Screening Test.

The SMAST (Selzer et al., 1975) is a brief, 13-item measure of alcohol-related problems. It is a short version of the self-report Michigan Alcoholism Screening Test (MAST; Selzer, 1971), which was originally a structured interview for the detection of alcoholism. The internal consistency of the SMAST has been found to range from a Cronbach's alpha of .57, across two samples of addiction facility patients and their families (Fleming & Barry, 1989) to .93 for a sample of alcoholics combined with a community sample of people who were renewing their driver's license (Selzer et al., 1975). A 10-day test-retest period yielded a correlation of .87 in the two addiction facility samples (Fleming & Barry, 1989).

Statistical analyses.

Data were analyzed using multiple regression techniques for examining multiple mediators (Preacher & Hayes, 2008). To begin, distributional assumptions were evaluated to determine if variables were normally distributed or if they needed to be transformed. Key variables used in analyses included age, sex (0=female, 1=male), the HPS total score and subscale scores, the five UPPS-P scales, past year alcohol quantity, frequency, and quantity x frequency, lifetime maximum number of drinks in a 24-hour period, binge drinking frequency, and alcohol related problems. All measures of alcohol use were positively skewed and a natural log transformation was used to produce a reasonably normal

distribution. First order correlations were examined. Next, regression analyses were conducted predicting alcohol variables from all of the HPS scales and separate analyses were run for HPS total score, and then all of the UPPS-P scales. All analyses controlled for age and sex, where indicated by first order correlations. For each of the HPS scales which uniquely predicted alcohol use, UPPS-P scales which also uniquely predicted alcohol use were examined as mediators in the HPS -> alcohol use relationship.

Lastly, diagnostics and assumptions were checked. Outliers were examined for leverage and influence and analyses were run with and without outliers to determine if they had a strong effect on results. No outliers needed to be excluded from final analyses. Assumption examinations, which in multiple regression analysis include homoscedasticity, normality of errors, fixed predictors, correctly specified functional relationship, and independent errors, were also examined and found to be reasonable.

Results

Due to the large number of variables being examined, alpha was set at $p = .01$ to reduce the likelihood of a Type I error occurring. Table 2 shows the means and standard deviations for all Study 1 variables. First order correlations between all Study 1 variables are presented in Table 3. Where age and/or sex were correlated with any measure involved in regression analyses, the relevant demographic variable(s) was included as a covariate in these analyses. Males scored significantly higher than females on all measures of alcohol use, HPS total score, Social Vitality, and Mood Volatility, as well as UPPS-P Sensation Seeking and Positive Urgency. Females scored significantly higher than males on UPPS-P Negative Urgency.

Table 2*Means and Standard Deviations for all Study 1 Variables*

	<i>N</i>	Untransformed		Log Transformed	
		Mean	SD	Mean	SD
Age	1194	21.11	3.23		
Alcohol Maximum	1194	12.53	7.91	2.42	0.64
Alcohol Quantity	1194	4.41	3.07	1.55	0.52
Alcohol Frequency	1194	58.73	62.16	3.52	1.17
Quantity x Frequency	1194	296.56	434.78	4.75	1.58
Binge Frequency	1189	19.20	31.33	2.01	1.46
Alcohol Problems	1194	1.14	1.52	0.56	0.61
HPS Total	1194	15.33	8.82		
HPS Social Vitality	1194	7.30	4.36		
HPS Mood Volatility	1194	5.14	3.62		
HPS Excitement	1194	1.98	2.19		
UPPS-P Negative Urgency	1194	2.25	0.54		
UPPS-P Premeditation	1194	1.97	0.43		
UPPS-P Perseverance	1194	2.03	0.47		
UPPS-P Sensation Seeking	1194	2.73	0.59		
UPPS-P Positive Urgency	1194	1.79	0.56		

Note: Alcohol Maximum = the lifetime maximum number of drinks within a 24-hour period, Alcohol Quantity = the average quantity of alcohol consumed per occasion in the past 12 months, Alcohol Frequency = the average frequency of alcohol consumption in the past 12 months, Quantity x Frequency = the quantity x frequency of drinks consumed in the past 12 months, Binge Frequency = the frequency of binge drinking in the last 12 months, Alcohol Problems = the Short Michigan Alcoholism Screening Test, HPS Total = Hypomanic Personality Scale Total Score, HPS Social Vitality = Hypomanic Personality Scale Social Vitality, HPS Mood Volatility = Hypomanic Personality Scale Mood Volatility, HPS Excitement = Hypomanic Personality Scale Excitement, UPPS-P Negative Urgency = UPPS-P Impulsive Behaviour Scale Negative Urgency, UPPS-P Premeditation = UPPS-P Impulsive Behaviour Scale Lack of Premeditation, UPPS-P Perseverance = UPPS-P Impulsive Behaviour Scale Lack of Perseveration, UPPS-P Sensation Seeking = UPPS-P Impulsive Behaviour Scale Sensation Seeking, UPPS-P Positive Urgency = UPPS-P Impulsive Behaviour Scale Positive Urgency.

Table 3*First Order Correlations for Study 1 (N=1194, except for Binge correlations N=1189)*

	Age	Sex	Alc Max	Alc Quant	Alc Freq	Quant x Freq	Binge	SMAST	HPS Total	HPS Soc Vit	HPS Mood Vol	HPS Excitement	UPPS-P Neg Urg	UPPS-P Premed	UPPS-P Persev	UPPS-P SS	UPPS-P Pos Urg
Age	.00		.05	-.15***	.08**	.00	-.12***	.03	-.04	.02	-.06*	-.10***	-.03	.07*	.05	-.06	-.07*
Sex			.27***	.19***	.16***	.20***	.10***	.12***	.13***	.19***	.09***	.01	-.07*	-.04	.01	.25***	.13***
Alc Max				.62***	.59***	.71***	.63***	.11***	.21***	.56***	.13***	.08**	.12***	.16***	.05	.33***	.11***
Alc Quant					.36***	.70***	.66***	.17***	.19***	.20***	.15***	.13***	.16***	.13***	.06*	.23***	.17***
Alc Freq						.92***	.61***	.06*	.20***	.24***	.12***	.11***	.12***	.17***	.04	.29***	.11***
Quant x Freq							.74***	.11***	.23***	.27***	.15***	.14***	.16***	.19***	.06	.32***	.15***
Binge								.17***	.23***	.22***	.16***	.18***	.20***	.19***	.09**	.28***	.19***
SMAST									.24***	.18***	.23***	.19***	.23***	.14***	.14***	.06*	.25***
HPS Total										.84***	.84***	.79***	.40***	.31***	.04	.41***	.50***
HPS Soc Vit											.47***	.51***	.16***	.21***	-.09**	.43***	.26***
HPS Mood Vol												.62***	.52***	.27***	.16***	.29***	.54***
HPS Excitement													.34***	.34***	.05	.27***	.47***
UPPS-P Neg Urg														.39***	.35***	.31***	.64***
UPPS-P Premed															.40***	.33***	.41***
UPPS-P Persev																-.03	.29***
UPPS-P SS																	.27***
UPPS-P Pos Urg																	

Note: Alc Max = the natural log transformation of the lifetime maximum number of drinks within a 24-hour period, Alc Quant = the natural log transformation of the average quantity of alcohol consumed per occasion in the past 12 months, Alc Freq = the natural log transformation of the average frequency of alcohol consumption in the past 12 months, Quant x Freq = the natural log transformation of the quantity x frequency of drinks consumed in the past 12 months, Binge = the natural log transformation of the frequency of binge drinking in the last 12 months, SMAST = the natural log transformation of the Short Michigan Alcoholism Screening Test, HPS Total = Hypomanic Personality Scale Total Score, HPS Soc Vit = Hypomanic Personality Scale Social Vitality, HPS Mood Vol = Hypomanic Personality Scale Mood Volatility, HPS Excitement = Hypomanic Personality Scale Excitement, UPPS-P Neg Urg = UPPS-P Impulsive Behaviour Scale Negative Urgency, UPPS-P Premed = UPPS-P Impulsive Behaviour Scale Lack of Premeditation, UPPS-P Persev = UPPS-P Impulsive Behaviour Scale Lack of Perseveration, UPPS-P SS = UPPS-P Impulsive Behaviour Scale Sensation Seeking, UPPS-P Pos Urg = UPPS-P Impulsive Behaviour Scale Positive Urgency.

* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$.

Hypomanic personality.

In general, higher levels of hypomanic personality were related to more alcohol use and problems and higher levels of impulsivity. The HPS total score and all three subscales were positively correlated with all measures of alcohol use (lifetime maximum number of drinks within a 24-hour period, quantity of alcohol use, frequency of alcohol use, quantity x frequency, binge drinking frequency, and alcohol related problems; $.08 \leq r \leq .56$, $.005 \leq p \leq .001$) and the Negative Urgency, lack of Premeditation, Sensation Seeking, and Positive Urgency scales of the UPPS-P ($.16 \leq r \leq .54$, $p \leq .001$). The UPPS-P lack of Perseverance scale was only positively correlated with HPS Mood Volatility ($r = .16$, $p \leq .001$), it was negatively correlated with HPS Social Vitality ($r = -.09$, $p = .002$) and unrelated to HPS total score or Excitement.

According to Baron and Kenny (1986), in order to show a mediation effect we first need to show that hypomanic personality significantly predicts alcohol use, using a regression analysis. This establishes that there is an effect to be mediated. Next, we need to show that hypomanic personality significantly predicts impulsivity using a regression analysis. Third, we need to show that impulsivity predicts alcohol use in a multiple regression analysis controlling for hypomanic personality. Lastly, for full mediation, we need to establish that the direct relationship between hypomanic personality and alcohol use is no longer significant when impulsivity is included in the model. The steps of this process are described below.

When HPS total score was entered into regression analyses predicting each measure of alcohol use and controlling for the effects of age and sex, where relevant, HPS total score was found to uniquely positively predict each measure of alcohol use above and beyond the effects of age and sex ($.17 \leq \beta \leq .23$, $p \leq .001$), consistent with my hypotheses. When all HPS subscales were entered into regression analyses predicting each measure of alcohol use and controlling for

the effects of age and sex, only Social Vitality uniquely predicted alcohol use frequency ($\beta = .21, p \leq .001$), quantity ($\beta = .14, p \leq .001$), quantity x frequency ($\beta = .22, p \leq .001$), lifetime maximum number of drinks in 24-hours ($\beta = .22, p \leq .001$), and binge drinking frequency ($\beta = .16, p \leq .001$). Mood Volatility ($\beta = .15, p \leq .001$) uniquely predicted alcohol related problems (SMAST). Table 4 summarizes the results of these regression analyses.

When HPS total score was entered into regression analyses predicting each scale of the UPPS-P and controlling for the effects of age and sex, where relevant, HPS total score was found to uniquely positively predict each scale of the UPPS-P above and beyond the effects of sex ($.32 \leq \beta \leq .49, p \leq .001$), except for lack of Perseverance which was unrelated. This was consistent with my hypotheses. When all relevant HPS subscales were entered into regression analyses predicting each UPPS-P scale and controlling for the effects of age and sex where relevant, Social Vitality and Mood Volatility uniquely predicted Negative Urgency ($\beta = -.10, p \leq .001$; $\beta = .54, p \leq .001$, respectively). However, Social Vitality was negatively related and Mood Volatility was positively related. Mood Volatility uniquely positively predicted lack of Perseverance ($\beta = .16, p \leq .001$). Social Vitality also uniquely positively predicted Sensation Seeking ($\beta = .35, p \leq .001$). Mood Volatility and Excitement uniquely predicted lack of Premeditation ($\beta = .10, p = .005$; $\beta = .27, p \leq .001$, respectively), both positively. Lastly, Social Vitality (negatively), Mood Volatility (positively) and Excitement (positively) all uniquely predicted Positive Urgency ($\beta = -.08, p = .005$; $\beta = .40, p \leq .001$; $\beta = .26, p \leq .001$, respectively). Table 5 summarizes the results of these regression analyses.

Table 4*Summary of Regression Results for Study 1 Predicting Alcohol Use*

Dependent Variable	Alcohol Use	Lifetime Maximum	Quantity	Frequency	Quantity x Frequency	Binge Drinking	Problems (SMAST)
Unique Predictors	Hypomanic Personality Scale	Total Score (+) Soc Vitality (+)	Total Score (+) Soc Vitality (+)	Total Score (+) Soc Vitality (+)	Total Score (+) Soc Vitality (+)	Total Score (+) Soc Vitality (+)	Total Score (+) Mood Volatility (+)
	UPPS-P	Neg Urg (+) Premed (+) SS (+) Pos Urg (-)	Neg Urg (+) SS (+)	Neg Urg (+) Pos Urg (+)			

Note: HPS and UPPS-P scales listed above were found to be unique predictors of each alcohol use measure after controlling for the effects of age and sex where relevant. All 18 models examined had at least one significant predictor. Alpha was set at $p = .01$ for significance. The (+) or (-) denotes whether the variable was positively or negatively related to alcohol use. Soc Vitality = Social Vitality, UPPS-P = UPPS-P Impulsive Behaviour Scale, Neg Urg = Negative Urgency, Premed = lack of Premeditation, SS = Sensation Seeking, Pos Urg = Positive Urgency.

Table 5*Summary of Regression Results for Study 1 Predicting Impulsivity*

Dependent Variable	UPPS-P	Negative Urgency	Lack of Premeditation	Lack of Perseverance	Sensation Seeking	Positive Urgency
Unique Predictors	HPS	Total Score (+) Social Vitality (-) Mood Volatility (+)	Total Score (+) Mood Volatility (+) Excitement (+)	 Mood Volatility (+)	Total Score (+) Social Vitality (+)	Total Score (+) Social Vitality (-) Mood Volatility (+) Excitement (+)

Note: HPS scales listed above were found to be unique predictors of each UPPS-P Scale after controlling for the effects of age and sex where relevant. Nine of the 10 models examined had at least one significant predictor. Alpha was set at $p = .01$ for significance. The (+) or (-) denotes whether the variable was positively or negatively related to the UPPS-P Scale. HPS = Hypomanic Personality Scale, UPPS-P = UPPS-P Impulsive Behaviour Scale.

Impulsivity.

UPPS-P Negative Urgency, lack of Premeditation, and Positive Urgency scales were all positively correlated with all measures of alcohol use ($.11 \leq r \leq .25, p \leq .001$). Sensation Seeking was positively correlated with all measures of alcohol use ($.23 \leq r \leq .33, p \leq .001$) except for alcohol related problems. Higher levels of impulsivity were related to more alcohol use and problems. UPPS-P lack of Perseverance was positively correlated with binge drinking frequency ($r = .09, p = .002$) and alcohol related problems (SMAST; $r = .14, p \leq .001$), but only marginally related to alcohol use quantity ($r = .06, p = .036$) and quantity x frequency ($r = .06, p = .051$) and unrelated to lifetime maximum number of drinks within a 24-hour period and frequency of alcohol use.

When all relevant UPPS-P scales were entered into regression analyses predicting each measure of alcohol use and controlling for the effects of age and sex where relevant, Sensation Seeking ($\beta = .25, p \leq .001$), Negative Urgency ($\beta = .14, p \leq .001$), lack of Premeditation ($\beta = .09, p = .006$), and Positive Urgency ($\beta = -.11, p = .002$) all uniquely predicted lifetime maximum number of drinks in a 24-hours period. Interestingly, Positive Urgency was negatively related to alcohol use, whereas the other variables were positively related. Sensation Seeking ($\beta = .24, p \leq .001$) and Negative Urgency ($\beta = .12, p \leq .001$) both uniquely positively predicted alcohol use frequency. Sensation Seeking ($\beta = .15, p \leq .001$) and Negative Urgency ($\beta = .12, p \leq .001$) both uniquely positively predicted alcohol use quantity. Sensation Seeking ($\beta = .25, p \leq .001$) and Negative Urgency ($\beta = .15, p \leq .001$) both uniquely positively predicted quantity x frequency of alcohol use. Sensation Seeking ($\beta = .23, p \leq .001$) and Negative Urgency ($\beta = .15, p \leq .001$) both uniquely positively predicted binge drinking frequency. Lastly, Negative Urgency ($\beta = .12, p =$

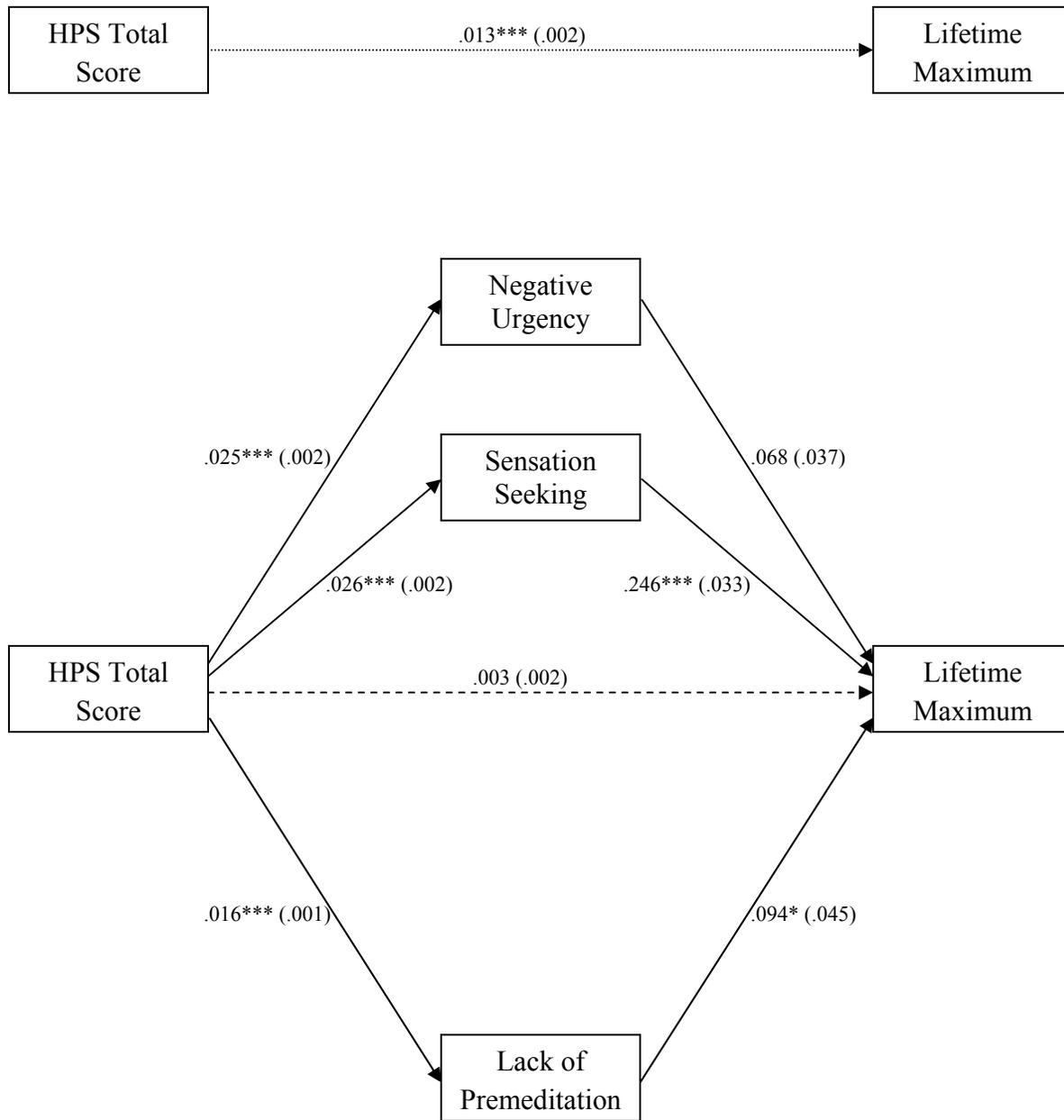
.002) and Positive Urgency ($\beta = .14, p \leq .001$) both uniquely positively predicted alcohol related problems. Table 4 summarizes the results of these regression analyses.

Consistent with my hypotheses, higher Sensation Seeking predicted greater frequency of alcohol use and binge drinking frequency. However, Negative Urgency also positively predicted frequency of use. Also in line with my hypotheses, Negative Urgency uniquely positively predicted quantity x frequency of alcohol use. However, Sensation Seeking also uniquely positively predicted quantity x frequency of use and lack of Premeditation and Positive Urgency did not. Consistent with my hypotheses, Negative Urgency uniquely positively predicted binge drinking frequency and alcohol related problems, but Positive Urgency was only uniquely positively related to alcohol related problems, and lack of Perseverance was unrelated to both variables of alcohol use.

Mediation models using the HPS total score.

It was anticipated that the relationships between the HPS total score and alcohol use would be mediated by the UPPS-P scales related to the different patterns of alcohol use. Overall, the findings were consistent with a mediation model. Mediation models were conducted when HPS total or subscale scores positively predicted UPPS-P Scales and alcohol use in regression analyses controlling for the effects of age and sex where relevant. Only the UPPS-P scales that significantly predicted alcohol use were included in the mediation models. When more than one UPPS-P scale was a potential mediator, all related scales were included in a multiple mediator model using an SPSS script developed by Preacher and Hayes (2008). Where variables were negatively related in the preliminary regression analyses (i.e., Positive Urgency and Social Vitality), they were not included in mediation analyses because they did not follow the direction of the model being investigated.

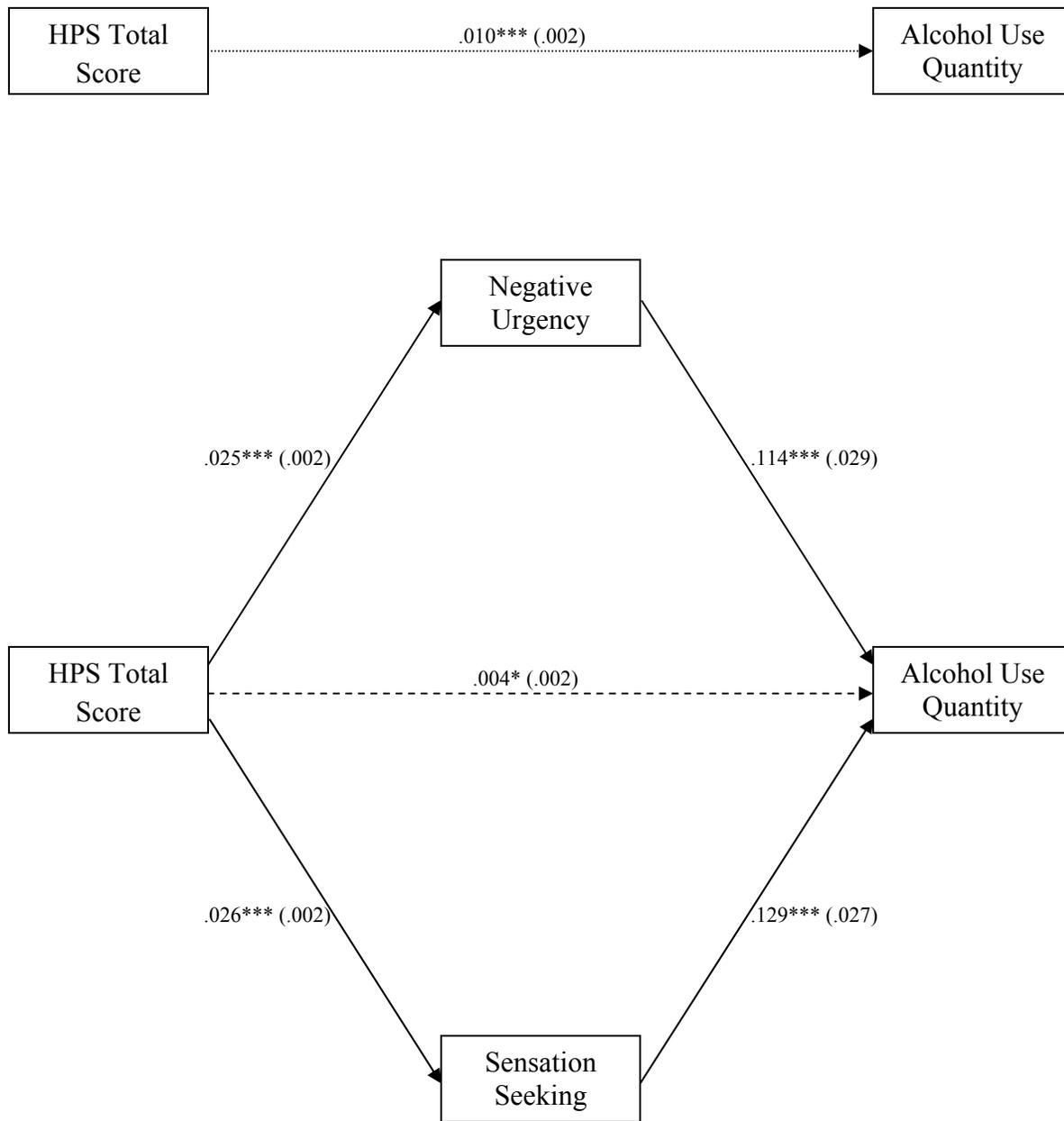
In a mediation model predicting lifetime maximum number of drinks within a 24-hour period from HPS total score with Negative Urgency, Sensation Seeking, and lack of Premeditation as possible mediators and controlling for the effects of sex, the model indicated full mediation, meaning the relationship between hypomanic personality and alcohol use was no longer significant when impulsivity was included in the model. The total indirect effect of hypomanic personality on lifetime maximum alcohol use mediated by Negative Urgency, Sensation Seeking, and lack of Premeditation was $b = .010$, 95% CI [.007, .012]. Indirect effects, including confidence intervals, were determined through bootstrapping resampling methods, using 10,000 resamples. This model, depicted in Figure 1, accounted for a significant proportion of the variance in lifetime maximum alcohol use, R^2 adjusted = .16, $F(5, 1188) = 44.89$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model, but Negative Urgency and lack of Premeditation were not.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 1. Mediation model predicting lifetime maximum number of drinks within a 24-hour period from the Hypomanic Personality Scale total score with Negative Urgency, Sensation Seeking, and Lack of Premeditation as mediators.

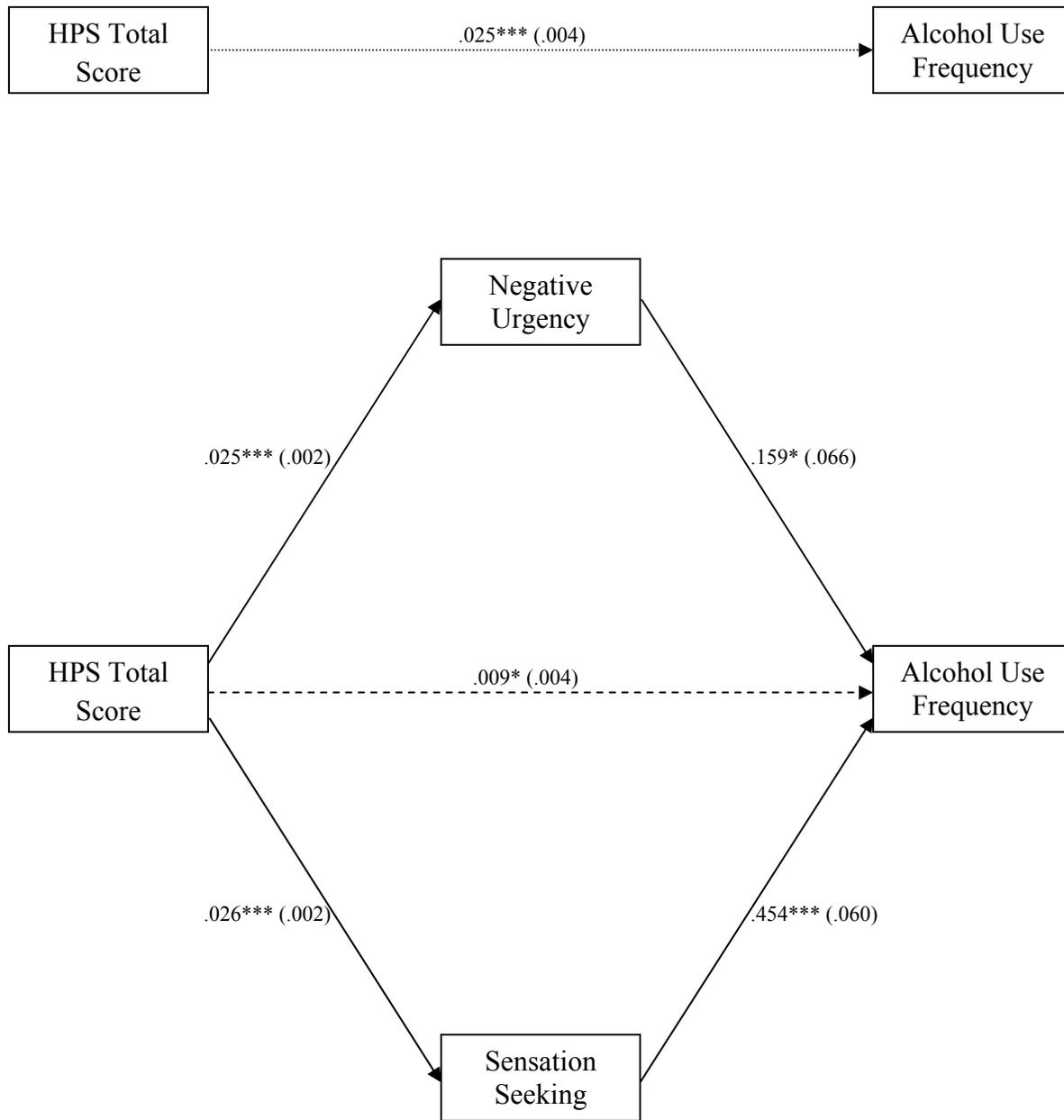
In a mediation model predicting quantity of alcohol use from HPS total score with Negative Urgency and Sensation Seeking as possible mediators and controlling for the effects of sex, the model indicated full mediation. The total indirect effect of hypomanic personality on quantity of alcohol use mediated by Negative Urgency and Sensation Seeking was $b = .006$, 95% CI [.004, .008]. This model, depicted in Figure 2, accounted for a significant proportion of the variance in quantity of alcohol use, R^2 adjusted = .09, $F(4, 1189) = 31.49$, $p \leq .001$. Negative Urgency ($p \leq .001$) and Sensation Seeking ($p \leq .001$) were both significant mediators in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 2. Mediation model predicting alcohol use quantity from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.

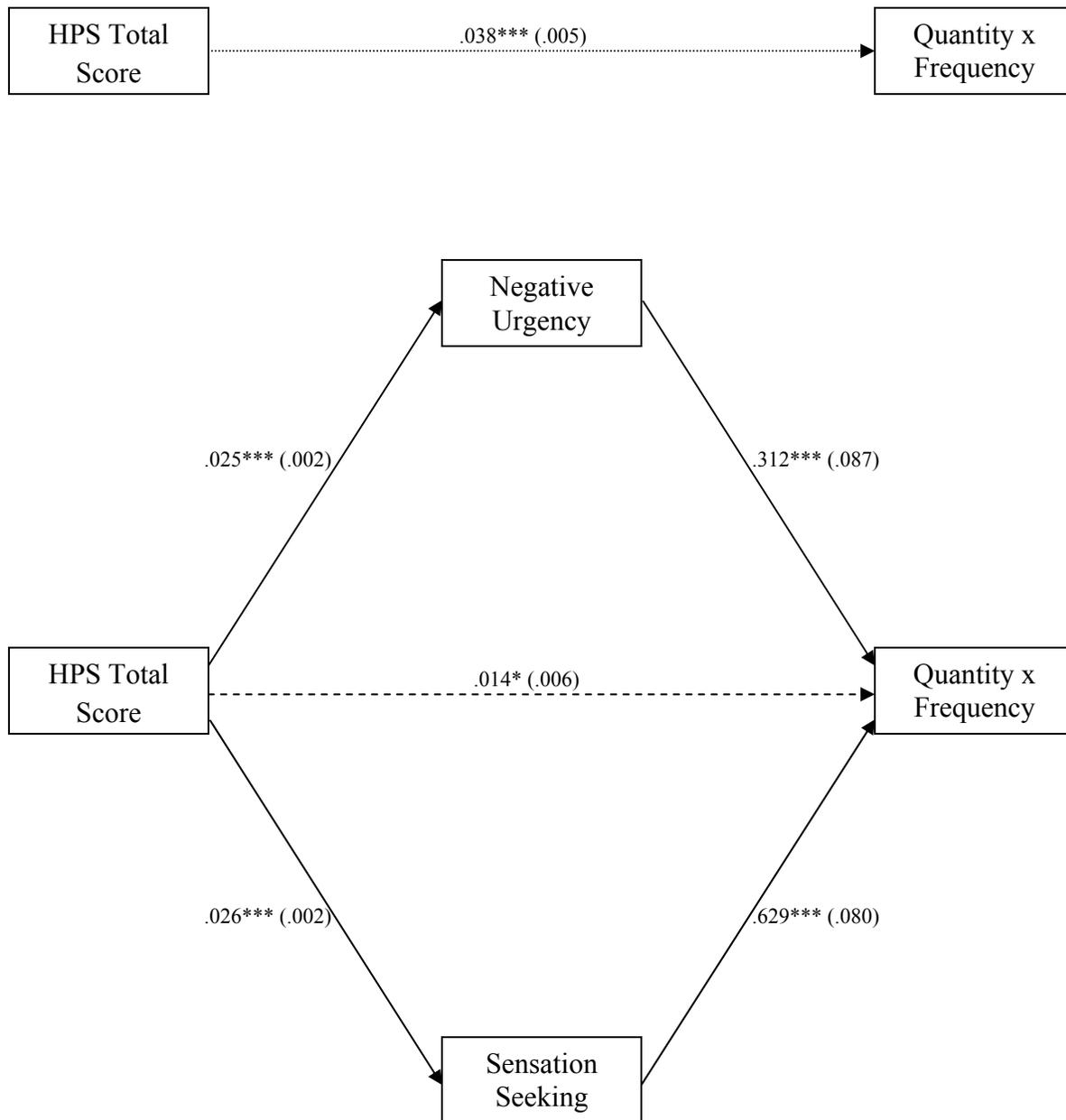
In a mediation model predicting frequency of alcohol use from HPS total score with Negative Urgency and Sensation Seeking as possible mediators and controlling for the effects of age and sex, the model indicated full mediation. The total indirect effect of hypomanic personality on frequency of alcohol use mediated by Negative Urgency and Sensation Seeking was $b = .016$, 95% CI [.011, .021]. This model, depicted in Figure 3, accounted for a significant proportion of the variance in frequency of alcohol use, R^2 adjusted = .11, $F(5, 1188) = 30.85$, $p \leq .001$. Only Sensation Seeking ($p \leq .001$) was a significant mediator in this model, Negative Urgency was not.



Note: control variables = age and sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 3. Mediation model predicting alcohol use frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.

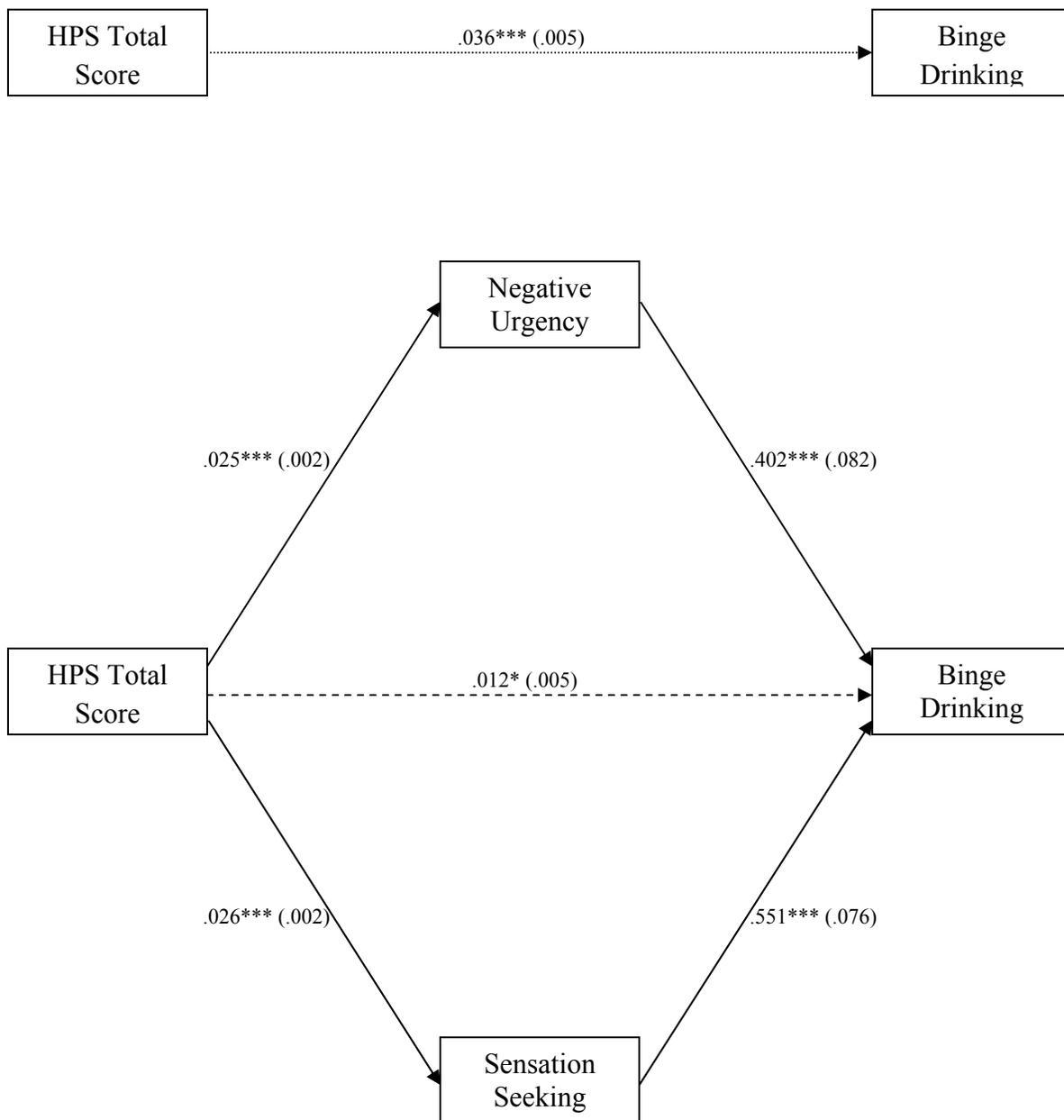
In a mediation model predicting quantity x frequency of alcohol use from HPS total score with Negative Urgency and Sensation Seeking as possible mediators and controlling for the effects of sex, the model indicated full mediation. The total indirect effect of hypomanic personality on quantity x frequency of alcohol use mediated by Negative Urgency and Sensation Seeking was $b = .024$, 95% CI [.018, .031]. This model, depicted in Figure 4, accounted for a significant proportion of the variance in quantity x frequency of alcohol use, R^2 adjusted = .14, $F(4, 1189) = 47.69, p \leq .001$. Negative Urgency ($p \leq .001$) and Sensation Seeking ($p \leq .001$) were both significant mediators in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 4. Mediation model predicting alcohol use quantity x frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.

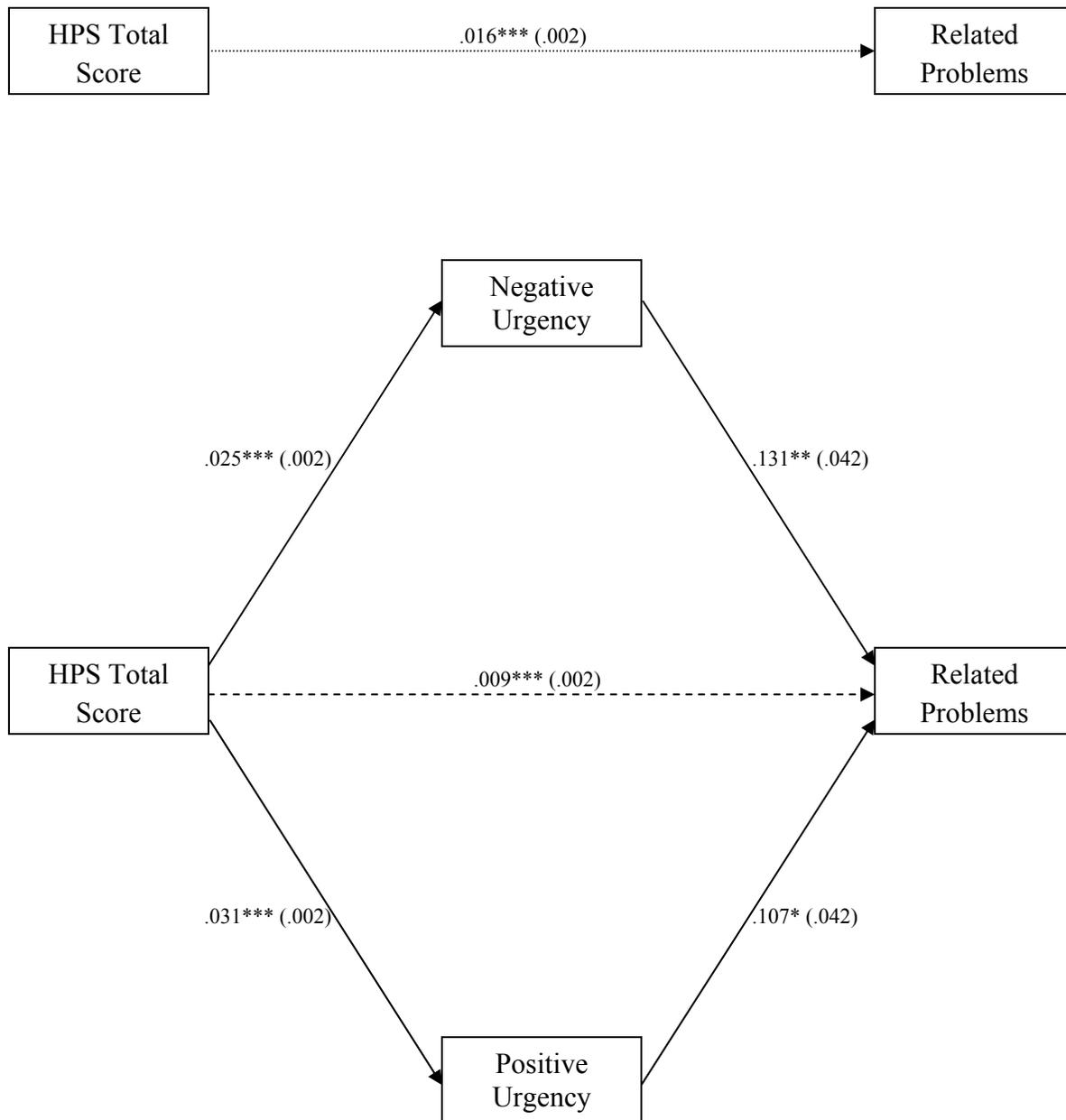
In a mediation model predicting binge drinking frequency from HPS total score with Negative Urgency and Sensation Seeking as possible mediators and controlling for the effects of sex, the model indicated full mediation. The total indirect effect of hypomanic personality on binge drinking frequency mediated by Negative Urgency and Sensation Seeking was $b = .024$, 95% CI [.019, .031]. This model, depicted in Figure 5, accounted for a significant proportion of the variance in binge drinking frequency, R^2 adjusted = .11, $F(4, 1184) = 37.82$, $p \leq .001$. Negative Urgency ($p \leq .001$) and Sensation Seeking ($p \leq .001$) were both significant mediators in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 5. Mediation model predicting binge drinking frequency from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.

In a mediation model predicting alcohol related problems from HPS total score with Negative Urgency and Positive Urgency as possible mediators and controlling for the effects of sex, the model indicated partial mediation, meaning the relationship between hypomanic personality and alcohol use was reduced, but still significant when impulsivity was included in the model. The total indirect effect of hypomanic personality on alcohol related problems mediated by Negative Urgency and Positive Urgency was $b = .007$, 95% CI [.004, .009]. This model, depicted in Figure 6, accounted for a significant proportion of the variance in alcohol related problems, R^2 adjusted = .09, $F(4, 1189) = 30.48$, $p \leq .001$. Negative Urgency ($p = .002$) was a significant mediator in this model, but Positive Urgency was not.

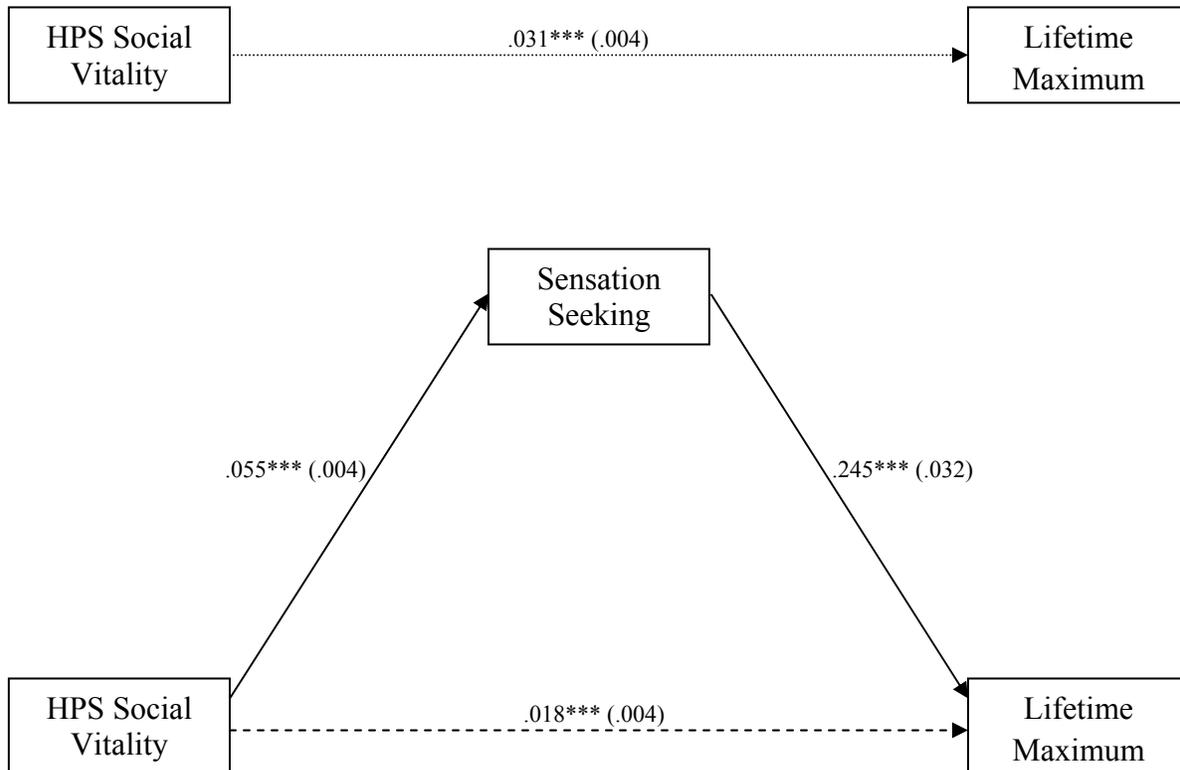


Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 6. Mediation model predicting alcohol related problems from the Hypomanic Personality Scale total score with Negative Urgency and Sensation Seeking as mediators.

Mediation models using the HPS subscales.

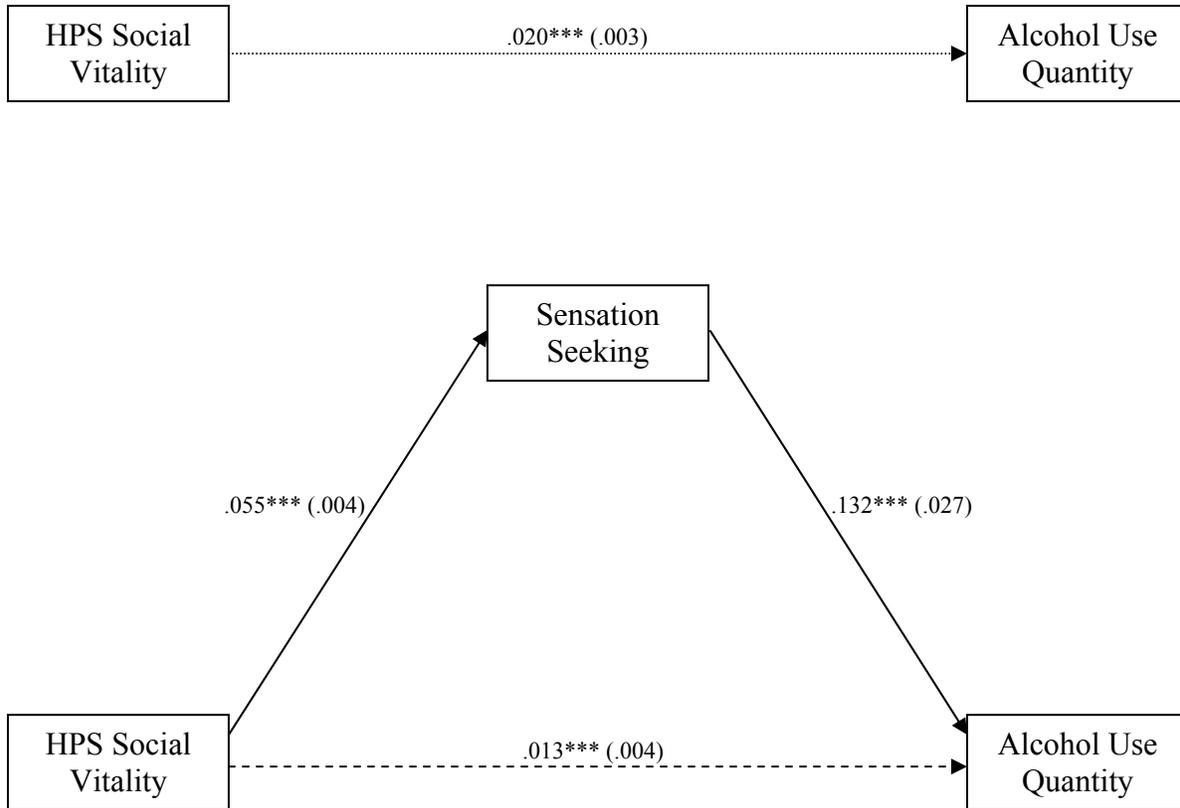
Mediation models were also examined where HPS subscales predicted alcohol use measures with UPPS-P scales as mediators. As indicated previously, Social Vitality was the HPS subscale most often found to be a unique predictor of alcohol use. Where Social Vitality is the independent variable in question, only Sensation Seeking from the UPPS-P was uniquely positively predicted by Social Vitality and therefore examined as a potential mediator. In a mediation model predicting lifetime maximum alcohol use within a 24-hour period from Social Vitality with Sensation Seeking as a possible mediator and controlling for the effect of sex, the model indicated partial mediation. The total indirect effect of Social Vitality on lifetime maximum alcohol use mediated by Sensation Seeking was $b = .013$, 95% CI [.010, .018]. This model, depicted in Figure 7, accounted for a significant proportion of the variance in lifetime maximum alcohol use, R^2 adjusted = .16, $F(3, 1190) = 74.32$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 7. Mediation model predicting lifetime maximum number of drinks within a 24-hour period from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.

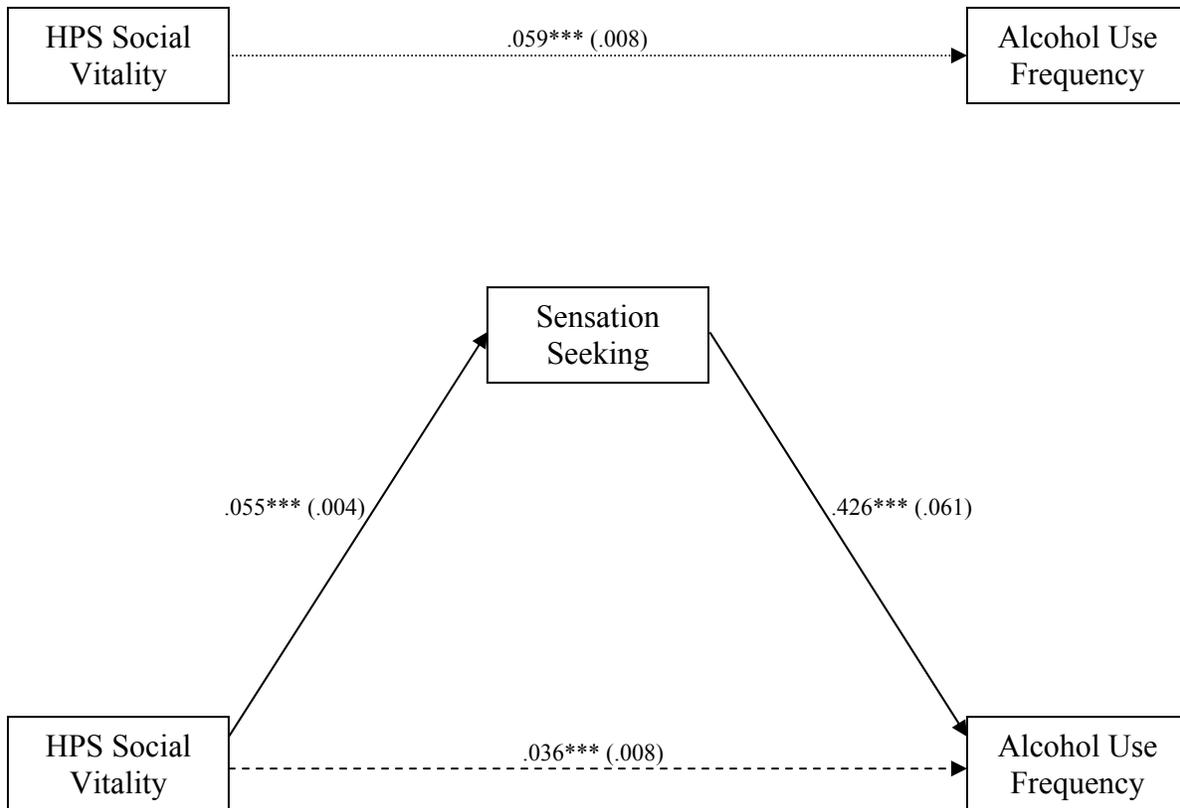
In a mediation model predicting quantity of alcohol use from Social Vitality with Sensation Seeking as a possible mediator and controlling for the effects of sex, the model indicated partial mediation. The total indirect effect of Social Vitality on quantity of alcohol use mediated by Sensation Seeking was $b = .007$, 95% CI [.004, .010]. This model, depicted in Figure 8, accounted for a significant proportion of the variance in quantity of alcohol use, $R^2_{\text{adjusted}} = .08$, $F(3, 1190) = 35.72$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 8. Mediation model predicting alcohol use quantity from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.

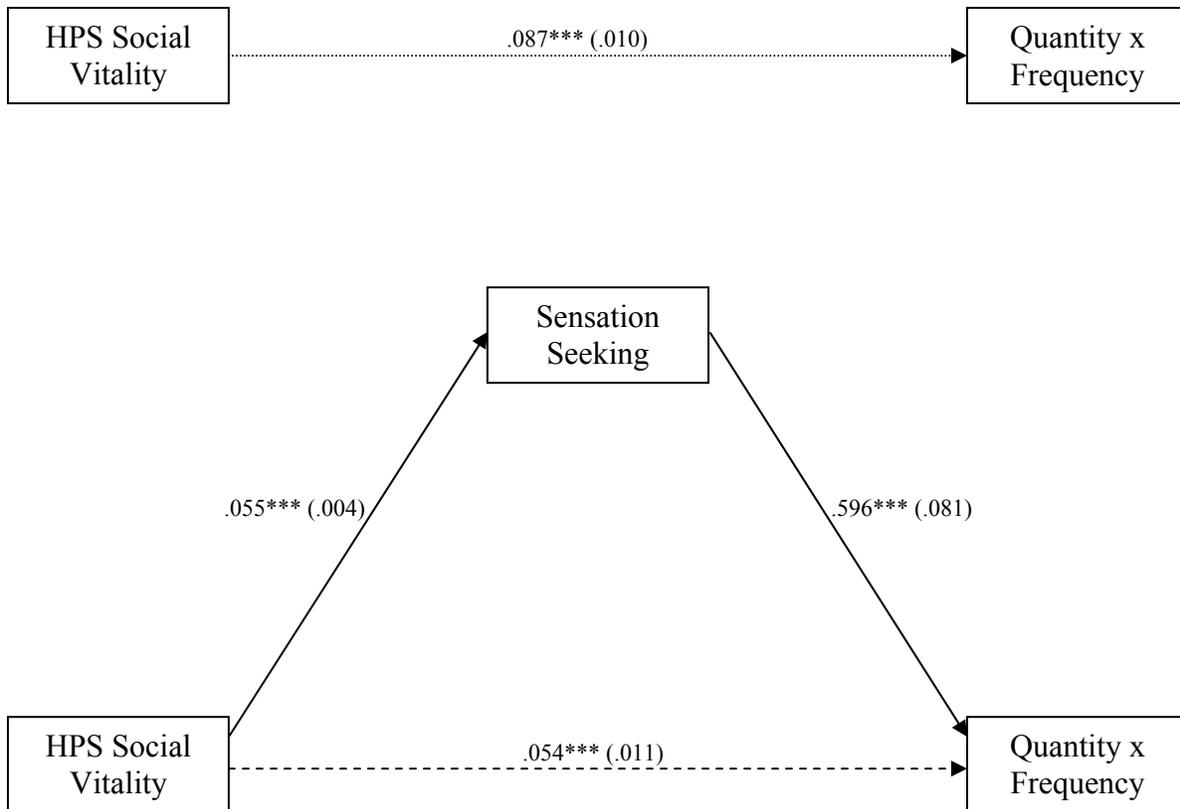
In a mediation model predicting frequency of alcohol use from Social Vitality with Sensation Seeking as a possible mediator and controlling for the effects of age and sex, the model indicated partial mediation. The total indirect effect of Social Vitality on frequency of alcohol use mediated by Sensation Seeking was $b = .023$, 95% CI [.016, .031]. This model, depicted in Figure 9, accounted for a significant proportion of the variance in frequency of alcohol use, R^2 adjusted = .12, $F(4, 1189) = 39.18$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model.



Note: control variables = age and sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 9. Mediation model predicting alcohol use frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.

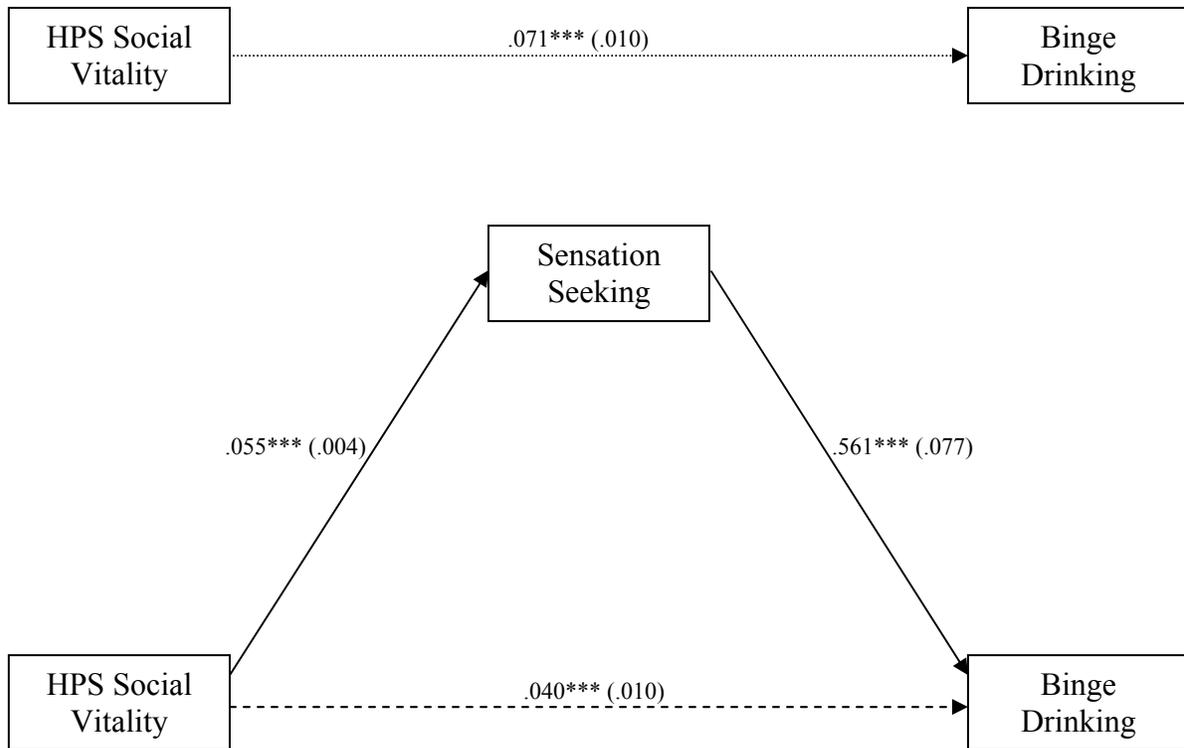
In a mediation model predicting quantity x frequency of alcohol use from Social Vitality with Sensation Seeking as a possible mediator and controlling for the effect of sex, the model indicated partial mediation. The total indirect effect of Social Vitality on quantity x frequency of alcohol use mediated by Sensation Seeking was $b = .033$, 95% CI [.023, .043]. This model, depicted in Figure 10, accounted for a significant proportion of the variance in quantity x frequency of alcohol use, R^2 adjusted = .13, $F(3, 1190) = 61.84$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 10. Mediation model predicting alcohol use quantity x frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.

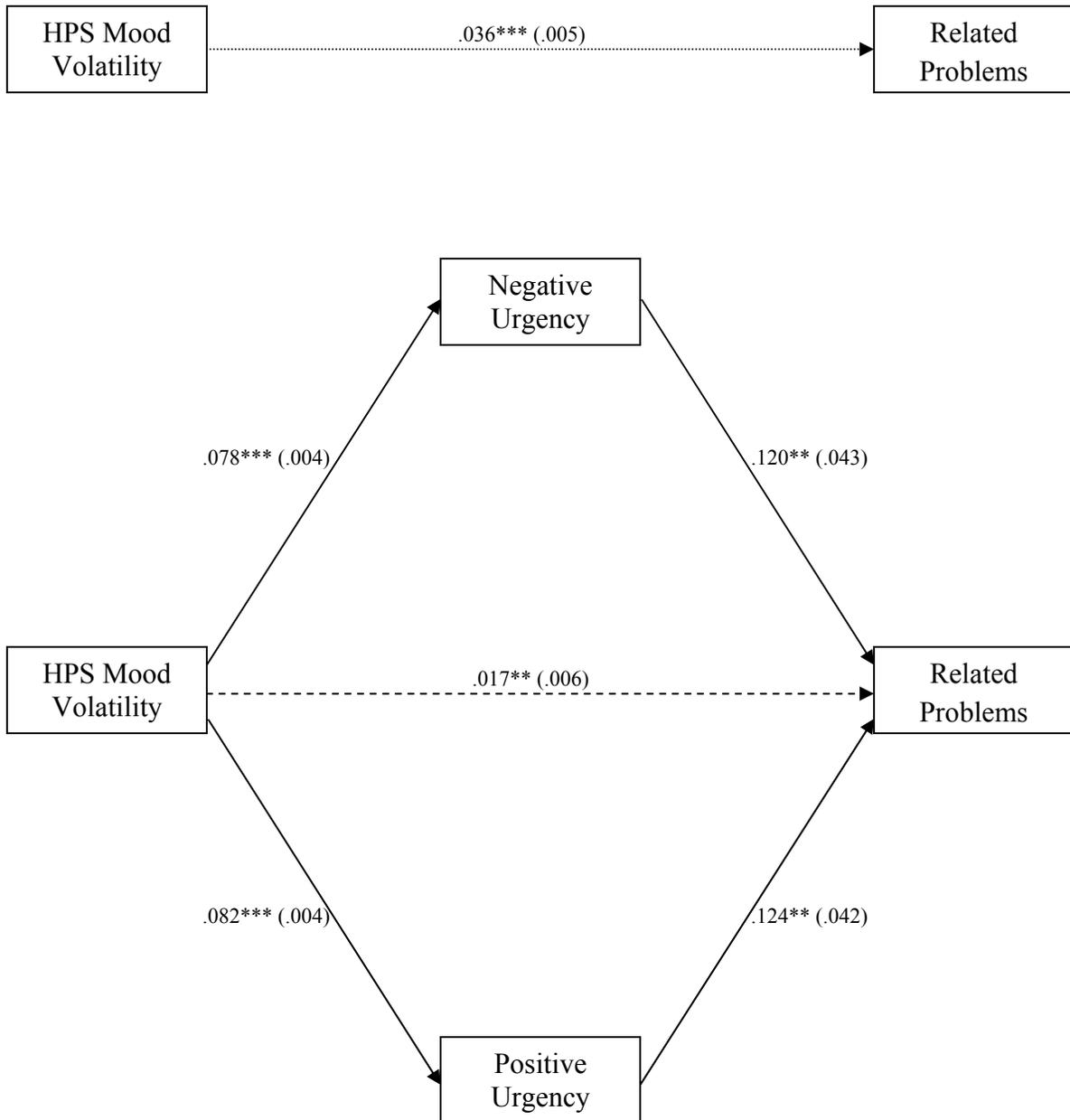
In a mediation model predicting binge drinking frequency from Social Vitality with Sensation Seeking as a possible mediator and controlling for the effects of sex, the model indicated partial mediation. The total indirect effect of Social Vitality on binge drinking frequency mediated by Sensation Seeking was $b = .031$, 95% CI [.021, .039]. This model, depicted in Figure 11, accounted for a significant proportion of the variance in binge drinking frequency, R^2 adjusted = .91, $F(3, 1185) = 40.39$, $p \leq .001$. Sensation Seeking ($p \leq .001$) was a significant mediator in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 11. Mediation model predicting binge drinking frequency from the Hypomanic Personality Scale Social Vitality subscale with Sensation Seeking as a mediator.

In a mediation model predicting alcohol related problems from Mood Volatility with Negative Urgency and Positive Urgency as possible mediators and controlling for the effects of sex, the model indicated partial mediation. The total indirect effect of Mood Volatility on alcohol related problems mediated by Negative Urgency and Positive Urgency was $b = .020$, 95% CI [.013, .026]. This model, depicted in Figure 12, accounted for a significant proportion of the variance in binge drinking frequency, R^2 adjusted = .08, $F(4, 1189) = 28.40$, $p \leq .001$. Negative Urgency ($p = .006$) and Positive Urgency ($p = .003$) were both significant mediators in this model.



Note: control variable = sex; unstandardized regression coefficients are reported with standard errors in parentheses; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

Figure 12. Mediation model predicting alcohol related problems from the Hypomanic Personality Scale Mood Volatility subscale with Negative Urgency and Positive Urgency as mediators.

Discussion

Overall, Study 1 supported the hypothesis that the relationship between hypomanic personality and alcohol use is mediated by impulsivity.

Hypomanic personality related to impulsivity.

Higher levels of hypomanic personality were related to more impulsivity. The HPS total score was positively correlated with all UPPS-P scales, except for lack of Perseverance. Another study also found no relationship between the HPS total score and lack of Perseverance. The authors suggested that this finding is in line with the idea that individuals with bipolar disorder are highly invested in achieving goals and therefore tend to persevere (Johnson et al., 2012). All three HPS subscales were positively correlated with Negative Urgency, lack of Premeditation, Sensation Seeking, and Positive Urgency. However, lack of Perseverance was only positively correlated with Mood Volatility and negatively correlated with Social Vitality. It remained unrelated to Excitement. The negative relationship between lack of Perseverance and Social Vitality was unexpected and difficult to explain. However, Schalet and colleagues (2011) found Social Vitality to be positively related to measures of positive emotionality, such as social potency and extraversion, and negatively related to measures of negative emotionality, whereas Mood Volatility was positively related to measures of negative emotionality. It may be that the Social Vitality scale is more representative of the manic phase of bipolar disorder and some people in a manic state may be more goal-focused. If this were true, one would expect a negative relationship between Social Vitality and lack of Perseverance. It should be noted that there is very limited research on the validity of the HPS subscales and as such, conclusions and interpretations regarding the subscales should not be given a higher weight than the HPS total score. The HPS total score is much more established and has ample research to support its

validity. It should, therefore, be considered the main scale of interest with the subscales being only supplemental information. Future research is needed on the HPS subscales.

In regression analyses controlling for the effects of age and sex, where relevant, HPS total score continued to positively predict all scales of the UPPS-P, except for lack of Perseverance. When all HPS subscales were included in regression analyses predicting each scale of the UPPS-P and controlling for age and sex where relevant, HPS subscale scores were differentially related to UPPS-P scales. Mood Volatility positively predicted Negative Urgency, lack of Premeditation, lack of Perseverance, and Positive Urgency. Excitement positively predicted lack of Premeditation and Positive Urgency. Social Vitality negatively predicted Negative Urgency and Positive Urgency, and positively predicted Sensation Seeking. In the study that determined the HPS subscales, Social Vitality was found to be negatively related, or unrelated, to measures of negative emotionality (Schalet et al., 2011). This is in line with the idea that individuals high on Social Vitality may be low on Negative Urgency, the tendency to act rashly when in a negative mood state. With regard to the negative relationship with Positive Urgency, this relationship fits with the idea that Social Vitality represents healthier aspects of hypomanic personality (Schalet et al., 2011), so rather than acting rashly when in a positive mood state, perhaps individuals higher on Social Vitality engage in more adaptive, socially acceptable, behaviour (e.g., sports) while in a positive mood state. It makes sense that Social Vitality would be positively related to Sensation Seeking, as both scales measure aspects of sociability and extraversion. It is important to keep in mind that the HPS is a measure of personality traits, not clinical symptoms, and therefore the Social Vitality scale may represent more adaptive, goal-directed behaviour associated with hypomania.

Hypomanic personality related to alcohol use.

Higher levels of hypomanic personality were related to more alcohol use and problems. The HPS total score was positively correlated with all measures of alcohol use (lifetime maximum number of drinks within a 24-hour period, quantity of alcohol use, frequency of alcohol use, quantity x frequency, binge drinking frequency, and self-reported alcohol related problems), as were all three HPS subscales. This is consistent with past research supporting the relationship between the HPS total score to various measures of alcohol use (Krumm-Merabet & Meyer, 2005; Kwapil et al., 2000; Meyer et al., 2007). However, in the study which derived the three HPS subscales, only Social Vitality was related to their measure of substance use, which included questions related to alcohol and drug use (Schalet et al., 2011).

In regression analyses controlling for the effects of age and sex, where relevant, HPS total score continued to positively predict each measure of alcohol use above and beyond the effects of age and sex. When all three HPS subscales were entered into regression analyses predicting each measure of alcohol use and controlling for the effects of age and sex, where relevant, Social Vitality was the only subscale to uniquely predict lifetime maximum number of drinks in 24-hours, quantity of alcohol use, frequency of alcohol use, quantity x frequency, and binge drinking frequency. Mood Volatility uniquely positively predicted alcohol related problems. In the validation of the HPS subscales, Schalet and colleagues (2011) found that Social Vitality was the subscale most strongly, positively related to a composite score of drug and alcohol use. Since individual measures of alcohol use were not examined separately, one can only speculate about the differential relationship of Mood Volatility to alcohol related problems. However, it seems reasonable that the instability and negative emotionality associated with Mood Volatility would make problematic alcohol use more likely. In the same way that

alcohol related problems have been related to Negative Urgency (Coskunpinar et al., 2013; LaBrie, Kenney, Napper, & Miller, 2014; Littlefield, Stevens, & Sher, 2014; Smith et al., 2007; Stautz & Cooper, 2013).

Impulsivity related to alcohol use.

Higher levels of impulsivity were also related to more alcohol use and problems. UPPS-P Negative Urgency, lack of Premeditation, and Positive Urgency were all positively correlated with all measures of alcohol use. While Sensation Seeking was positively correlated with all measures of alcohol use, except for alcohol related problems. Previous research has also failed to find a relationship between Sensation Seeking and alcohol related problems (Magid & Colder, 2007) possibly because any relationship between Sensation Seeking and alcohol related problems is likely indirect and due to the relationship between Sensation Seeking and overall consumption levels (Curcio & George, 2011). Lack of Perseverance was only positively correlated with binge drinking frequency and alcohol related problems. This is consistent with another study that found Lack of Perseverance was correlated with alcohol related problems, but not frequency or quantity of alcohol use (Cyders et al., 2009). Overall, the hypothesis that greater impulsivity would be related to greater alcohol use and problems was supported.

In regression analyses controlling for the variance between scales, Negative Urgency and Sensation Seeking emerged as the two scales uniquely predictive of alcohol use quantity, alcohol use frequency, quantity x frequency, and binge drinking frequency. This is consistent with past research showing that the UPPS-P scales Sensation Seeking and Negative Urgency were most likely to predict multiple measures of alcohol use (Shin et al., 2012). Both Negative and Positive Urgency uniquely predicted alcohol related problems, consistent with past research (Coskunpinar et al., 2013; Cyders et al., 2009; Cyders et al., 2007; LaBrie et al., 2014; Littlefield et al., 2014;

Smith et al., 2007; Stautz & Cooper, 2013). Negative Urgency, Sensation Seeking, and lack of Premeditation uniquely predicted lifetime maximum number of drinks within a 24-hour period. Past research has found Sensation Seeking to be related to various measures of alcohol use (Andrucci, Archer, Pancoast, & Gordon, 1989; Cloninger, Sigvardsson, & Bohman, 1988; Donohew et al., 2000; La Grange, Jones, Erb, & Reyes, 1995; LaBrie et al., 2014; Shin et al., 2012; Stautz & Cooper, 2013; Zuckerman, 1994, 2007), so it is not surprising that it emerged as a key UPPS-P scale related to alcohol use. Likewise, Negative Urgency has also been found to be related to alcohol use in past research (Coskunpinar et al., 2013; LaBrie et al., 2014; Shin et al., 2012). Lack of Perseverance was not related to any measure of alcohol use after controlling for the other scales. This is in line with another study which found no relationship between Lack of Perseverance and any measure of alcohol use (i.e., frequency of alcohol use, alcohol related problems, binge drinking, and alcohol use disorders; Shin et al., 2012). Therefore, Sensation Seeking and Negative Urgency are the UPPS-P scale most strongly related to alcohol use in this study.

Mediation.

As hypothesized, there was a positive relationship between hypomanic personality (HPS total score) and alcohol use, and that relationship was mediated by impulsivity. Higher levels of hypomanic personality were related to more impulsivity, which in turn was related to higher levels of alcohol use. This suggests that the positive relationship between hypomanic personality and alcohol use may be a product of the relationship between hypomanic personality and impulsivity and that it is likely a person's impulsivity that predicts their alcohol use. Specifically, when predicting lifetime maximum number of drinks within a 24-hour period from hypomanic personality, only Sensation Seeking was a significant mediator of the relationship.

This was also true for frequency of alcohol use. When predicting quantity of alcohol use, quantity x frequency, and binge drinking frequency, both Negative Urgency and Sensation Seeking were significant mediators. These models all indicated full mediation, meaning that the relationship between hypomanic personality and alcohol use was no longer significant when impulsivity was included in the model. When predicting alcohol related problems, only Negative Urgency was a significant mediator and this model only supported partial mediation (hypomanic personality was still a significant predictor of alcohol use with impulsivity in the model).

When examining HPS subscales as predictors of alcohol use, Social Vitality was the subscale most often related to alcohol use. It is likely this scale driving the relationships of the HPS total score to most measures of alcohol use. Social Vitality predicted lifetime maximum number of drinks within a 24-hour period, quantity of alcohol use, frequency of alcohol use, quantity x frequency, and binge drinking frequency, and all these relationships were partially mediated by Sensation Seeking. Mood Volatility was the HPS subscale which predicted alcohol related problems, and this relationship was partially mediated by Negative and Positive Urgency. As with the HPS total score, Sensation Seeking and Negative Urgency were found to be significant mediators of the hypomanic personality – alcohol use relationship, with the addition of Positive Urgency. Therefore, while adaptive socializing traits are associated with alcohol use, it is through a desire for novelty or excitement. In contrast, endorsing volatile mood states is related to alcohol related problems, and seems to occur in the context of mood-related drinking behaviours.

Chapter 3: Study 2

Introduction and Hypotheses

The second study examined the relationships between questionnaire responses from the first study to a behavioural measure of delay discounting and auditory and visual oddball EEG tasks. Individuals at all levels of hypomanic personality, which has been shown to predict both bipolar and alcohol use disorders longitudinally (Kwapil et al., 2000), were invited to participate in this second study. Most P300 studies have focused on individuals who have already been diagnosed with bipolar or alcohol related disorders or who are at high risk of developing a disorder based on family history. This study used a self-report measure of hypomanic personality which could easily be employed on university campuses to screen for individuals at risk for both disorders.

Most studies use either auditory or visual oddball ERP tasks; few have used both paradigms in the same sample. This study extends the current knowledge by examining P300 amplitude using both auditory and visual oddball tasks in undergraduates who have previously completed measures of hypomanic and impulsive personality and alcohol use. The role of self-report impulsivity, which has previously been linked to both disorders, was examined as a mediator between hypomanic personality or alcohol use and delay discounting performance, or P300 amplitude. Determining methods of identifying individuals at subclinical levels of risk is an important step in early interventions designed to prevent or reduce the impact of these debilitating disorders.

For the second study, it was hypothesized that:

1. UPPS-P scores from study 1 would predict delay discounting performance, with higher impulsivity related to steeper discounting rates. Specifically, lack of Premeditation was

expected to be most strongly related to discounting with Negative Urgency and Sensation Seeking less strongly related.

2. HPS scores from study 1 would predict delay discounting performance, with higher levels of hypomanic personality related to steeper discounting rates.
 - a. UPPS-P scores were expected to mediate the relationship between hypomanic personality and delay discounting.
3. Alcohol use from study 1 would predict delay discounting performance, with higher rates of alcohol use and problems related to steeper discounting rates.
 - a. UPPS-P scores were expected to mediate the relationship between alcohol use and discounting rates.
4. UPPS-P scales would be related to smaller P300 amplitudes in both auditory and visual oddball EEG tasks, with higher levels of impulsivity related to smaller amplitudes. Specifically, lack of Premeditation was expected to be most strongly negatively related to P300 amplitude with Negative Urgency and lack of Perseverance less strongly related.
5. HPS scores would be related to smaller P300 amplitudes in both auditory and visual oddball EEG tasks, with higher levels of hypomanic personality related to smaller amplitudes.
 - a. UPPS-P scores were expected to mediate the relationship between hypomanic personality and P300 amplitude reductions.
6. Alcohol use would be related to smaller P300 amplitudes in both auditory and visual oddball EEG tasks, with higher levels of alcohol use and problems related to smaller amplitudes.

- a. UPPS-P scores were expected to mediate the relationship between alcohol use and P300 amplitude reductions.

Method

Participants.

One hundred and twenty-three participants were recruited for Study 2 from the sample of people who completed the online survey described in Study 1. Individuals who consented to be contacted for a follow-up study were invited to participate. Participants received two course credits through the Human Subject Pool system, or \$20 if they did not require course credit at that time. Participants provided informed consent and all procedures were approved by the appropriate institutional ethics review board.

Participants (62% female) ranged in age from 19 to 33 ($M = 20.75$; $SD = 2.27$). Sixty-seven percent described themselves as European or of European descent, while another 29% described themselves as East Asian or of East Asian descent. Other ethnicities represented included: Indian-South Asian, Latin American-Hispanic, Middle Eastern, or a combination of more than one. Participants were excluded from participating in this second study if they reported impaired hearing, a history of a head injury resulting in loss of consciousness greater than five minutes, a learning disability, a neurological disorder (e.g., seizures or stroke), taking medication for a psychiatric disorder, previous history of drug abuse or treatment, or if their corrected vision was less than 20/50. Participants were asked not to consume any alcohol or non-prescription drugs for the 24-hour period prior to the study. Participants were excluded from all analyses if they reported speaking English for less than five years or if they failed to endorse two or more items on the HPS validity scale. Participants were excluded from individual analyses if one or more measures being examined was deemed to be unusable. For

example, if they had less than 20 segments for EEG measures, had abnormal EEG data, had consumed alcohol or drugs in the 24 hours prior to the study, or had nonsystematic delay discounting data as outlined by Johnson & Bickel (2008). Johnson & Bickel (2008) set out two criteria by which data should be identified as nonsystematic and possibly invalid. First, “if any indifference point (starting with the second delay) was greater than the preceding indifference point by a magnitude greater than 20% of the larger later reward” (p. 268). Second, “if the last indifference point was not less than the first indifference point by at least a magnitude equal to 10% of the larger later reward” (p. 268). If either criterion were met, the authors suggested that the validity of the results should be questioned.

Procedure.

Potential participants were contacted via email and invited to participate in this follow-up study. Participants who agreed to participate came to the Clinical and Cognitive Neuroscience lab in the Kenny building at UBC. After giving informed consent, participants completed the delay discounting task, questionnaires, and EEG measures.

Measures.

Questionnaires.

Participants completed the same demographic and alcohol use questionnaires as in Study 1. They also completed questions to identify factors that could affect EEG recording, such as mood related questions from the Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995), how much caffeine they had consumed that day, how much sleep they had the previous night, how much nicotine, alcohol, or other substances they had consumed recently, the amount and type of food last consumed, and how typical all these amounts were for them.

Delay discounting.

The delay discounting task (Rachlin et al., 1991) is a measure of the ability to delay rewards. In this computerized task, provided to us by Dr. Leonard Green (e.g., Ahn et al., 2011; Myerson, Green, Hanson, Holt, & Estle, 2003), participants made a series of choices between smaller immediate rewards (e.g., \$625 now) and larger delayed rewards (\$2500 in a year). After a few practice trials, participants made six hypothetical choices for each of six temporal delays (2 weeks, 1 month, 6 months, 1 year, 3 years, and 10 years). The order of the delay times were randomized for each participant. For all temporal delays, the delay amount was kept at \$2500 and the first choice was half of the delay amount (i.e., \$1250). If the participant chose the immediate reward, the next choice was half of that amount (e.g., \$625). If the participant chose the delayed reward, the next choice increased by half the difference between the last choice and the delayed amount (e.g., \$1875). This pattern continued for six trials, after which the indifference point was determined. The indifference point is the amount that would be presented in the seventh trial, if there was one. It is the last presented immediate amount and half of the previous adjustment up or down depending on whether the delay or immediate amount was chosen in the last (6th) trial. The indifference point represents an estimate of the immediate amount at which the participant is indifferent between that immediate amount and the delayed amount; it is the subjective immediate value that they place on the delayed reward (Myerson et al., 2003).

Using nonlinear regression, each participant's discounting rate (k) was calculated by fitting the hyperbolic model to the observed data using the equation: $V = A/(1+kD)$, where V is the indifference point, A is the delay amount (\$2500), and D is the delay (in weeks). The discounting rate is a measure of the weight an individual places on the future reward. Higher

discounting rates imply that the individual places less weight on the future reward and is more likely to choose a smaller immediate reward (Mazur, 1987).

Studies have found that there is no difference in the discounting rate when using real compared to hypothetical rewards, suggesting that hypothetical rewards are a valid option for studying delay discounting (Johnson & Bickel, 2002; Madden, Begotka, Raiff, & Kastern, 2003; Madden et al., 2004). Test-retest reliability of discounting rates (k) was found to be .91 over a one-week period (Simpson & Vuchinich, 2000), .77 over a five week period and .71 over a one year period (Kirby, 2009).

Event-related potential measures.

Electroencephalogram (EEG) procedure.

EEG data were collected with a Brain Products Inc, QuickAmp 72 System. Brain Vision Recorder was used to record the data and Brain Vision Analyzer was used to process and score ERPs. Brain activity was recorded from Ag/AgCl electrodes at the F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 scalp sites and referenced offline to averaged mastoids. All impedances were kept below 10 k Ω . Electrooculographic (EOG) data were also recorded from Ag/AgCl electrodes above and below the left eye, to detect vertical eye movements (e.g., blinks), and on both temples to detect horizontal eye movements. These were used to correct for blinks and ocular artifacts (Gratton, Coles, and Donchin, 1983). Both EEGs and EOGs were continuously sampled at 500 Hz. Signals were digitally filtered offline with a low pass frequency filter at 25 Hz (24dB/octave roll-off). Stimulus presentation and behavioural response acquisition were controlled by E-Prime Version 2.0 software.

The auditory P300 oddball task.

Subjects sat in a comfortable chair in a sound-attenuated room separate from the recording equipment, and were presented with an auditory oddball task (e.g., Polich & Kok, 1995; Shin et al., 2010) via disposable in-ear inserts. Participants were presented with 75 target auditory stimuli in a two-tone auditory oddball paradigm which required a button press to targets. Target stimuli were tones of 1500 Hz, while non-targets were 1000 Hz. Stimuli were 86 dB SPL tone pips presented binaurally for 50ms with a random interstimulus interval of between 1000-3000 ms (approximately 2000ms on average). Subjects were instructed to remain as still as possible and to fixate on a small cross on a computer monitor. In total, subjects received 75 target tones randomly interspersed among 425 non-target tones. Only target responses were used in analysis.

The data were baseline corrected prior to averaging. A computer algorithm was used to identify the largest positive (P300) voltage in a window from 250-800 ms following stimulus onset, which was then manually verified, and corrected when needed.

The visual P300 oddball task.

The Rotated Heads task (Begleiter et al., 1984) was used to assess the P300 in a visual modality. Participants viewed circles on a computer monitor representing either the superior view of a human head, with a nose and one ear, or a plain circle (see Figure 13). The plain (nontarget) circles occurred in 160 trials and did not require a behavioural response. Participants were asked to press the left button on a Psychology Software Tools, Inc. Deluxe Serial Response Box if a left ear was displayed and press the right button if a right ear was displayed. In the “normal” (easy) condition, the nose of the head is pointing up and the ear is on the same side as the button to be pressed (e.g., the right ear is on the right side of the screen). In the “rotated”

(hard) condition, the nose is pointing down and the ear is displayed on the opposite side as the button to be pressed (e.g., the right ear is on the left side of the screen). There were 80 target trials with an equal number of left and right ears in the normal and rotated conditions (i.e., 20 of each combination). Stimuli were presented in the same randomized order for all participants. Stimulus duration was 96 ms, and trial epoch included a 500 ms baseline with a 1500 ms period after stimulus onset. The intertrial interval varied randomly between 1000 and 2000 ms. Participants were given practice trials to ensure that they understand the task. They were instructed to respond as quickly and accurately as possible, to remain as still as possible, and to fixate on a small cross on the computer monitor. Only correct trials were used in analysis.

The data were baseline corrected prior to averaging. In addition to being examined separately, normal and rotated conditions were averaged together for a total peak condition, as amplitude for these conditions has been shown to be highly correlated (e.g., Iacono et al., 2002). A computer algorithm was used to identify the largest positive (P300) voltage in a window from 300-800 ms following stimulus onset, which was then manually verified, and corrected when needed, by a rater. See figure 14 for a typical P300 waveform. Test-retest reliability for P300 amplitude in this task has been found to be .71-.79 over three years (Carlson & Iacono, 2006).

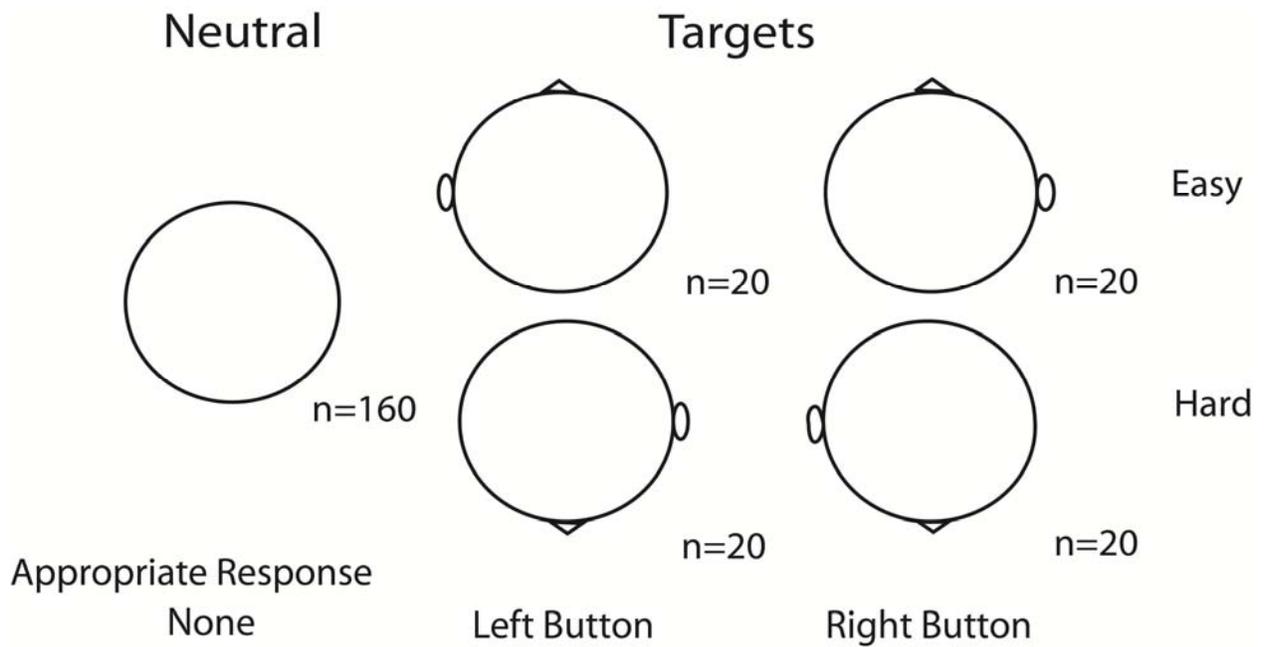


Figure 13. Rotated Heads task stimuli.

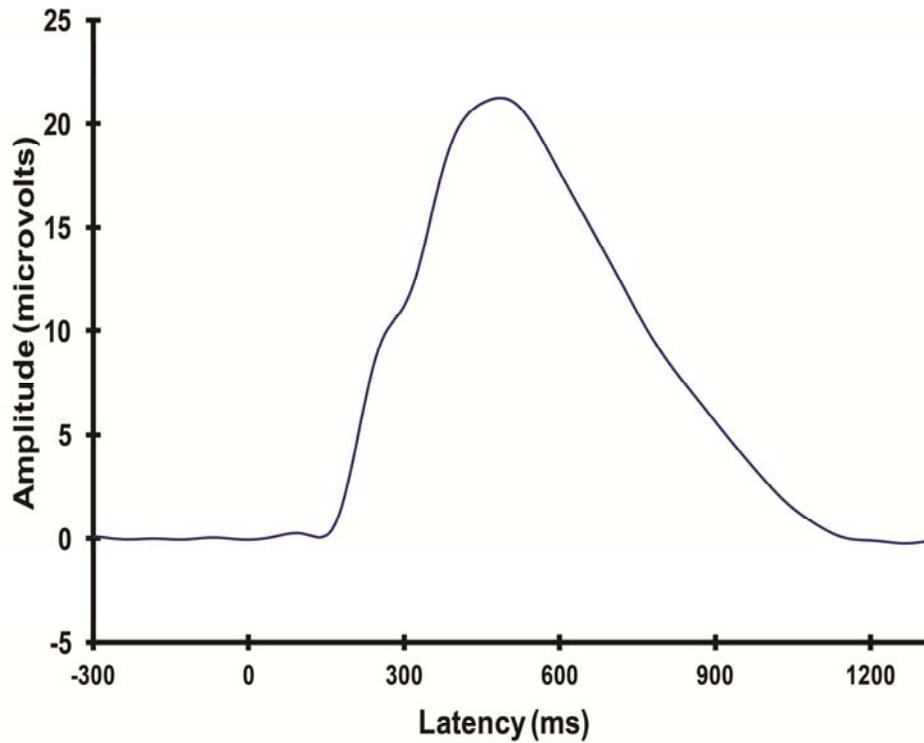


Figure 14. Typical P300 waveform obtained from the Rotated Heads task.

Statistical analyses.

Data were analyzed using multiple regression techniques as described in Study 1. Key variables used in analyses included age, sex (0=female, 1=male), the HPS total score and subscale scores, the five UPPS-P scales, and alcohol use measures (lifetime maximum number of drinks within a 24-hour period, quantity, frequency, quantity x frequency, binge drinking frequency, and alcohol related problems), from Study 1, as well as variables from Study 2 including delay discounting rate, auditory and visual P300 amplitude and latency, and time between Study 1 and Study 2. Delay discounting rates were positively skewed and a natural log transformation was used to produce a reasonably normal distribution. Diagnostics and assumptions were examined as described in Study 1 and found to be reasonable. Outliers were examined for leverage and influence and analyses were run with and without outliers to determine if they had a strong effect of results. No outliers needed to be excluded from final analyses.

Results

While Study 2 also examines a large number of variables, alpha remained at $p = .05$ in order to examine the maximum possible number of associations. Setting alpha higher would have resulted in very few interpretable findings. In light of this, a limitation of this study is the increased likelihood of a Type I error and future studies following up on these results should set a more stringent alpha. Tables 6, 8, and 9 present the means and standard deviations for all variables in Study 2. Table 7 compares the means from Study 1 and Study 2 and found that the samples differed only on alcohol use frequency. Table 10 presents the first order correlations for Study 2 variables. In this subsample of participants from the first study, HPS total score was significantly positively correlated with all UPPS-P subscales ($p \leq .001$), except for lack of

Perseverance. Social Vitality ($p \leq .004$), Mood Volatility ($p \leq .001$), and Excitement ($p \leq .001$) were also significantly positively correlated with all UPPS-P subscales, except for lack of Perseverance. As in the first study, HPS total score was significantly positively correlated with all measures of alcohol use (lifetime maximum number of drinks within a 24-hour period, frequency of alcohol use, quantity of alcohol use, quantity x frequency, binge drinking frequency, and alcohol related problems; $p \leq .001$). Social Vitality ($p \leq .002$), Mood Volatility ($p \leq .002$), and Excitement ($p \leq .001$) were also significantly positively correlated with all measures of alcohol use. UPPS-P Negative Urgency and Positive Urgency were both positively correlated with all measures of alcohol use ($p \leq .010$). Lack of Premeditation and Sensation Seeking were both positively correlated with all measures of alcohol use ($p \leq .010$), except for alcohol related problems. Lack of Perseverance was unrelated to alcohol use. Time, measured as number of days, between Study 1 and Study 2 was unrelated to any variable and therefore not included in further analyses.

Table 6*Means and Standard Deviations for all Study 1 Variables for the Study 2 Sample*

	<i>N</i>	Untransformed		Log Transformed	
		Mean	SD	Mean	SD
Age	123	20.73	2.29		
Alcohol Maximum	123	13.76	8.91	2.49	0.68
Alcohol Quantity	123	4.69	3.72	1.58	0.55
Alcohol Frequency	123	70.42	67.40	3.74	1.16
Quantity x Frequency	123	357.51	401.93	5.01	1.61
Binge Frequency	123	24.66	37.59	2.17	1.57
Alcohol Problems	123	1.40	1.78	0.66	0.63
HPS Total	123	16.81	10.70		
HPS Social Vitality	123	7.95	5.01		
HPS Mood Volatility	123	5.56	4.23		
HPS Excitement	123	2.37	2.61		
UPPS-P Negative Urgency	123	2.31	0.58		
UPPS-P Premeditation	123	2.00	0.47		
UPPS-P Perseverance	123	2.07	0.50		
UPPS-P Sensation Seeking	123	2.84	0.67		
UPPS-P Positive Urgency	123	1.84	0.61		

Note: Alcohol Maximum = the lifetime maximum number of drinks within a 24-hour period, Alcohol Quantity = the average quantity of alcohol consumed per occasion in the past 12 months, Alcohol Frequency = the average frequency of alcohol consumption in the past 12 months, Quantity x Frequency = the quantity x frequency of drinks consumed in the past 12 months, Binge Frequency = the frequency of binge drinking in the last 12 months, Alcohol Problems = the Short Michigan Alcoholism Screening Test, HPS Total = Hypomanic Personality Scale Total Score, HPS Social Vitality = Hypomanic Personality Scale Social Vitality, HPS Mood Volatility = Hypomanic Personality Scale Mood Volatility, HPS Excitement = Hypomanic Personality Scale Excitement, UPPS-P Negative Urgency = UPPS-P Impulsive Behaviour Scale Negative Urgency, UPPS-P Premeditation = UPPS-P Impulsive Behaviour Scale Lack of Premeditation, UPPS-P Perseveration = UPPS-P Impulsive Behaviour Scale Lack of Perseveration, UPPS-P Sensation Seeking = UPPS-P Impulsive Behaviour Scale Sensation Seeking, UPPS-P Positive Urgency = UPPS-P Impulsive Behaviour Scale Positive Urgency.

Table 7*Comparing Means for Study 1 and Study 2 Samples*

<i>Variables</i>	<i>Study 1</i> (<i>N = 1194</i>)	<i>Study 2</i> (<i>N = 123</i>)	<i>t</i>	<i>p</i>
Age	21.11	20.73	1.27	.204
†Percent Male	33.20	38.20	1.23	.268
†Percent European Descent	54.00	61.00	2.23	.135
Alcohol Maximum	2.42	2.49	1.15	.251
Alcohol Quantity	1.55	1.58	0.61	.545
Alcohol Frequency	3.52	3.74	1.99	.047
Quantity x Frequency	4.75	5.01	1.73	.083
Binge Frequency	2.01	2.17	1.15	.251
Alcohol Problems	0.56	0.66	1.73	.085
HPS Total	15.33	16.81	1.73	.083
HPS Social Vitality	7.30	7.95	1.55	.121
HPS Mood Volatility	5.14	5.56	1.20	.228
HPS Excitement	1.98	2.37	1.84	.065
UPPS-P Negative Urgency	2.25	2.31	1.17	.244
UPPS-P Premeditation	1.97	2.00	0.73	.465
UPPS-P Perseverance	2.03	2.07	0.89	.372
UPPS-P Sensation Seeking	2.73	2.84	1.94	.052
UPPS-P Positive Urgency	1.79	1.84	0.93	.350

†Gender and ethnicity were compared using a Chi-square test of the proportion of males/European descent. Note: Alcohol Maximum = the natural log transformation of the lifetime maximum number of drinks within a 24-hour period, Alcohol Quantity = the natural log transformation of the average quantity of alcohol consumed per occasion in the past 12 months, Alcohol Frequency = the natural log transformation of the average frequency of alcohol consumption in the past 12 months, Quantity x Frequency = the natural log transformation of the quantity x frequency of drinks consumed in the past 12 months, Binge Frequency = the natural log transformation of the frequency of binge drinking in the last 12 months, Alcohol Problems = the natural log transformation of the Short Michigan Alcoholism Screening Test, HPS Total = Hypomanic Personality Scale Total Score, HPS Social Vitality = Hypomanic Personality Scale Social Vitality, HPS Mood Volatility = Hypomanic Personality Scale Mood Volatility, HPS Excitement = Hypomanic Personality Scale Excitement, UPPS-P Negative Urgency = UPPS-P Impulsive Behaviour Scale Negative Urgency, UPPS-P Premeditation = UPPS-P Impulsive Behaviour Scale Lack of Premeditation, UPPS-P Perseverance = UPPS-P Impulsive Behaviour Scale Lack of Perseverance, UPPS-P Sensation Seeking = UPPS-P Impulsive Behaviour Scale Sensation Seeking, UPPS-P Positive Urgency = UPPS-P Impulsive Behaviour Scale Positive Urgency.

Table 8

Means and Standard Deviations for Study 2 Delay Discounting and the Auditory Oddball EEG Task Variables

	<i>N</i>	Mean	SD
Delay Discounting	102	.03	.04
Cz_AUD_Amplitude	91	8.65	4.51
C3_AUD_Amplitude	91	7.84	3.93
C4_AUD_Amplitude	91	6.50	3.81
Pz_AUD_Amplitude	91	10.98	4.43
P3_AUD_Amplitude	91	9.38	3.89
P4_AUD_Amplitude	91	8.87	4.00
Cz_AUD_Latency	91	381.38	57.06
C3_AUD_Latency	91	383.27	55.19
C4_AUD_Latency	91	386.86	58.30
Pz_AUD_Latency	91	379.19	50.73
P3_AUD_Latency	91	378.59	49.58
P4_AUD_Latency	91	380.00	52.41

Note: delay discounting = the natural log transformation of discounting rates, Cz_AUD_Amplitude = amplitude for the Cz scalp site of the auditory oddball EEG task, C3_AUD_Amplitude = amplitude for the C3 scalp site of the auditory oddball EEG task, C4_AUD_Amplitude = amplitude for the C4 scalp site of the auditory oddball EEG task, Pz_AUD_Amplitude = amplitude for the Pz scalp site of the auditory oddball EEG task, P3_AUD_Amplitude = amplitude for the P3 scalp site of the auditory oddball EEG task, P4_AUD_Amplitude = amplitude for the P4 scalp site of the auditory oddball EEG task, Cz_AUD_Latency = latency for the Cz scalp site of the auditory oddball EEG task, C3_AUD_Latency = latency for the C3 scalp site of the auditory oddball EEG task, C4_AUD_Latency = latency for the C4 scalp site of the auditory oddball EEG task, Pz_AUD_Latency = latency for the Pz scalp site of the auditory oddball EEG task, P3_AUD_Latency = latency for the P3 scalp site of the auditory oddball EEG task, P4_AUD_Latency = latency for the P4 scalp site of the auditory oddball EEG task.

Table 9*Means and Standard Deviations for Study 2 Visual Oddball EEG Task Variables*

Amplitude	<i>N</i>	Mean	SD	Latency	<i>N</i>	Mean	SD
Fz_Total	103	9.48	6.56	Fz_Total	103	423.75	79.78
Fz_Easy	103	9.51	6.62	Fz_Easy	103	425.81	77.69
Fz_Hard	103	9.78	6.74	Fz_Hard	103	419.22	66.08
F3_Total	103	9.13	5.78	F3_Total	103	424.78	79.26
F3_Easy	103	9.27	5.61	F3_Easy	103	424.91	76.30
F3_Hard	103	9.24	6.20	F3_Hard	103	419.40	65.59
F4_Total	103	9.71	5.98	F4_Total	103	423.61	82.12
F4_Easy	103	9.75	6.28	F4_Easy	103	424.17	76.84
F4_Hard	103	9.88	6.13	F4_Hard	103	418.58	65.29
Cz_Total	104	15.33	7.19	Cz_Total	104	422.48	56.90
Cz_Easy	104	15.42	7.21	Cz_Easy	104	427.58	63.10
Cz_Hard	104	15.65	7.48	Cz_Hard	104	419.52	53.20
C3_Total	103	11.61	5.86	C3_Total	103	422.80	56.60
C3_Easy	103	11.95	5.96	C3_Easy	103	427.18	62.29
C3_Hard	103	11.64	5.96	C3_Hard	103	420.85	52.61
C4_Total	104	12.36	5.90	C4_Total	104	420.62	55.65
C4_Easy	104	12.58	6.08	C4_Easy	104	426.33	62.54
C4_Hard	104	12.40	6.07	C4_Hard	104	417.75	51.85
Pz_Total	104	16.71	7.37	Pz_Total	104	408.69	51.00
Pz_Easy	104	17.07	7.45	Pz_Easy	104	414.96	58.69
Pz_Hard	104	16.72	7.55	Pz_Hard	104	404.81	52.69
P3_Total	104	11.79	6.85	P3_Total	104	408.56	51.53
P3_Easy	104	12.44	7.18	P3_Easy	104	412.37	56.63
P3_Hard	104	11.50	6.67	P3_Hard	104	404.89	54.60
P4_Total	104	11.95	6.41	P4_Total	104	406.29	51.18
P4_Easy	104	12.52	6.52	P4_Easy	104	411.19	56.53
P4_Hard	102	11.78	6.60	P4_Hard	104	405.00	53.68

Table 10*First Order Correlations for Study 2*

	Age	Sex	Alc Max	Alc Quant	Alc Freq	Quant x Freq	Binge	SMAST	HPS Total	HPS Soc Vit	HPS Mood Vol	HPS Excitement	UPPS-P Neg Urg	UPPS-P Premed	UPPS-P Persev	UPPS-P SS	UPPS-P Pos Urg
Age		.06	.08	-.15	.11	.02	-.02	.21*	-.09	.02	-.19*	-.11	-.05	.01	.03	-.13	-.19*
Sex			.27**	.05	.16	.13	-.04	.17	.04	.08	.05	-.07	-.04	-.08	.15	.24**	.07
Alc Max				.63***	.69***	.79***	.68***	.28***	.39***	.36***	.37***	.29***	.31**	.28**	.11	.36***	.27**
Alc Quant					.39***	.72***	.68***	.31***	.37***	.27**	.35***	.40***	.31***	.16	.07	.18*	.34***
Alc Freq						.92***	.62***	.19*	.31***	.28***	.28**	.23*	.25**	.35***	.17	.29***	.28**
Quant x Freq							.76***	.27***	.38***	.32***	.35***	.34***	.33***	.33***	.15	.29***	.35***
Binge								.29**	.41***	.32***	.40***	.38***	.38***	.37***	.16	.26**	.36***
SMAST									.34***	.32***	.29***	.27**	.27**	.03	.12	.06	.24**
HPS Total										.87***	.88***	.85***	.43***	.37***	.02	.49***	.58***
HPS Soc Vit											.58***	.62***	.26**	.27**	-.04	.46***	.37***
HPS Mood Vol												.73***	.53***	.36***	.12	.44***	.64***
HPS Excitement													.36***	.37***	-.06	.35***	.53***
UPPS-P Neg Urg														.35***	.36***	.16	.66***
UPPS-P Premed															.36***	.38***	.46***
UPPS-P Persev																.04	.37***
UPPS-P SS																	.40***
UPPS-P Pos Urg																	

Table 10 continued*First Order Correlations for Study 2 continued*

	k	Fz_E_L	F3_H_L	F4_E_L	F4_H_L
Age	-.17	-.16	-.10	-.14	-.09
Sex	.18	-.11	-.11	-.10	-.15
Alc Max	.19	-.01	-.01	.00	-.01
Alc Quant	.26*	.15	.11	.15	.10
Alc Freq	.11	.13	.09	.12	.08
Quant x Freq	.19	.15	.12	.15	.10
Binge	.25*	.14	.12	.11	.14
SMAST	.06	-.05	-.04	-.04	-.04
HPS Total	.21*	.22*	.18	.21*	.17
HPS Soc Vit	.17	.13	.08	.12	.07
HPS Mood Vol	.18	.30**	.25**	.29**	.26**
HPS Excitement	.21*	.15	.08	.13	.07
UPPS-P Neg Urg	.12	.20*	.20*	.21*	.21*
UPPS-P Premed	.12	.11	.05	.07	.05
UPPS-P Persev	.07	.07	.11	.05	.10
UPPS-P SS	.18	.14	.09	.10	.06
UPPS-P Pos Urg	.20*	.06	.03	.03	.03
k		-.05	-.03	-.05	-.08
Fz_E_L			.81***	.99***	.81***
F3_H_L				.81***	.98***
F4_E_L					.82***
F4_H_L					

Note: Alc Max = the natural log transformation of the lifetime maximum number of drinks within a 24-hour period, Alc Quant = the natural log transformation of the average quantity of alcohol consumed per occasion in the past 12 months, Alc Freq = the natural log transformation of the average frequency of alcohol consumption in the past 12 months, Quant x Freq = the natural log transformation of the quantity x frequency of drinks consumed in the past 12 months, Binge = the natural log transformation of the frequency of binge drinking in the last 12 months, SMAST = the natural log transformation of the Short Michigan Alcoholism Screening Test, HPS Total = Hypomanic Personality Scale Total Score, HPS Soc Vit = Hypomanic Personality Scale Social Vitality, HPS Mood Vol = Hypomanic Personality Scale Mood Volatility, HPS Excitement = Hypomanic Personality Scale Excitement, UPPS-

P Neg Urg = UPPS-P Impulsive Behaviour Scale Negative Urgency, UPPS-P Premed = UPPS-P Impulsive Behaviour Scale Lack of Premeditation, UPPS-P Persev = UPPS-P Impulsive Behaviour Scale Lack of Perseveration, UPPS-P SS = UPPS-P Impulsive Behaviour Scale Sensation Seeking, UPPS-P Pos Urg = UPPS-P Impulsive Behaviour Scale Positive Urgency, k = the natural log transformation of delay discounting rates, Fz_E_L = latency for the Fz scalp site of the easy condition of the visual oddball EEG task, $F3_H_L$ = latency for the F3 scalp site of the hard condition of the visual oddball EEG task, $F4_E_L$ = latency for the F4 scalp site of the easy condition of the visual oddball EEG task, $F4_H_L$ = latency for the F4 scalp site of the hard condition of the visual oddball EEG task.

* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$.

Hypomanic personality related to delay discounting.

The HPS total score was positively correlated with delay discounting rates ($p = .038$). Higher levels of hypomanic personality were related to steeper discounting rates. The HPS Excitement subscale was also positively correlated with discounting ($p = .037$). Neither Social Vitality nor Mood Volatility were correlated with delay discounting. When the HPS total score was examined in a regression analysis predicting discounting, the model accounted for a significant amount of the variance of discounting, R^2 adjusted = .03, $F(1, 100) = 4.44$, $p = .038$, and the HPS total score was a significant predictor ($\beta = .206$, $p = .038$). Related subscales were also examined in regression analyses predicting discounting rates and controlling for the effects of age and sex where relevant. In a model predicting discounting rates from Excitement, the model accounted for a significant amount of the variance of discounting, R^2 adjusted = .03, $F(1, 100) = 4.48$, $p = .037$, and Excitement was a significant predictor ($\beta = .207$, $p = .037$). These regression results are summarized in Table 11.

Table 11

Summary of Variables Involved in Mediation Models for Study 2 Predicting Delay Discounting

Dependent Variable		Delay Discounting (<i>k</i>)
Unique Predictors	Hypomanic Personality Scale (HPS)	Total Score (+) Excitement (+)
	UPPS-P	Positive Urgency (+)
	Alcohol Use	Quantity (+) Binge Drinking Frequency (+)

Note: HPS, UPPS-P, and alcohol use measures listed above were found to be unique predictors of delay discounting rates. The (+) or (-) denotes whether the variable was positively or negatively related to delay discounting.

Impulsivity related to delay discounting.

UPPS-P Positive Urgency was the only impulsivity scale correlated with delay discounting rates ($p = .041$). Individuals higher on Positive Urgency had steeper discounting rates than those lower on Positive Urgency. That is, these individuals were less likely to wait for the delayed rewards. Negative Urgency, Lack of Premeditation, Lack of Perseverance, and Sensation Seeking were not related to delay discounting.

In a regression analysis predicting discounting from Positive Urgency, the model accounted for a significant amount of the variance of discounting, R^2 adjusted = .03, $F(1, 100) = 4.30$, $p = .041$, and Positive Urgency was a significant predictor ($\beta = .203$, $p = .041$).

Mediation models using the HPS scales.

It was anticipated that the relationships between the HPS total score and delay discounting would be mediated by the related UPPS-P scales (i.e., Positive Urgency). Overall, the findings do not support mediation. In a mediation model predicting delay discounting from HPS total score with Positive Urgency as a possible mediator, the model did not indicate mediation. The model was not significant when both HPS total score and Positive Urgency were included in the model and neither were significant predictors. Likewise, when predicting delay discounting from the HPS Excitement subscale with Positive Urgency as a possible mediator, the model was not significant and neither predictor was significant. Therefore, it does not appear that the relationship between hypomanic personality and delay discounting is mediated by self-report impulsivity.

Alcohol use related to delay discounting.

In general, higher levels of alcohol use were related to steeper discounting rates. The only alcohol use measures positively correlated with delay discounting rates were quantity of

alcohol use ($p = .009$) and binge drinking frequency ($p = .010$). Lifetime maximum number of drinks within a 24-hour period, quantity x frequency, frequency of alcohol use, and alcohol related problems were not related to delay discounting rates.

Regression analyses were run predicting delay discounting from quantity of alcohol use and binge drinking frequency. In the regression model predicting discounting from quantity of alcohol use, the model accounted for a significant amount of the variance in discounting, R^2 adjusted = .06, $F(1, 100) = 7.14$, $p = .009$, and quantity of alcohol use was a significant predictor ($\beta = .258$, $p = .009$). In a regression model predicting discounting binge drinking frequency, the model accounted for a significant amount of the variance in discounting, R^2 adjusted = .06, $F(1, 100) = 6.92$, $p = .010$, and binge drinking frequency was a significant predictor ($\beta = .254$, $p = .010$). Overall, this is consistent with the hypothesis that higher alcohol use would be related to steeper delay discounting rates.

Mediation models using alcohol use.

It was anticipated that the relationships between the alcohol use and delay discounting would be mediated by the related UPPS-P scales (i.e., Positive Urgency). Overall, the findings do not support mediation. In a mediation model predicting delay discounting from quantity of alcohol use with Positive Urgency as a possible mediator, the model did not indicate mediation. That is to say, the model was significant when both quantity of alcohol use and Positive Urgency were included in the model, R^2 adjusted = .06, $F(2, 99) = 4.32$, $p = .016$, but only quantity of alcohol use was a significant predictor ($\beta = .212$, $p = .043$). Positive Urgency did not significantly mediate the relationship between quantity of alcohol use and delay discounting. Likewise, when predicting delay discounting from the binge drinking frequency with Positive Urgency as a possible mediator, the model was significant, R^2 adjusted = .06, $F(2, 99) = 4.26$, p

= .017, but only binge drinking frequency was a significant predictor ($\beta = .209, p = .046$).

Positive Urgency did not significantly mediate the relationship between binge drinking frequency and delay discounting. Therefore, it does not appear that the relationship between alcohol use and delay discounting is mediated by self-report impulsivity.

Hypomanic personality related to EEG measures.

There were fewer than anticipated correlations between hypomanic personality and the P300 ERP. The HPS total score was negatively correlated with P300 peak amplitude at Fz for the easy condition of the visual oddball task ($r = -.20, p = .045$); higher hypomanic personality was related to a smaller peak. No other sites were related to the HPS total score or any subscale score for the visual oddball task. Given the large number of variables examined and the lack of consistent findings, this relationship is likely indicative of the need for a larger, or more impaired, sample. The HPS total score was positively correlated with P300 latency at frontal sites for some conditions of the visual oddball task (Fz_Total, Fz_Easy, F3_Total, F3_Easy, F4_Total, F4_Easy, $.21 \leq r \leq .28, .004 \leq p \leq .031$); higher hypomanic personality was related to longer latencies. The Social Vitality subscale of the HPS was positively correlated with P300 latency at F3 and F4 in the total condition of the visual oddball task ($r = .21, .035 \leq p \leq .032$). Mood Volatility was positively correlated with P300 latency at some frontal, central, and parietal sites across all conditions of the visual oddball task (Fz_Total, Fz_Easy, Fz_Hard, F3_Total, F3_Easy, F3_Hard, F4_Total, F4_Easy, F4_Hard, Cz_Total, Cz_Easy, Cz_Hard, C3_Total, C3_Easy, C3_Hard, C4_Total, C4_Easy, C4_Hard, Pz_Total, Pz_Hard, P3_Total, P4_Total, P4_Hard, $.20 \leq r \leq .32, .001 \leq p \leq .047$). Excitement was not correlated with P300 latency for the visual oddball task. Neither the HPS total score, nor any subscale was correlated with P300 amplitude or latency for the auditory oddball task.

Post-hoc analyses examined a smaller restricted sample ($n = 50$) of only subjects who scored one standard deviation above or below the mean for their sex on the HPS total score; high and low HPS scorers, respectively. In this sample, meant to approximate bipolar participants and controls, there were markedly more correlations between hypomanic personality and P300 amplitude. The HPS total score was significantly negatively correlated with P300 peak amplitude at some frontal and central sites across all conditions of the visual oddball EEG task (Fz_Total, Fz_Easy, Fz_Hard, F3_Total, F3_Easy, F3_Hard, F4_Easy, C3_Easy, C4_Easy, $-.35 \leq r \leq -.31$, $.025 \leq p \leq .050$), as was the Social Vitality subscale (Fz_Total, Fz_Easy, Fz_Hard, F3_Total, F3_Easy, F3_Hard, F4_Easy, C3_Total, C3_Easy, C3_Hard, C4_Total, C4_Easy, C4_Hard, $-.39 \leq r \leq -.31$, $.011 \leq p \leq .045$). Mood Volatility was not correlated with peak amplitude at any sites, and Excitement was correlated with only one parietal site (P4_Easy, $r = -.33$, $p = .033$) for the easy (normal) condition. Higher hypomanic personality was related to longer latencies in the visual oddball task. HPS total score was positively correlated with frontal and central sites across all conditions (Fz_Total, F3_Total, F4_Total, F4_Easy, Cz_Total, C3_Easy, C4_Total, C4_Hard, $.31 \leq r \leq .37$, $.017 \leq p \leq .046$). Social Vitality was positively correlated with frontal and central sites across all conditions (Fz_Total, F3_Total, F3_Easy, F4_Total, F4_Easy, Cz_Total, C4_Total, C4_Hard, $.31 \leq r \leq .42$, $.006 \leq p \leq .047$). Mood Volatility was positively correlated with frontal, central, and parietal sites across all conditions (Fz_Total, Fz_Easy, F3_Total, F3_Easy, F4_Total, F4_Easy, Cz_Total, Cz_Easy, Cz_Hard, C3_Total, C3_Easy, C4_Total, C4_Easy, C4_Hard, P4_Hard, $.31 \leq r \leq .40$, $.009 \leq p \leq .049$). Excitement was not related to P300 latency for any site of the visual oddball task. In this subsample, hypomanic personality remained unrelated to P300 amplitude and latency for any site of the auditory task.

In this subsample of high and low HPS scorers, group differences were found for P300 peak amplitude at some frontal and central sites for some conditions of the visual oddball EEG task (Fz_Total, Fz_Easy, F3_Total, F3_Easy, F4_Easy, C3_Easy, C4_Easy, $2.07 \leq t(40) \leq 2.41$, $.020 \leq p \leq .045$); high HPS scorers had significantly smaller peaks than low HPS scorers. Only one group difference was found for the auditory oddball EEG task. Contrary to our hypothesis, high HPS scorers had higher P300 peak amplitudes at the P3 site ($t(37) = -2.09$, $p = .044$). Group differences were found for P300 latency in the visual oddball task. High HPS scorers had significantly longer latencies than low HPS scorers at some frontal and central sites for the total and hard conditions (F3_Total, F4_Total, C4_Hard, $-2.32 \leq t(40) \leq -2.14$, $.026 \leq p \leq .043$). There were no group differences for latency in the auditory task.

Impulsivity related to EEG measures.

No UPPS-P scale was related to P300 peak amplitude in either the visual or auditory oddball EEG task. Negative Urgency was the only UPPS-P scale correlated with P300 latency in the visual oddball task. Negative Urgency was positively correlated with P300 latency at some frontal sites for some conditions (Fz_Easy, F3_Hard, F4_Easy, F4_Hard, $.20 \leq r \leq .21$, $.030 \leq p \leq .042$). For the auditory oddball paradigm, only Sensation Seeking was negatively correlated with P300 latency at one site (P4, $r = -.21$, $p = .048$). However, given the lack of consistent pattern and the large number of variables involved, this last finding is likely spurious.

In post-hoc analyses of high and low HPS scorers, findings for amplitude remained unchanged, no UPPS-P scale was related to P300 amplitude at any site for either the visual or auditory oddball tasks. However, more relationships were seen for latency in the visual paradigm. Negative Urgency was positively correlated with P300 latency at some frontal and parietal sites for some conditions of the visual oddball task (Fz_Easy, F3_Easy, F4_Easy,

F4_Hard, P4_Hard, $.31 \leq r \leq .36$, $.019 \leq p \leq .045$). Sensation Seeking was positively correlated with P300 latency at frontal, central, and parietal sites across all conditions of the visual oddball task (Fz_Total, F3_Total, F4_Total, Cz_Total, Cz_Easy, Cz_Hard, C3_Total, C3_Easy, C3_Hard, C4_Total, C4_Hard, Pz_Total, Pz_Easy, Pz_Hard, P3_Total, P3_Hard, P4_Total, P4_Easy, P4_Hard, $.32 \leq r \leq .48$, $.001 \leq p \leq .042$). Lack of Premeditation, lack of Perseverance, and Positive Urgency were unrelated to latency in the visual oddball task. There continued to be no correlations between any UPPS-P scale and P300 latency for the auditory task.

Mediation.

In the full Study 2 sample, six mediation models were examined predicting latency in the visual oddball paradigm. The HPS total score and Mood Volatility were each examined as independent variables predicting FZ_Easy with Negative Urgency as a mediator. Age was included as a covariate in the Mood Volatility model. In both of these models, mediation was not supported. In a mediation model predicting F3_Hard from Mood Volatility with Negative Urgency as a possible mediator and controlling for the effects of age, mediation was not supported. In a mediation model predicting F4_Easy from the HPS total score with Negative Urgency as a possible mediator, mediation was not supported. In a mediation model predicting F4_Easy from Mood Volatility with Negative Urgency as a possible mediator and controlling for the effects of age, mediation was not supported. Finally, in a mediation model predicting F4_Hard from Mood Volatility with Negative Urgency as a possible mediator and controlling for the effects of age, mediation was not supported. See Table 12 for a summary of the variables

used in these mediation models. No mediation models were supported predicting latency in the visual paradigm from hypomanic personality with impulsivity as a mediator.¹

Table 12

Summary of Variables Involved in Mediation Models for Study 2 Predicting EEG Measures

Dependent Variable	Visual P300 ERP Latency	FZ_Easy	F3_Hard	F4_Easy	F4_Hard
Unique Predictors	Hypomanic Personality Scale	Total Score (+)		Total Score (+)	
		Mood Volatility (+)	Mood Volatility (+)	Mood Volatility (+)	Mood Volatility (+)
	UPPS-P	Negative Urgency (+)	Negative Urgency (+)	Negative Urgency (+)	Negative Urgency (+)

Note: No alcohol use measure uniquely predicted P300 latency. The (+) or (-) denotes whether the variable was positively or negatively related to P300 latency. UPPS-P = UPPS-P Impulsive Behavior Scale.

Alcohol use related to EEG measures.

Contrary to my hypothesis, no measure of alcohol use was related to P300 peak amplitude at any site in the visual oddball task. Quantity of alcohol use was negatively correlated with P300 peak amplitude at the C3 site for the auditory task ($r = -.23, p = .029$). Quantity of alcohol use was positively correlated with P300 latency at Fz, F3, and F4 for the total condition of the visual oddball task ($.21 \leq r \leq .23, .018 \leq p \leq .034$). Also in the total condition of the visual oddball task, quantity x frequency of alcohol use was positively correlated with latency at F3 ($r = .19, p = .049$) and binge drinking frequency was positively correlated with latency at F4 ($r = .21, p = .039$). No measure of alcohol use was related to P300 latency in the

¹ In the interest of being thorough, mediation models were also examined in the subsample of high and low HPS scorers. In this sample, there were 30 potential mediation models, only two of which were significant for full mediation. These significant findings were likely spurious given the number of analyses run.

auditory task. As there were no UPPS-P scales that could be potential mediators of the above relationships, no mediation models were examined.

In post-hoc analyses of high and low HPS scorers, alcohol related problems (SMAST) was negatively correlated with P300 peak amplitude at some frontal and central sites for some conditions of the visual oddball EEG task (Fz_Total, Fz_Easy, Fz_Hard, F3_Total, F3_Easy, F3_Hard, F4_Easy, C3_Total, C3_Easy, $-.41 \leq r \leq -.31$, $.007 \leq p \leq .048$). Higher alcohol related problems was related to lower P300 peak amplitude. For the auditory task, quantity of alcohol use was negatively correlated with C3 and C4 peak amplitude ($-.36 \leq r \leq -.34$, $.024 \leq p \leq .035$); higher quantity of alcohol use was related to lower peaks. With regard to latency, higher alcohol use was related to longer latencies. Quantity x frequency of alcohol use was positively correlated with P300 latency at F3 and F4 in the total condition of the visual oddball task ($.31 \leq r \leq .32$, $.040 \leq p \leq .046$) and binge drinking frequency was positively correlated with frontal sites across the total and easy conditions (Fz_Total, F3_Total, F3_Easy, F4_Total, F4_Easy $.30 \leq r \leq .34$, $.030 \leq p \leq .050$). No other measure of alcohol use was related to P300 latency in the visual task. No sites were correlated with any measure of alcohol use for P300 latency in the auditory task.²

Additional post-hoc analyses examined high and low drinkers, those scoring one standard deviation or more above (high) or below (low) the mean on the log transformed quantity x frequency measure of alcohol use. In this restricted sample ($n = 28$), meant to approximate clinical and control participants, group differences between high and low drinkers were found for several EEG measures. In the visual Rotated Heads task, smaller P300 amplitudes were seen in high, compared to low, drinkers at the Cz, C3, and C4 sites in the total condition ($2.20 \leq t(26) \leq$

² In this subsample of high and low HPS scorers, 7 possible mediation models were examined, but none were found to be significant.

2.36, $.026 \leq p \leq .037$) and the Fz, Cz, C3, and C4 sites in the hard condition ($2.32 \leq t(25) \leq 2.88$, $.008 \leq p \leq .029$). Additional marginally significant group differences were seen at the Fz site of the total condition, the F3, F4, and Pz sites of the hard condition, and Fz, Cz, C3, and C4 sites of the easy condition. There were also group differences for longer latencies in high drinkers at the F3 and F4 sites of the total condition ($-2.53 \leq t(25) \leq -2.26$, $.018 \leq p \leq .043$) and at the F4 site for the easy condition ($t(25) = -2.29$, $p = .044$). Marginally significant group differences were also seen at the Fz site of the total condition, the Fz, F3, and F4 sites of the hard condition, and the Fz, F3, Cz, C3, and C4 sites for the easy condition. In the auditory task, significantly smaller P300 amplitudes were found for high drinkers at all sites ($2.83 \leq t(26) \leq 4.24$, $p \leq .009$). However, there were no differences for P300 latency.

Male only subsample.

Since some literature on the visual oddball task has focused solely on male participants (e.g., Begleiter et al., 1984; Carlson & Iacono, 2006; Carlson, Katsanis, Iacono, & Mertz, 1999; Iacono et al., 2002), a subsample ($n = 36$) of only males was examined to ensure that relationships were not being masked by the inclusion of females. In this subsample, there were markedly more first order correlations between P300 amplitude and latency in the visual oddball task and UPPS-P scales, HPS scales, and alcohol use measures. However, ultimately, no significant mediation models were found.

Discussion

In general, correlations between the variables examined in Study 1 (hypomanic personality, impulsivity, and alcohol use) remained consistent in this Study 2 subsample of participants.

Hypomanic personality related to delay discounting.

HPS total score was positively correlated with delay discounting rates this relationship remained in regression analyses where HPS total score uniquely predicted discounting rates. Individuals higher on hypomanic personality had steeper discounting rates, meaning they were less inclined to wait the delay period to receive the larger rewards. In examining the HPS subscales, only Excitement was positively correlated with discounting rates and this relationship remained in a regression analysis. Since the HPS subscales are not well researched, it is difficult to say why this scale emerged as being related to discounting rates. However, this scale is a measure of an energetic and cheerful mood and behavioural impulsivity, such as that measured by the delay discounting task, has been found to be state dependent in bipolar participants. That is, subjects in manic states performed more impulsively than those in mixed or euthymic states (Najt et al., 2007; Strakowski et al., 2009). Therefore, it is possible that high scores on the Excitement scale would suggest manic tendencies and this is related to the inability to delay rewards.

Impulsivity related to delay discounting.

UPPS-P Positive Urgency was the only impulsivity scale positively correlated with delay discounting rates. Higher impulsivity was related to steeper discounting rates, meaning that those higher on impulsivity were more likely to choose the smaller immediate reward over the larger delayed reward. As with the HPS Excitement scale, Positive Urgency relates to positive mood states and therefore may be more likely than Negative Urgency or other UPPS-P scales to be related to steeper delay discounting rates.

No mediation models were significant when predicting delay discounting from hypomanic personality with impulsivity as a possible mediator.

Alcohol use related to delay discounting.

Overall, more alcohol use was related to steeper discounting rates. That is, individuals who consumed more alcohol were less likely to wait for the larger rewards. Both quantity of alcohol use and binge drinking frequency were related to delay discounting rates. Correlational relationships held up in regression analyses and these results are in line with past research supporting the relationship between higher alcohol use and steeper discounting rates (Bobova et al., 2009; Kollins, 2003; MacKillop et al., 2011; Petry, 2001; Vuchinich & Simpson, 1998; Yi et al., 2010). Quantity of alcohol use and binge drinking frequency are similar in that they are measures of excessive amounts of alcohol consumption. Quantity of alcohol use measures how many drinks, on average, the participants consumes when they drink and binge drinking frequency asks how often they consume high amounts of alcohol. Therefore, delay discounting appears to be related to excessive drinking (i.e., large amounts of alcohol at one time), rather than social drinking or frequency of drinking.

No mediation models were significant when predicting delay discounting from alcohol use with impulsivity as a possible mediator.

Hypomanic personality related to EEG measures.

A pattern emerged showing hypomanic personality related to P300 latency, but not amplitude, for the visual paradigm and no associations were found for the auditory task. HPS total score was positively related to P300 latency at frontal sites for the visual oddball task. Frontal sites may be implicated because of the poor emotion regulation inherent in bipolar disorder, or those high on hypomanic personality. When examining the HPS subscales, Mood Volatility emerged as the scale with the most associations. Mood Volatility was related to P300 latency at frontal, central, and parietal sites in the visual oddball task. As this scale encompasses

the emotion dysregulation aspect of hypomanic personality and bipolar disorder, it may be more sensitive to differences in P300 latency. One previous study also found differences between bipolar participants and controls on latency, but not amplitude, in a visual oddball paradigm (Bange & Bathien, 1998). That study examined bipolar participants in a depressive state, suggesting that latency differences are sensitive to mood state.

Impulsivity related to EEG measures.

There were few findings of impulsivity being related to EEG measures in the current study. No UPPS-P scale was related to P300 amplitude in either the visual or auditory oddball task. This is contrary to the findings of other studies relating higher self-report impulsivity to smaller P300 amplitudes (Barratt et al., 1997; Chen et al., 2007; De Pascalis, Strippoli, Riccardi, & Vergari, 2004; Harmon-Jones et al., 1997; Moeller et al., 2004; Shen, Lee, & Chen, 2014; Venables, Patrick, Hall, & Bernat, 2011). However, not all of these studies used the same oddball paradigms to elicit the P300 (e.g., De Pascalis et al., 2004; Shen et al., 2014). One study examining bipolar participants also failed to find any relationship between self-report impulsivity and EEG measures, although this study was looking at the P50, N100, and P200 evoked potentials (Swann et al., 2013). Only Negative Urgency was related to P300 latency for some frontal sites in the visual oddball task, such that higher scores on Negative Urgency were related to longer latencies. Latency is not as commonly studied as amplitude with regard to the P300 ERP as related to impulsivity. However, one study did report no association between P300 latency in a visual oddball paradigm and self-report impulsivity as measured by the BIS-11 (Russo et al., 2008).

There were no significant mediation models predicting P300 amplitude or latency in the visual or auditory oddball tasks from hypomanic personality with impulsivity as a mediator. The

most likely explanation for this lack of mediation is that this particular sample had a restricted range combined with the use of measures which may not be sensitive enough to robustly differentiate within a non-clinical sample.

Alcohol use related to EEG measures.

No measure of alcohol use was related to P300 peak amplitude at any site in the visual oddball task. These findings are very surprising given the large body of research supporting a relationship between alcohol use and smaller P300 amplitudes on visual oddball tasks (e.g., Carlson et al., 2007; Chen et al., 2007; Hesselbrock et al., 2001; Iacono et al., 2003; Polich et al., 1994; Porjesz & Begleiter, 1990). Quantity of alcohol use was negatively related to P300 amplitude at one site in the auditory oddball task. Quantity of alcohol use, quantity x frequency of alcohol use, and binge drinking frequency showed positive correlations with latency at some frontal sites in the visual oddball task. No measure of alcohol use was related to P300 latency in the auditory task. Since no UPPS-P scales were potential mediators for the above relationships, no mediation models were examined.

In post-hoc analyses of the high and low HPS scorers (the subsample with those scoring at the extremes on hypomanic personality), alcohol related problems was negatively correlated with P300 peak amplitude at some frontal and central sites in the visual oddball task. In addition, quantity of alcohol use was negatively correlated with some central sites in the auditory task. Higher alcohol use was also related to longer latencies in the visual oddball task. Quantity x frequency of alcohol use and binge drinking frequency were both positively correlated with latency at frontal sites. Alcohol use was not related to latency in the auditory task. These additional associations are more in line with past research supporting the relationship between higher alcohol use and lower P300 amplitude and longer latencies. This suggests that the current

sample may not be impaired enough, with regard to hypomanic personality or alcohol use, for the previously established associations between alcohol use and the P300 ERP to be seen.

Male only subsample.

In a small subsample of only males, there were markedly more correlations between measures of impulsivity, hypomanic personality, and alcohol use and P300 amplitude and latency in the visual oddball task. However, any potential mediation models were not significant, likely due to the low power in this very small sample. Future research may want to focus solely on males, with a large enough sample of males, there may be the mediation models that were not found here.

Chapter 4: General Discussion

Study 1 found that higher hypomanic personality predicted higher impulsivity and alcohol use. Higher impulsivity also predicted more alcohol use and mediation models examining the relationship between hypomanic personality and alcohol use as mediated by impulsivity were significant. Specifically, for analyses involving the HPS total score, Negative Urgency and Sensation Seeking were the UPPS-P scales most often found to mediate the relationship between hypomanic personality and alcohol use. It is not surprising that Sensation Seeking and Negative Urgency emerged as the only UPPS-P scales to mediate the relationship between hypomanic personality and alcohol use given that these were the scales most often related to measures of alcohol use. Individuals high on Sensation Seeking may view alcohol use as an exciting activity and therefore engage in more use (Stautz & Cooper, 2013; Zuckerman, 1994). This may be especially true in undergraduate samples, where many individuals have just reached legal drinking age and are increasing their social use of alcohol. However, those high on Negative Urgency not only partake in consumption, but also have more alcohol related problems, possibly because of the strong emotional involvement (Stautz & Cooper, 2013) or because they are using alcohol as a coping mechanism (Jones, Chryssanthakis, & Groom, 2014).

Previous research suggests that alcohol use disorders, bipolar disorder, and impulsivity can all be linked to a broader externalizing dimension related to behavioural disinhibition (Iacono et al., 2006; Krueger et al., 2002; Widiger & Clark, 2000), which has been found to be highly heritable (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Krueger et al., 2002). It may be this underlying externalizing dimension which is inherited, but which may be expressed differently in different individuals. This is supported by Study 1 findings that the relationship between hypomanic personality and alcohol use is mediated by impulsivity, a form of

behavioural disinhibition. The current study further adds to this line of research by suggesting different motivational pathways based on how this externalizing dimension is expressed.

Specifically, if this externalizing dimension manifests as Sensation Seeking, the individual may be more susceptible to participate in risky behaviours (e.g., alcohol use) as a means of novelty seeking. Whereas, if it manifests as Negative Urgency, the individual may be more susceptible to participate in risky behaviours as a means of coping with strong emotionality.

While there are common etiological factors for bipolar and alcohol use disorders, there are also important etiological and vulnerability differences. For example, individuals may be more vulnerable to the development of alcohol use disorders based on sex (Cooper, Russell, Skinner, Frone, & Mudar, 1992; Hilton, 1987), age at first alcohol use (DeWit, Adlaf, Offord, & Ogborne, 2000), exposure to stressful life events (Cooper et al., 1992; Keyes, Hatzenbuehler, & Hasin, 2011), coping styles (Cooper, Frone, Russell, & Mudar, 1995; Cooper et al., 1992), peer influence (Trucco, Colder, & Wieczorek, 2011), or alcohol outcome expectancies (Cooper et al., 1992). Whereas vulnerability to bipolar disorder may be marked by cognitive deficits (Clark, Iversen, & Goodwin, 2002; Robinson et al., 2006; Zalla et al., 2004) or early parental loss (due to separation or death; Agid et al., 1999).

When examining the HPS subscales Social Vitality was positively related to all measures of alcohol use, except for alcohol related problems, and all these relationships were partially mediated by Sensation Seeking. Mood Volatility positively predicted alcohol related problems, and this relationship was partially mediated by Negative and Positive Urgency. The relationship between Social Vitality and Sensation Seeking may be related to the common interest of risk-taking. In the study which determined the HPS subscales, Social Vitality reflected a tendency towards risk taking and was the subscale most strongly related to measures of positive

emotionality, such as extraversion and social potency (Schalet et al., 2011). Sensation Seeking has also been related to risk-taking behaviour (Donohew et al., 2000; Rosenbloom, 2003; Whiteside & Lynam, 2001; Zuckerman, 2007) and extraversion (Aluja, García, & García, 2003; Eysenck & Zuckerman, 1978). The relationship between Mood Volatility and Negative and Positive Urgency likely has to do with the high level of emotionality linked to all these measures.

Because of the lack of research on the HPS subscales, it is difficult to determine why the Social Vitality scale stands out as the subscale of interest. However, based on the description of the scale as a measure of sociability, it stands to reason that Social Vitality may be most related to alcohol use in this undergraduate sample, as undergraduates tend to be especially social drinkers. Likewise, other studies have frequently found Sensation Seeking to be related to alcohol use in undergraduate samples (Curcio & George, 2011; Cyders, 2013; Cyders et al., 2009; Jones et al., 2014; LaBrie et al., 2014; Magid & Colder, 2007; Stautz & Cooper, 2013)

Study 2 found that hypomanic personality (total score and Excitement) was positively related to delay discounting rates. This is consistent with past research finding steeper discounting rates in bipolar participants or those high on hypomania, compared to controls (Ahn et al., 2011; Mason et al., 2012). These findings contribute to the sparse, but growing, literature on the relationship between hypomanic personality and delay discounting rates.

UPPS-P Positive Urgency was positively correlated with delay discounting rates, supporting this hypothesis. However, no other scales of the UPPS-P were related to discounting rates. The finding that Positive Urgency, but not Negative Urgency, is positively related to discounting rates is consistent with past findings that behavioural impulsivity is state-dependent in bipolar participants (Najt et al., 2007; Strakowski et al., 2009). This highlights the idea that subjects in a positive mood state (e.g., manic), but not a negative mood state, are more likely to

show impulsivity on this kind of behavioural task. It has been suggested that behavioural impulsivity is more state-dependent, whereas self-report impulsivity reflects more of a stable personality trait (Dick et al., 2010). Additionally, animal models examining the difference between impulsive choice (i.e., impulsive decision making), which is measured by delay discounting tasks, and impulsive action (i.e., behavioural disinhibition; e.g., Dalley, Everitt, & Robbins, 2011; Uslaner & Robinson, 2006; Winstanley, Dalley, Theobald, & Robbins, 2004; Winstanley, Theobald, Dalley, Cardinal, & Robbins, 2006) further support the idea of different facets of impulsivity which are separately measurable. One study found impulsive choice, impulsive action, and self-report impulsivity to be separate, unrelated constructs in healthy human volunteers, indicating that self-report impulsivity and behavioural measures of impulsivity tap different facets (Broos, Schmaal, Wiskerke et al (2012). In that study, self-report impulsivity was measured by the BIS-11 which is reflected in the UPPS-P scales of lack of Premeditation and Negative Urgency (Whiteside & Lynam, 2001), consistent with the current study's findings that these scales were unrelated to delay discounting. This is also consistent with other research finding no relationship between self-report impulsivity and behavioural measures of impulsivity (Ahn et al., 2011; Crean et al., 2000; Strakowski et al., 2010). Since there are clearly several different facets of impulsivity and not all facets are necessarily present in the same individual or at the same time, it then follows that delay discounting, a behavioural measure of impulsive choice, would not be related to most scales of the UPPS-P, a self-report measure of trait impulsivity, with the exception of the Positive Urgency scale measuring a tendency to act rashly in a positive affective state which, as mentioned above, has been related to delay discounting previously.

Quantity of alcohol use and binge drinking frequency were related to steeper discounting rates. This is consistent with the idea that those who drink excessively do so for the immediate rewards that alcohol offers (e.g., increased sociability) without considering the long-term consequences of drinking heavily (e.g., being hung over; Mitchell, 2011; Yi et al., 2010). No mediation models were significant predicting delay discounting from hypomanic personality or alcohol use with impulsivity as a mediator.

With regard to EEG data, both hypomanic personality and impulsivity were related to P300 latency at some frontal sites in the visual paradigm, but no relationships were seen with amplitude and no relationships were seen with amplitude or latency for the auditory task. For alcohol use, there was one association with P300 amplitude for the auditory task, and some associations with latency for the visual task, but much fewer than were anticipated given past research.

There are many possible explanations for this unexpected lack of findings, most of which apply to the alcohol use - P300 amplitude relationship, and are discussed below. However there are also some possible explanations for the lack of relationship between the P300 ERP and hypomanic personality and impulsivity, which will be discussed next. It is possible that another type of task would have been better able to examine the relationship between hypomanic personality and ERP amplitude. For example, one study found a significant negative relationship between amplitude, using intensity dependence of auditory evoked potentials (IAEP), and hypomanic personality. IAEP examines change in amplitude related to increasing intensity of the tones being presented, as measured by the N100 and P200 components (Hensch, Herold, & Brocke, 2007). IAEP is thought to be a biological indicator of serotonergic neurotransmission (Hegerl, Gallinat, & Juckel, 2001; Hegerl & Juckel, 1993), which has also been linked to bipolar

disorder (Cuellar, Johnson, & Winters, 2005; Mahmood & Silverstone, 2001). Future studies attempting to link ERP amplitude and hypomanic personality may want to incorporate IAEP.

This sample of undergraduate students may not be impaired enough (have enough high scorers on the HPS) to show the same results as previous studies using samples of bipolar individuals (Bange & Bathien, 1998; Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009; Hall et al., 2007; O'Donnell et al., 2004; Schulze et al., 2008). The mean HPS score for the current sample was significantly lower than mean HPS score in the sample used to validate the HPS (Eckblad & Chapman, 1986). Consistent with this idea, in post-hoc analyses with the subsample of high and low HPS scorers, the HPS total score and Social Vitality subscale showed markedly more negative associations with P300 peak amplitude at frontal and central sites for the visual oddball task. Additionally, group differences were seen for amplitude and latency at some Rotated Heads sites. This supports the idea that this study sample may not be impaired enough to highlight these associations in the full sample. Likewise, there were more associations found in this subsample for higher hypomanic personality (HPS total score, Social Vitality, and Mood Volatility) being related to longer P300 latencies at both frontal and central sites for the visual oddball task. Hypomanic personality remained unrelated to P300 amplitude and latency in the auditory task. While the P300 ERP seems to differentiate well between bipolar participants and healthy controls, it may not be sensitive enough to highlight differences within non-clinical personality traits along the bipolar spectrum.

As with the HPS, past research has sometimes used clinical participants when showing associations between impulsivity and EEG measures (e.g., Chen et al., 2007; Moeller et al., 2004) so, as mentioned above, it is possible that the participants in the current study were not impaired, or impulsive, enough for these associations to come out. While the current samples

had UPPS-P scores similar to the undergraduate samples on which the scales were validated, studies using clinical samples (e.g., Whiteside & Lynam, 2003; Whiteside, Lynam, Miller, & Reynolds, 2005) had markedly higher scores on Negative Urgency, lack of Premeditation, lack of Perseverance, and Sensation Seeking. Positive Urgency was not measured in these studies. Post-hoc analyses in the more restricted sample of participants scoring at either extreme (one standard deviation above or below the mean) on hypomanic personality continued to show no relationship between impulsivity and P300 amplitude in either the visual or auditory task. However, more correlations emerged for latency in the visual paradigm. Negative Urgency remained positively correlated with P300 latency at some frontal sites and also a parietal site. In addition, Sensation Seeking was positively correlated with P300 latency at frontal, central, and parietal sites. Lack of Premeditation, Lack of Perseverance, and Positive Urgency remained unrelated to P300 latency in the visual task and there continued to be no correlations between impulsivity and P300 latency in the auditory task.

Additionally, one study found an association between impulsivity and reduced P300 amplitude in males, but not females, using a visual paradigm (Justus et al., 2001). Other studies have also focused on males only when examining relationships with the P300 ERP (e.g., Begleiter et al., 1984; Carlson & Iacono, 2006; Carlson, Katsanis, Iacono, & Mertz, 1999; Iacono et al., 2002). Since the current sample was 62 percent female, this may have impaired our ability to see associations between impulsivity and P300 amplitude. When examining only males from this sample, who were only significantly higher than females on Sensation Seeking, there were more correlations between impulsivity and P300 amplitude and latency in the visual paradigm. Perhaps in a larger sample that included more participants at extreme levels of hypomanic personality or impulsivity (i.e., more impaired) and more male participants, there would be more

clear associations between impulsivity and the EEG measures. Future studies may want to focus on male only samples, or clinical samples which better capture the full range hypomanic personality and impulsivity.

While there is ample research to support reduced P300 amplitude related to alcohol use (e.g., Carlson et al., 2007; Chen et al., 2007; Hesselbrock et al., 2001; Iacono et al., 2003; Polich et al., 1994; Porjesz & Begleiter, 1990), there have been many other studies which failed to find this association (Biggins, MacKay, Poole, & Fein, 1995; Cadaveira, Grau, Roso, & Sanchez-Turet, 1991; Crego et al., 2012; Cuzen, Andrew, Thomas, Stein, & Fein, 2013; Hill, Locke, & Steinhauer, 1999; Hill, Steinhauer, & Locke, 1995; Keenan, Freeman, & Harrell, 1997; Steinhauer, Hill, & Zubin, 1987). Some possible reasons for failing to find a relationship between higher alcohol use and a reduced P300 amplitude include having samples with less severe levels of alcohol use (Cuzen et al., 2013; Steinhauer et al., 1987), a lower genetic risk for alcohol use disorders (i.e., lack of family history of alcohol use disorders in the group of interest; Cuzen et al., 2013; Pfefferbaum et al., 1991; Polich et al., 1994), a lack of psychiatric comorbidity (Cuzen et al., 2013), adult participants, versus children or adolescents (Hill et al., 1995, 1999; Polich et al., 1994), a restricted range of intelligence (Polich et al., 1994), or a lack of male participants (Euser et al., 2012; Parsons, Sinha, & Williams, 1990).

Many previous studies have compared groups of current or abstinent alcoholics, those diagnosed with past or present alcohol use disorders, to control subjects (Carlson et al., 2002; Euser et al., 2012; Hesselbrock et al., 2001). In addition, a meta-analysis found that effect sizes for P300 amplitude reductions were larger when participants were recruited from treatment centres, compared to community or family study recruitment, suggesting that severity of the substance use disorder is important (Euser et al., 2012). The current study examined alcohol use

(lifetime maximum number of drinks within a 24-hour period, quantity of alcohol use, frequency of alcohol use, quantity x frequency, binge drinking frequency, and alcohol related problems) in university students. While consuming at least one drink in the past year was a requirement of the study and there were some heavy drinkers in our sample, alcohol use was positively skewed and needed to be log transformed because there were many more light to moderate drinkers in the sample. It is possible that the current sample had relatively lower lifetime exposure of alcohol use and that their use was not high enough to show the associations previously found with the P300. This hypothesis is supported by group differences between high and low drinkers on several EEG measures. These findings suggest that our sample did not have enough heavy drinkers as would be needed to see these associations in the full sample. As mentioned with regard to impulsivity and hypomanic personality, studies examining the P300 ERP would likely benefit from using clinical samples, or ensuring that there are enough heavy drinkers to represent the full spectrum of alcohol use.

There is strong evidence to suggest that the P300 ERP is genetically transmitted and that those with a family history of alcohol use disorders will show reduced P300 amplitude, regardless of their own disorder status or drinking history (Pfefferbaum et al., 1991; Porjesz et al., 1998). For example, in an auditory oddball task, alcoholics with alcoholic relatives had significantly reduced P300 amplitudes compared to controls, while no significant difference was found between controls and alcoholics without alcoholic relatives (Cohen et al., 1995). The current study did not include a measure of family history of alcohol use disorders, so it may be that those with a family history of, and genetic predisposition to, alcohol use disorders would show reduced P300 amplitude compared to those without a family history of alcohol use

disorders, regardless of their level of alcohol use. That is to say, that perhaps family history moderates the relationship between alcohol use and P300 amplitude.

There is also evidence that psychiatric comorbidity is related to the P300 ERP. Studies have found reduced P300 amplitude or longer latencies related to schizophrenia (Bramon, Rabe-Hesketh, Sham, Murray, & Frangou, 2004; Jeon & Polich, 2003), depression (Hill et al., 1999; Himani, Tandon, & Bhatia, 1999), bipolar disorder (Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009; Hall et al., 2007; O'Donnell et al., 2004), and antisocial personality disorder (Bauer, O'Connor, & Hesselbrock, 1994; Costa et al., 2000). For example, Hill and colleagues (1999) failed to find a P300 amplitude reduction, using both visual and auditory paradigms, in adult alcoholics unless they were comorbid for depression. Participants were excluded from participating in the current study if they were taking medication for a psychiatric disorder or had a history of drug abuse or treatment and since no diagnostic interview was conducted to determine if participants met criteria for a psychiatric disorder, this variable could not be used in analysis. Psychiatric comorbidity may have also affected results examining the relationships of P300 amplitude and latency to impulsivity and hypomanic personality.

In two studies conducted by Hill and colleagues (Hill et al., 1995, 1999) there was no difference found between adult alcoholics and controls on P300 amplitude using both visual and auditory paradigms. The authors suggested that P300 amplitude reductions commonly seen in children and adolescents at high-risk for developing alcoholism may represent a “neurodevelopmental delay that normalizes by adulthood” (p.982; Hill et al., 1999). This hypothesis is supported by findings in a meta-analysis that greater P300 reductions were seen in samples under age 18, compared to older samples (Polich et al., 1994). However, many studies have found P300 differences in adult samples (Chen et al., 2007; Euser et al., 2012; Polich et al.,

1994; Porjesz & Begleiter, 1990; Ramachandran et al., 1996) and a second meta-analysis found marginally larger effect sizes for older (over 18 years of age) compared to younger samples (Euser, et al., 2012), making this explanation less likely.

Since more effortful processing is related to smaller P300 amplitudes and longer latencies (Polich, 2007) and intelligence has been found to be positively correlated with P300 amplitude in a visual oddball task (Russo et al., 2008), it is possible that this undergraduate sample was not challenged enough by the tasks. Because our sample was university students, there may be a restricted range with regard to intelligence and they may be of greater than average intelligence. This may limit the variability in the P300 seen in this sample. Intelligence and education level have also been found to be inversely related to delay discounting rates (de Wit et al., 2007), which may have impacted results with that variable as well. A community or clinical sample may have more variability in this respect as well as with regard to impairment.

Another factor that may influence the results seen with regard to the P300 is gender distribution of the sample. Much of the research supporting the relationship between higher alcohol use and smaller P300 ERPs has been solely conducted on males (Begleiter et al., 1984; Carlson & Iacono, 2006; Carlson et al., 1999; Iacono et al., 2002; Polich et al., 1994) and some studies have even found associations in males, but not females (Euser et al., 2012; Parsons et al., 1990). As mentioned with regard to impulsivity, since 62 percent of the current sample was female, there may not have been enough males to show the associations found in previous studies. Restricted range of intelligence and lack of male participants may have also affected results examining the relationships of P300 amplitude and latency to impulsivity and hypomanic personality.

Contributions to the Literature

With over a thousand undergraduate student drinkers having completed demographic information, multiple measures of personality (i.e., the HPS and UPPS-P), and alcohol use, this dataset is rich with information. The large sample size ($n = 1194$), is a strength of Study 1 which also sets it apart from many other studies in the literature.

Study 1 examined the important link between hypomanic personality and alcohol use as mediated by impulsivity. Hypomanic personality no longer (or less strongly) predicted alcohol use when impulsivity was included in the model. This is an important finding given the amount of research supporting the relationship between hypomanic personality and alcohol use. It suggests that a person's level of impulsivity may be more important than their level of hypomanic personality in determining their risk for alcohol use and problems. It also suggests that the relationship between bipolar disorder and alcohol use may be dependent on impulsivity, highlighting the idea that impulsivity may be the underlying construct common to both bipolar disorder and alcohol use disorders.

This study also contributes to the growing literature on differential relationships of facets of impulsivity related to different alcohol use outcome measures. Specifically, those high on Sensation Seeking may be at risk for increased frequency of use and a higher lifetime maximum number of drinks within a 24-hour period. Those high on Negative Urgency may be at risk for increased alcohol related problems. Individuals high on either Sensation Seeking or Negative Urgency may be at risk for higher quantity of alcohol use, higher overall consumption levels (quantity x frequency), and more frequent binge drinking. These trait specific risk factors may have implications for targeting interventions to individuals most at risk. This is particularly useful on college campuses where it may be important to differentiate between social drinkers

(high Sensation Seekers) and those at risk for serious alcohol-related problems due to negative emotionality (those high on Negative Urgency). High Sensation Seekers may benefit from interventions aimed at re-directing their quest for novelty and excitement to more socially adaptable goals (e.g., meeting new people, playing sports) rather than having them focus on the stimulation brought on by drinking. Individuals high on Negative Urgency may be targeted for psychological intervention of mood or given strategies, other than drinking, for improving their mood or coping with distress. Stautz and Cooper (2013) suggested that the emotion regulation and distress tolerance skills of dialectical behaviour therapy (DBT) may be helpful for individuals high on Negative Urgency. In fact, these skills have previously been adapted to apply specifically to substance use (Dimeff & Linehan, 2008) and have been used to successfully reduce substance use (Linehan et al., 1999, 2002).

Personality specific interventions have also been examined by Conrod, Castellanos-Ryan, and Mackie (2011) who looked at the long-term effects of personality-targeted group interventions for alcohol use in adolescents. Personality traits targeted were Impulsivity, Sensation Seeking, Hopelessness, and Anxiety Sensitivity. They examined intervention effects on drinking quantity x frequency, binge drinking frequency, problem drinking, and motivations for alcohol use at 6, 12, 18, and 24 months post-intervention. In the treatment groups, alcohol use (quantity x frequency and binge drinking frequency) showed a trend toward being lower than the control groups at only the 6 month follow-up, but benefits of treatment remained to the 2 year follow-up for alcohol related problems. This suggests that, at least in the short-term, personality-based interventions may be effective in reducing alcohol use in young people. The intervention used by Conrod and colleagues (2011) consisted of two 90-minute group sessions designed to

promote personality-specific coping skills. Perhaps more intervention sessions, or sessions repeated over some intervals (e.g., every 6 months) may produce longer-lasting effects.

Palmgreen and colleagues (e.g., Palmgreen, Donohew, Lorch, Hoyle, & Stephenson, 2001; Palmgreen, Donohew, Lorch, Rogus, Helm, & Grant, 1991; Palmgreen, Lorch, Stephenson, Hoyle, & Donohew, 2007) have studied how to present anti-drug public service announcements to appeal to high sensation seeking adolescents, which has been shown to reduce their cannabis use (Palmgreen et al., 2001; Palmgreen et al., 2007). These campaigns, which used the Sensation Seeking Targeting (SENTAR) prevention approach, featured dramatic depictions of the negative consequences of drug use to target high Sensation Seekers by eliciting strong sensory arousal (Palmgreen et al., 2007). These same tactics may be adapted for the prevention or reduction of alcohol use in high Sensation Seekers.

Study 2 examined the relationships between impulsivity, hypomanic personality, and alcohol use to delay discounting and EEG measures. While there were not as many significant findings as anticipated and mediation models were not significant predicting delay discounting and not able to be examined predicting P300 amplitude, this study nonetheless makes important contributions to the literature. This study adds to the literature regarding delay discounting and self-report impulsivity. Specifically, this was the first study, of which I am aware, to examine the relationship between delay discounting and the UPPS-P, a measure of self-report impulsivity which is gaining popularity. This study found that only Positive Urgency was related to delay discounting, possibly because a maladaptive positive mood state is related to the inability to delay rewards. The lack of relationship between most UPPS-P scales and delay discounting is consistent with other studies reporting no relationship between delay discounting and self-report impulsivity (Ahn et al., 2011; Crean et al., 2000; Strakowski et al., 2010). This suggests that

these two measures likely tap different aspects of impulsivity. Further research is needed to clarify this and establish the functional consequences of it.

Study 2 also contributes to the literature regarding the relationship between bipolar disorder and delay discounting. To date, only one other study (Mason et al., 2012) has examined the relationship between the HPS, which has been shown to predict bipolar disorder longitudinally (Kwapil et al., 2000), and delay discounting. The current study finding that HPS total score was related to steeper discounting rates is consistent with Mason et al.'s study finding steeper discounting related to high scores on the HPS. This growing body of literature suggests that reward discounting may be impaired in non-clinical samples and prior to the development of bipolar disorder.

The finding of Study 2 that more alcohol use was related to steeper discounting rates is consistent with previous research (Bobova et al., 2009; Kollins, 2003; MacKillop et al., 2011; Petry, 2001; Vuchinich & Simpson, 1998; Yi et al., 2010). However, this study provides evidence of differential relationships for different measures of alcohol use (i.e., quantity of alcohol use and binge drinking frequency). Previous studies have generally focused on grouping participants as alcoholics or problem drinkers (e.g., Dom et al., 2006; Mitchell et al., 2005; Vuchinich & Simpson, 1998) rather than examining continuous relationships with measures of alcohol use, such as quantity and frequency. These findings may be particularly important for university student samples who may not yet meet criteria as problem drinkers, but who are exhibiting initial signs of problematic drinking behaviour.

I believe this is the first study to examine the relationship between the UPPS-P and P300 amplitude and latency. Previous studies have largely focused on the BIS-11 as a measure of self-report impulsivity and the current study helps extend that literature and incorporate findings for

the relatively newer measure, the UPPS-P. While there were no correlations between the UPPS-P and P300 amplitude in either the visual or auditory task, there were some correlations between Negative Urgency and longer latencies in the visual paradigm. Latency is not as commonly studied as amplitude with regard to the P300 ERP and its relationship to impulsivity, and the current findings may suggest that latency, as a general measure of processing speed, may be informative when exploring relationships between impulsivity and the P300 ERP.

To my knowledge, this is also the first study to examine the relationship of the HPS to the P300 ERP. Previous research examining the P300 ERP has focused on comparing bipolar individuals to control subjects (Bange & Bathien, 1998; Bestelmeyer et al., 2009; Degabriele & Lagopoulos, 2009; Hall et al., 2007; O'Donnell et al., 2004; Ryu et al., 2010). The current study results suggest that sub-clinical levels of hypomanic personality may be related to P300 latency (a measure of processing speed) at some scalp sites, but amplitude differences may not exist in non-clinical samples.

There is ample evidence to support a relationship between alcohol use and the P300 ERP (Carlson et al., 2007; Chen et al., 2007; Euser et al., 2012; Hesselbrock et al., 2001; Iacono et al., 2003; Polich et al., 1994; Porjesz & Begleiter, 1990). However, the current study suggests that the relationship between P300 and alcohol use is not robust enough to be found in all samples. The findings of this study indicate that mixed sex undergraduates with mostly low to moderate levels of alcohol use and unknown family history of alcohol use disorders may not evidence the relationship between alcohol use and the P300 ERP commonly found in clinical or male only samples. This is an important limitation of this line of research that should be indicated in the literature.

Limitations

Study 1 was limited by a number of factors. The cross-sectional nature of the data for study 1 does not allow any inferences about the causal nature of the relationships being examined. While this limits the interpretation of the mediation analyses, it is important to note, nonetheless, that hypomanic personality no longer (or less strongly) predicted alcohol use when impulsivity was included in the model. This study used an undergraduate sample and the findings, therefore, may not generalize to older or non-student samples. Because the study focused on subclinical levels of hypomanic personality, and not bipolar disorder, the findings may also not generalize to clinical samples of bipolar individuals. Due to the large number of variables and analyses conducted, there may be a greater risk of a Type I error. Alpha for significant results was set at .01 to attempt to reduce the likelihood of a Type I error. In addition, most findings in study 1 are quite robust, falling well below the .001 *p*-value. It is therefore presumed that a Type I error is unlikely and that the results of this study are valid. This study also relied on self-report questionnaire data. Although participants were assured of anonymity and allowed to complete web-based questionnaires outside of the lab, response bias may still be possible.

Many of the limitations of study 2 have been discussed above. This sample may not have enough participants who score at the higher levels of impulsivity, hypomanic personality, and alcohol use to draw out associations previously seen between these variables and delay discounting and EEG measures. In addition, this sample may have too few males, did not consider family history of alcohol use disorders, cannot examine psychiatric comorbidity, and may have a restricted range of intelligence. Additional limitations of this study are the small sample size and large number of variables. While the size of the sample should have had enough

power, based on power calculations, to see the associations found in previous research, it seems that the above mentioned factors may have dampened the findings. Due to the large number of variables being examined and the large number of analyses conducted, there may be a greater risk of a Type I error. In study 1, I was able to use a more stringent alpha level for significant results to counteract this risk. However, in study 2, using a more stringent alpha level would have resulted in very few interpretable results and in the interest of discussing as many findings as possible, alpha was left at .05. Therefore, the likelihood of a Type I error is greater in study 2 and the findings discussed should be considered tenuous.

Directions for Future Research

Future studies would benefit from longitudinal study designs to examine the relationship between hypomanic personality, impulsivity, and alcohol use in a manner that can infer causality. For example, one could measure hypomanic personality and impulsivity in early adolescence and then monitor the start and type of alcohol use through late adolescence and early adulthood. This could help confirm that higher levels of impulsivity lead to future alcohol use, rather than alcohol use leading to higher levels of impulsivity. Previous research along these lines have found hypomanic personality (Kwapil et al., 2000) and impulsivity (Cyders et al., 2009) to each predict alcohol use longitudinally. However, no single study, to my knowledge, has examined the relationships of hypomanic personality and impulsivity to alcohol use, in a longitudinal design.

It would be interesting to examine whether impulsivity continued to be the strongest predictor of alcohol use for individuals with bipolar disorder, who likely would score higher on the HPS than our undergraduate sample. It would also be informative to study whether early impulsivity longitudinally predicts the development of bipolar disorder, over and above early

levels of hypomanic personality. This would support the idea that impulsivity is a common underlying construct that potentially contributes to both alcohol use and bipolar disorders.

Many studies have shown that drinking motives mediate the relationship between personality and alcohol use (Adams, Kaiser, Lynam, Charnigo, & Milich, 2012; Kuntsche, Knibbe, Gmel, & Engels, 2006; Littlefield et al., 2014). For example, Sensation Seeking has been shown to be associated with drinking for enhancement reasons (Adams et al., 2012; Woicik, Stewart, Pihl, & Conrod, 2009), whereas Negative Urgency has been related to drinking for enhancement and coping reasons (Adams et al., 2012). As such, future studies should investigate the role of drinking motives in the hypomania-impulsivity-alcohol use relationship.

As has been done with previous studies, future studies would benefit from using clinical samples or samples representing the full range of scores for impulsivity, hypomanic personality, and alcohol use. Focusing on samples which represent the full spectrum of impairment would better highlight the relationships between these variables and delay discounting and EEG measures. I believe that a sample better representing high and low scorers on hypomanic personality and alcohol use would be more likely to resemble past studies on bipolar and alcoholic participants and mediation models that were not significant or not able to be examined in the current study may be more informative. As also mentioned above, future studies may want to focus on only male participants or on samples with enough male participants to look at gender differences. It would likely be informative to examine gender differences with regard to delay discounting, as I believe this area of research is largely underdeveloped. In addition, future studies examining the P300 ERP should consider implementing measures of family history of alcohol use and comorbidity for psychiatric disorders, as these have been shown to be related to P300 amplitude.

Conclusion

Overall, study 1 showed that the positive relationship between hypomanic personality and alcohol use is mediated by impulsivity. Specifically, Negative Urgency and Sensation Seeking were the UPPS-P scales of interest as mediators. Either or both of Negative Urgency and Sensation Seeking mediated the relationship between hypomanic personality and all measures of alcohol use. In study 2, no mediation models predicting delay discounting were significant and only Positive Urgency was positively correlated with delay discounting rates. In addition, hypomanic personality and higher alcohol use were found to be related to steeper discounting rates. While no mediation models were significant predicting P300 latency and no mediation models were possible predicting P300 amplitude, there were some correlations with EEG measures. Post-hoc analyses suggest that this sample did not have enough males or high enough scores on impulsivity, hypomanic personality, or alcohol use to show associations previously found in clinical samples.

References

- Acheson, A. Vincent, A. S., Sorocco, K. H., & Lovallo, W. R. (2011). Greater discounting of delayed rewards in young adults with family histories of alcohol and drug use disorders: Studies from the Oklahoma family health patterns project. *Alcoholism: Clinical and Experimental Research, 35*, 1607-1613. doi:10.1111/j.1530-0277.2011.01507.x
- Adams, Z. W., Kaiser, A. J., Lynam, D. R., Charnigo, R. J., & Milich, R. (2012). Drinking motives as mediators of the impulsivity-substance use relation: Pathways for negative urgency, lack of premeditation, and sensation seeking. *Addictive Behaviors, 37*, 848-855. doi:10.1016/j.addbeh.2012.03.016
- Adlaf, E. M., Begin, P., & Sawka, E. (Eds.). (2005). *Canadian Addiction Survey (CAS): A national survey of Canadians' use of alcohol and other drugs: Prevalence of use and related harms: Detailed report*. Ottawa: Canadian Centre on Substance Abuse.
- Agid, O., Shapira, B., Zislin, J., Ritsner, M., Hanin, B., Murad, H., ...Lerer, B. (1999). Environmental and vulnerability to major psychiatric illness: A case control study of early parental loss in major depression, bipolar disorder and schizophrenia. *Molecular Psychiatry, 4*, 163-172.
- Ahn, W.-Y., Rass, O., Fridberg, D. J., Bishara, A. J., Forsyth, J. K., Breier, A.,...O'Donnell, B. F. (2011). Temporal discounting of rewards in patients with bipolar disorder and schizophrenia. *Journal of Abnormal Psychology, 120* (4), 911-921. doi:10.1037/a0023333
- Aluja, A., García, Ó., & García, L. F. (2003). Relationships among extraversion, openness to experience, and sensation seeking. *Personality and Individual Differences, 35*, 671-680.

- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*. Arlington, VA: Author.
- Andersen, A., Due, P., Holstein, B. E., & Iversen, L. (2003). Tracking drinking behavior from age 15-19 years. *Addiction, 98*, 1505-1511.
- Andrucci, G. L., Archer, R. P., Pancoast, D. L., & Gordon, R. A. (1989). The relationship of MMPI and sensation seeking scales to adolescent drug use. *Journal of Personality Assessment, 53*, 253-266
- Anokhin, A. P., Golosheykin, S., Grant, J. D., & Heath, A. C. (2011). Heritability of delay discounting in adolescence: A longitudinal twin study. *Behavior Genetics, 41*, 175-183. doi:10.1007/s10519-010-9384-7
- Bange, F. & Banthien, N. (1998). Visual cognitive dysfunction in depression: An event-related potential study. *Electroencephalography and Clinical Neurophysiology, 108*, 472-481.
- Barratt, E. S., Stanford, M. S., Kent, T. A., & Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. *Biological Psychiatry, 41*, 1045-1061.
- Bauer, L. O., O'Connor, S., & Hesselbrock, V. M. (1994). Frontal P300 decrements in antisocial personality disorder. *Alcoholism: Clinical and Experimental Research, 18*, 1300-1305.
- Begleiter, H., Porjesz, B., Bihari, B., & Kissin, B. (1984). Event-related potentials in boys at risk for alcoholism. *Science, 225 (4669)*, 1493-1496.
- Bestelmeyer, E. G., Phillips, L. H., Crombie, C., Benson, P., & St.Clair, D. (2009). P300 as a possible endophenotype for schizophrenia and bipolar disorder: Evidence from twin and patient studies. *Psychiatry Research, 169*, 212-219. doi:10.1016/j.psychres.2008.06.035

- Biggins, C. A., MacKay, S., Poole, N., & Fein, G. (1995). Delayed P3A in abstinent elderly male chronic alcoholics. *Alcoholism: Clinical and Experimental Research, 19*, 1032-1042.
- Bobova, L., Finn, P. R., Rickert, M. E., & Lucas, J. (2009). Disinhibitory psychopathology and delay discounting in alcohol dependence: Personality and cognitive correlates. *Experimental and Clinical Psychopharmacology, 17* (1), 51-61. doi:10.1037/a0014503
- Bohman, M., Cloninger, R., Sagvardsson, S., & Knorrning, A.-L. (1987). The genetics of alcoholisms and related disorders. *Journal of Psychiatric Research, 21* (4), 447-452.
- Bramon, E., Rabe-Hesketh, S., Sham, P., Murray, R. M., & Frangou, S. (2004). Meta-analysis of the P300 and P50 waveforms in schizophrenia. *Schizophrenia Research, 70*, 315-329. doi:10.1016/j.schres.2004.01.004
- Broos, N., Schmaal, L., Wiskerke, J., Kostelijk, L., Lam, T., Stoop, N., ... Goudriaan, A. E. (2012). The relationship between impulsive choice and impulsive action: A cross-species translational study. *PLoS ONE, 7*(5), e36781. doi: 10.1371/journal.pone.0036781
- Bruder, G. E., Kroppmann, C. J., Kayser, J., Stewart, J. W., McGrath, P. J., & Tenke, C. E. (2009). Reduced brain responses to novel sounds in depression: P3 findings in a novelty oddball task. *Psychiatry Research, 170*, 218-223. doi:10.1016/j.psychres.2008.10.023
- Buss, A. H., & Plomin, R. (1975). *A temperament theory of personality development*. New York: John Wiley & Sons.
- Cadaveira, F., Grau, C., Roso, M., & Sanchez-Turet, M. (1991). Multimodality exploration of event-related potentials in chronic alcoholics. *Alcoholism: Clinical and Experimental Research, 15*, 607-611.
- Carlson, S. R., & Iacono, W. G. (2006). Heritability of P300 amplitude development from adolescence to adulthood. *Psychophysiology, 43*, 470-480.

- Carlson, S. R., Iacono, W. G., & McGue, M. (2002). P300 amplitude in adolescent twins discordant and concordant for alcohol use disorders. *Biological Psychology*, *61*, 203-227.
- Carlson, S. R., Johnson, S. C., & Jacobs, P. C. (2010). Disinhibited characteristics and binge drinking among university student drinkers. *Addictive Behaviors*, *35*, 242-251.
doi:10.1016/j.addbeh.2009.10.020
- Carlson, S. R., Katsanis, J., Iacono, W. G., & Mertz, A. K. (1999). Substance dependence and externalizing psychopathology in adolescent boys with small, average, or large P300 event-related potential amplitude. *Psychophysiology*, *36*, 583-590.
- Carlson, S. R., McLarnon, M. E., & Iacono, W. G. (2007) P300 amplitude, externalizing psychopathology, and earlier- versus later-onset substance-use disorder. *Journals of Abnormal Psychology*, *116* (3), 565-577. doi:10.1037/0021-843X.116.3.565
- Carmirol, N., Peralta, J. M., Almasy, L., Contreras, J., Pacheco, A., Escamilla, M. A.,...Glahn, D. C. (2014). Shared genetic factors influence risk for bipolar disorder and alcohol use disorders. *European Psychiatry*, *29*, 282-287. doi:10.1016/j.eurpsy.2013.10.001
- Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry*, *53*, 1033-1039.
- Caspi, A. & Silva, P. A. (1995). Temperamental qualities at age three predict personality traits in young adulthood: Longitudinal evidence from a birth cohort. *Child Development*, *66*, 486-498.
- Cassidy, F., Ahearn, E. P., Carroll, B. J. (2001). Substance abuse in bipolar disorder. *Bipolar Disorder*, *3*, 181-188.

- Chen, A. C. H., Porjesz, B., Rangaswamy, M., Kamarajan, C., Tang, Y., Jones, K.,...Begleiter, H. (2007). Reduced frontal lobe activity in subjects with high impulsivity and alcoholism. *Alcoholism: Clinical and Experimental Research, 31*, 156-165. doi:10.1111/j.1530-0277.2006.00277.x
- Cherek, D. R., Moeller, F. G., Dougherty, D. M., & Rhoades, H. (1997). Studies of violent and nonviolent male parolees: II. Laboratory and psychometric measurements of impulsivity. *Biological Psychiatry, 41*, 523-529.
- Clark, L., Iversen, S. D., & Goodwin, G. M. (2002). Sustained attention deficit in bipolar disorder. *British Journal of Psychiatry, 180*, 313-319.
- Cloninger, C. R., Bohman, M., & Sigvardsson, S. (1981). Inheritance of alcohol abuse. *Archives of General Psychiatry, 38*, 861-868.
- Cloninger, C. R., Przybeck, T. R., & Svrakic, D. M. (1991). The Tridimensional Personality Questionnaire: US normative data. *Psychological Reports, 69*, 1047-1057.
- Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. *Alcoholism: clinical and Experimental Research, 12*, 494-505.
- Cohen, H. L., Wang, W., Porjesz, B., & Begleiter, H. (1995). Auditory P300 in young alcoholics: Regional response characteristics. *Alcoholism: Clinical and Experimental Research, 19*, 469-475.
- Conrad, P. J., Petersen, J. B., & Pihl, R. O. (1997). Disinhibited personality and sensitivity to alcohol reinforcement: Independent correlates of drinking behavior in sons of alcoholics. *Alcoholism: Clinical and Experimental Research, 21*, 1320-1332.

- Conrod, P. J., Castellanos-Ryan, N., & Mackie, C. (2011). Long-term effects of a personality-targeted intervention to reduce alcohol use in adolescents. *Journal of Consulting and Clinical Psychology, 79*, 296-306. doi:10.1037/a0022997
- 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*, 990-1005.
- Cooper, M. L., Russell, M., Skinner, J. B., Frone, M. R., & Mudar, P. (1992). Stress and alcohol use: Moderating effects of gender, coping, and alcohol expectancies. *Journal of Abnormal Psychology, 101*, 139-152.
- Coskunpinar, A., Dir, A. L., & Cyders, M. A. (2013). Multidimensionality in impulsivity and alcohol use: A meta-analysis using the UPPS model of impulsivity. *Alcoholism: Clinical and Experimental Research, 37*, 1441-1450. doi:10.1111/acer.12131
- Costa, L., Bauer, L., Kuperman, S., Porjesz, B., O'Connor, S., Hesselbrock, V.,...Begleiter, H. (2000). Frontal P300 decrements, alcohol dependence, and antisocial personality disorder. *Biological Psychiatry, 47*, 1064-1071.
- Costa, P. T. Jr, & McCrae, R. R. (1992). *Revised NEO personality inventory manual*. Odessa, FL: Psychological Assessment Resources.
- Crean, J. P., de Wit, H., & Richards, J. B. (2000). Reward discounting as a measure of impulsive behaviour in a psychiatric outpatient population. *Experimental and Clinical Psychopharmacology, 8* (2), 155-162. doi:10.1037//1064-1297.8.2.155
- Crego, A., Cadaveira, F., Parada, M., Corral, M., Caamano-Isorna, F., & Holguín, S. R. (2012). Increased amplitude of P3 event-related potential in young binge drinkers. *Alcohol, 46*, 415-425. doi:10.1016/j.alcohol.2011.10.002

- Cuellar, A. K., Johnson, S. L., & Winters, R. (2005). Distinctions between bipolar and unipolar depression. *Clinical Psychology Review, 25*, 307-339. doi: 10.1016/j.cpr.2004.12.002
- Curcio, A. L. & George, A. M. (2011). Selected impulsivity facets with alcohol use/problems: The mediating role of drinking motives. *Addictive Behaviors, 36*, 959-964.
doi:10.1016/j.addbeh.2011.05.007
- Cuzen, N. L., Andrew, C., Thomas, K. G. F., Stein, D. J., & Fein, G. (2013). Absence of P300 reduction in South African treatment-naïve adolescents with alcohol dependence. *Alcoholism: Clinical and Experimental Research, 37*, 40-48. doi:10.1111/j.1530-0277.2012.01837.x
- Cyders, M. A. (2013). Impulsivity and the sexes: Measurement and structural invariance of the UPPS-P Impulsive Behavior Scale. *Assessment, 20*, 86-97.
doi:10.1177/1073191111428762
- Cyders, M. A., Flory, K., Rainer, S., & Smith, G. T. (2009). The role of personality dispositions to risky behaviour predicting first-year college drinking. *Addiction, 104*, 193-202.
doi:10.1111/j.1360-0443.2008.02434.x
- Cyders, M. A. & Smith, G. T. (2007). Mood-based rash action and its components: Positive and negative urgency. *Personality and Individual Differences, 43*, 839-850.
doi:10.1016/j.paid.2007.02.008
- Cyders, M. A., Smith, G. T., Spillane, N. S., Fischer, S., Annus, A. M., & Peterson, C. (2007). Integration of impulsivity and positive mood to predict risky behavior: Development and validation of a measure of positive urgency. *Psychological Assessment, 19*, 107-118.
- Dalley, J. W., Everitt, B. J., & Robbins, T. W. (2011). Impulsivity, compulsivity, and top-down cognitive control. *Neuron, 69*, 680-694. doi: 10.1016/j.neuron.2011.01.020

- Degabriele, R. & Lagopoulos, J. (2009). A review of EEG and ERP studies in bipolar disorder. *Acta Neuropsychiatrica*, *21*, 58-66. doi:10.1111/j.1601-5215.2009.00359.x
- De Pascalis, V., Strippoli, E., Riccardi, P., & Vergari, F. (2004). Personality, event-related potential (ERP) and heart rate (HR) in emotional word processing. *Personality and Individual Differences*, *36*, 873-891. doi:10.1016/S0191-8869(03)00159-4
- DeWit, D. J., Adlaf, E. M., Offord, D. R., & Ogborne, A. C. (2000). Age at first alcohol use: A risk factor for the development of alcohol disorders. *American Journal of Psychiatry*, *157*, 745-750.
- de Wit, H., Flory, J. D., Acheson, A., McCloskey, M., & Manuck, S. B. (2007). IQ and nonplanning impulsivity are independently associated with delay discounting in middle-aged adults. *Personality and Individual Differences*, *42*, 111-121.
doi:10.1016/j.paid.2006.06.026
- Di Florio, A., Craddock, N., & van den Bree, M. (2014). Alcohol misuse in bipolar disorder. A systematic review and meta-analysis of comorbidity rates. *European Psychiatry*, *29*, 117-124. doi:10.1016/j.eurpsy.2013.07.004
- Dick, D. M., Smith, G., Olausson, P., Mitchell, S., Leeman, R. F., O'Malley, S. S., & Sher, K. (2010). Understanding the construct of impulsivity and its relationship to alcohol use disorders. *Addictive Behaviors*, *15* (2), 217-226. doi:10.1111/j.1369-1600.2009.00190.x
- Dickman, S. J. (1990). Functional and dysfunctional impulsivity: personality and cognitive correlates. *Journal of Personality and Social Psychology*, *58*, 95-102.
- Dimeff, L. A. & Linehan, M. M. (2008). Dialectical behavior therapy for substance abusers. *Addiction Science & Clinical Practice*, *4*, 39-47.

- Do, E. K. & Mezuk, B. (2013). Comorbidity between hypomania and substance use disorders. *Journal of Affective Disorders, 150*, 974-980. doi:10.1016/j.jad.2013.05.023
- Doege, K., Bates, A. T., White, T. P., Das, D., Boks, M. P., & Liddle, P. F. (2009). Reduced event-related low frequency EEG activity in schizophrenia during an auditory oddball task. *Psychophysiology, 46*, 566-577. doi:10.1111/j.1469-8986.2009.00785.x
- Dom, G., D'haene, P., Hulstijn, W., & Sabbe, B. (2006). Impulsivity in abstinent early- and late-onset alcoholics: Differences in self-report measures and a discounting task. *Addiction, 101*, 50-59. doi:10.1111/j.1360-0443.2005.01270.x
- Dom, G., De Wilde, B., Hulstijn, W., & Sabbe, B. (2007). Dimensions of impulsive behaviour in abstinent alcoholics. *Personality and Individual Differences, 42*, 465-476. doi:10.1016/j.paid.2006.08.007
- Donchin, E. (1981) Surprise!...Surprise? *Psychophysiology, 18* (5), 493-513.
- Donohew, L., Zimmerman, R., Cupp, P. S., Novak, S., Colon, S., & Abell, R. (2000). Sensation seeking, impulsive decision-making, and risky sex: Implications for risk-taking and design of interventions. *Personality and Individual Differences, 28*, 1079-1091.
- Eckblad, M., & Chapman, L. J. (1986). Development and validation of a scale for Hypomanic Personality. *Journal of Abnormal Psychology, 95* (3), 214-222.
- Euser, A. S., Arends, L. R., Evans, B. E., Greaves-Lord, K., Huizink, A. C., & Franken, I. H. A. (2012). The P300 event-related brain potential as a neurobiological endophenotype for substance use disorders: A meta-analytic investigation. *Neuroscience and Biobehavioral Reviews, 36*, 572-603. doi:10.1016/j.neubiorev.2011.09.002s

- Eysenck, S. B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*, 613-619.
- Eysenck, S. & Zuckerman, M. (1978). The relationship between sensation-seeking and Eysenck's dimensions of personality. *British Journal of Psychology, 69*, 483-487.
- Finn, P. R., Bobava, L., Wehner, E. Fargo, S., & Rickert, M. E. (2005). Alcohol expectancies, conduct disorder and early-onset alcoholism: Negative alcohol expectancies are associated with less drinking in non-impulsive versus impulsive subjects. *Addiction, 100*, 953-962.
- Finn, P. R. & Hall, J. (2004). Cognitive ability and risk for alcoholism: Short-term memory capacity and intelligence moderate personality risk for alcohol problems. *Journal of Abnormal Psychology, 113*, 569-581.
- Fleming, M. F. & Barry, K. L. (1989). A study examining the psychometric properties of the SMAST-13. *Journal of Substance Abuse, 1*, 173 – 182.
- Flory, J. D., Harvey, P. D., Mitropoulou, V., New, A. S., Silverman, J. M., Siever, L. J., & Manuck, S. B. (2006). Dispositional impulsivity in normal and abnormal samples. *Journal of Psychiatric Research, 40*, 438–477. doi:10.1016/j.jpsychires.2006.01.008
- Ford, J. M., White, P., Lim, K. O., & Pfefferbaum, A. (1994). Schizophrenics have fewer and smaller P300s: A single-trial analysis. *Biological Psychiatry, 35*, 96-103.
- Gangadhar, B. N., Ancy, J., Janakiramaiah, N., & Umaphy, C. (1993). P300 amplitude in non-bipolar, melancholic depression. *Journal of Affective Disorders, 28*, 57-60.

- Gilmore, C. S., Malone, S. M., & Iacono, W. G. (2010). Brain electrophysiological endophenotypes for externalizing psychopathology: A multivariate approach. *Behavioral Genetics, 40*, 186-200. doi:10.1007/s10519-010-9343-3
- Goldberg, J. F. (2001). Bipolar disorder with comorbid substance abuse: Diagnosis, prognosis, and treatment. *Journal of Psychiatric Practice, March*, 109-122.
- Granö, N., Virtanen, M., Vahtera, J., Elovainio, M., & Kivimäki, M. (2004) Impulsivity as a predictor of smoking and alcohol consumption. *Personality and Individual Differences, 37*, 1693-1700.
- Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W.,...Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the national epidemiology survey on alcohol and related conditions. *Archives of General Psychiatry, 61*, 807-816.
- Grant, B. F., Stinson, F. S., Hasin, D. S., Dawson, D. A., Chou, S. P., Ruan, W. J., & Huang, B. (2005). Prevalence, correlates, and comorbidity of bipolar I disorder and axis I and II disorders: Results from the national epidemiology Survey on alcohol and related conditions. *Journal of Clinical Psychiatry, 66*, 1205-1215.
- Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology, 55(4)*, 468-484.
- Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5*, 33-36. doi:10.1111/j.1467-9280.1994.tb00610.x
- Green, L. & Myerson, J. (2010). Experimental and correlational analyses of delay and probability discounting. In: G. J. Madden & W. K. Bickel, (Eds.), *Impulsivity: The*

- Behavioral and Neurological Science of Discounting. American Psychological Association, Washington, DC, pp. 67–92.
- Guy, S.M., Smith, G. M., & Bentler, P. M. (1994). Consequences of adolescent drug use and personality factors on adult drug use. *Journal of Drug Education, 24*, 109-132.
- Hall, M.-H., Rijdsdijk, F., Kalidindi, S., Schulze, K., Kravariti, E., Kane, F.,...Murray, R. M. (2007). Genetic overlap between bipolar illness and event-related potentials. *Psychological Medicine, 37*, 667-678. doi:10.1017/S003329170600972X
- Harmon-Jones, E., Barratt, E. S., & Wigg, C. (1997). Impulsiveness, aggression, reading, and the P300 of the event-related potential. *Personality and Individual Differences, 22* (4), 439-445.
- Haugen Light, W. J. (1986). *Neurobiology of Alcohol Abuse*. Springfield, IL: Charles C. Thomas.
- Heath, A. C., Bucholz, K. K., Madden, P. A. F., Dinwiddie, S. H., Slutske, W. S., Bierut, L. J.,...Martin, N. G. (1997). Genetic and environmental contributions to alcohol dependence risk in a national twin sample: consistency of findings in men and women. *Psychological Medicine, 27*, 1381-1396.
- Hegerl, U., Gallinat, J., & Juckel, G. (2001). Event-related potentials: Do they reflect central serotonergic neurotransmission and do they predict clinical response to serotonin agonists? *Journal of Affective Disorders, 62*, 93-100.
- Hegerl, U. & Juckel, G. (1993). Intensity dependence of auditory evoked potentials as an indicator of central serotonergic neurotransmission: A new hypothesis. *Biological Psychiatry, 33*, 173-187.

- Henderson, M. J., Goldman, M. S., Coovert, M. D., & Carnevalla, N. (1994). Covariance structure models of expectancy. *Journal of Studies on Alcohol*, *55*, 315-326.
- Hensch, T., Herold, U., & Brocke, B. (2007). An electrophysiological endophenotype of hypomanic and hyperthymic personality. *Journal of Affective Disorders*, *101*, 13-26.
doi:10.1016/j.jad.2006.11.018
- Hesselbrock, V., Begleiter, H., Porjesz, B., O'Connor, S., & Bauer, L. (2001). P300 event-related potential amplitude as an endophenotype of alcoholism – Evidence from the collaborative study on the genetics of alcoholism. *Journal of Biomedical Science*, *8*, 77-82.
- Hicks, B. M., Bernat, E., Malone, S. M., Iacono, W. G., Patrick, C., Krueger, R. F., & McGue, M. (2007). Genes mediate the association between P3 amplitude and externalizing disorders. *Psychophysiology*, *44*(1), 98-105.
- Hicks, B. M., Krueger, R. F., Iacono, W. G., McGue, M., & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders. *Archives of General Psychiatry*, *61*, 922-928.
- Hill, S. Y., Locke, J., & Steinhauer, S. R. (1999). Absence of visual and auditory P300 reduction in nondepressed male and female alcoholics. *Biological Psychiatry*, *46*, 982-989.
- Hill, S. Y., Shen, S., Lowers, L., & Locke, J. (2000). Factors predicting the onset of adolescent drinking in families at high risk for developing alcoholism. *Biological Psychiatry*, *48*, 265-275.
- Hill, S. Y., Steinhauer, S., Locke, J. (1995). Event related potentials in alcoholic men, their high-risk relatives, and low-risk male controls. *Alcoholism: Clinical and Experimental Research*, *19*, 567-576.
- Hill, S. Y., Steinhauer, S. R., Locke-Wellman, J., & Ulrich, R. (2009). Childhood risk factors for young adult substance dependence outcome in offspring from multiplex alcohol

- dependence families: A prospective study. *Biological Psychiatry*, 66, 750-757.
doi:10.1016/j.biopsych.2009.05.030
- Hilton, M. E. (1987). Drinking patterns and drinking problems in 1984: Results from a general population survey. *Alcoholism: Clinical and Experimental Research*, 11, 167-175. doi: 10.1111/j.1530-0277.1987.tb01283
- Himani, A., Tandon, O. P., & Bhatia, M. S. (1999). A study of P300 event related evoked potential in the patients of major depression. *Indian Journal of Physiological Pharmacology*, 43, 367-372.
- Hittner, J. B. & Swickert, R. (2006). Sensation seeking and alcohol use: A meta-analytic review. *Addictive Behaviors*, 31, 1383-1401.
- Holmes, M. K., Bearden, C. E., Barguil, M., Fonseca, M., Monkul, E. S., Nery, F. G., ...Minta, J. (2009). Conceptualizing impulsivity and risk taking in bipolar disorder: Importance of history of alcohol use. *Bipolar Disorder*, 11, 33-40. doi: 10.1111/j.1399-5618.2008.00657.x
- Iacono, W. G., Carlson, S. R., Malone, S. M., & McGue, M. (2002). P3 event-related potential amplitude and the risk for disinhibitory disorders in adolescent boys. *Archives of General Psychiatry*, 59, 750-757.
- Iacono, W. G., Malone, S. M. & McGue, M. (2003). Substance use disorders, externalizing psychopathology, and P300 event-related potential amplitude. *International Journal of Psychophysiology*, 48, 147-178. doi:10.1016/S0167-8760(03)00052-7
- Iacono, W. G., McGue, M., & Krueger, R. F. (2006). Minnesota center for twin and family research. *Twin Research and Human Genetics*, 9 (6), 978-984.

- Jackson, D. N. (1984). *Personality research from manual*. Goshen, NY: Research Psychologists Press.
- Jeon, Y.-W. & Polich, J. (2003). Meta-analysis of P300 and schizophrenia: Patients, paradigms, and practical implications. *Psychophysiology*, *40*, 684-701.
- Johnson, R. (1993). On the neural generators of the P300 component of the event-related potential. *Psychophysiology*, *30*, 90-97.
- Johnson, M. W. & Bickel, W. K. (2002). Within-subject comparison of real and hypothetical money rewards in delay discounting. *Journal of the Experimental Analysis of Behavior*, *77*, 129-146.
- Johnson, M. W. & Bickel, W. K. (2008). An algorithm for identifying nonsystematic delay-discounting data. *Experimental and Clinical Psychopharmacology*, *16*, 264-274.
doi:10.1037/1064-1297.16.3.264
- Johnson, S. L., Carver, C. S., Mulé, S., & Joorman, J. (2012). Impulsivity and risk for mania: Towards a greater specificity. *Psychology and Psychotherapy: Theory, Research and Practice*. doi:10.1111/j.2044-8341.2012.02078.x
- Jones, K. A., Chryssanthakis, A., & Groom, M. J. (2014). Impulsivity and drinking motives predict problem behaviours relating to alcohol use in University students. *Addictive Behaviors*, *39*, 289-296. doi:10.1016/j.addbeh.2013.10.024
- Jones, M. C. (1968). Personality correlates and antecedents of drinking patterns in adult males. *Journal of Consulting and Clinical Psychology*, *32*, 2-12.
- Jones, M. C. (1971). Personality antecedents and correlates of drinking patterns in women. *Journal of Consulting and Clinical Psychology*, *36*, 61-69.

- Justus, A. N., Finn, P. R., & Steinmetz, J. E. (2001). P300, disinhibited personality, and early-onset alcohol problems. *Alcoholism: Clinical and Experimental Research, 25* (10), 1457-1466.
- Katsanis, J., Iacono, W. G., McGue, M. K., & Carlson, S. R. (1997). P300 event-related potential heritability in monozygotic and dizygotic twins. *Psychophysiology, 34*, 47-58.
- Keenan, J. P., Freeman, P. R., & Harrell, R. (1997). The effects of family history, sobriety length, and drinking history in younger alcoholics on P300 auditory-evoked potentials. *Alcohol & Alcoholism, 32*, 233-239.
- Keyes, K. M., Hatzenbuehler, M. L., & Hasin, D. S. (2011). Stressful like experiences, alcohol consumption, and alcohol use disorders: The epidemiologic evidence for four main types of stressors. *Psychopharmacology, 218*, 1-17. doi: 10.1007/s00213-011-2236-1
- Kirby, K. N. (2009). One-year temporal stability of delay-discount rates. *Psychonomic Bulletin & Review, 16*(3), 457-462. doi:10.3758/PBR.16.3.457
- Kirby, K. N., & Petry, N. M. (2004). Heroin and cocaine abusers have higher discount rates for delayed rewards than alcoholics or non-drug-using controls. *Addiction, 99*, 461-471. doi:10.1111/j.1360-0443.2004.00669.x
- Klein, D. N., Lewinsohn, P. M., & Seeley, J. R. (1996). Hypomanic personality traits in a community sample of adolescents. *Journal of Affective Disorders, 38*, 135-143. doi:10.1016/0165-0327(96)00005-5
- Kollins, S. H. (2003). Delay discounting is associated with substance use in college students. *Addictive Behaviors, 28*, 1167-1173. doi:10.1016/S0306-4603(02)00220-4
- Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56*, 921-926. doi:10.1001/archpsyc.56.10.921

- Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiological connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*, 411-424.
- Krumm-Merabet, C. & Meyer, T. D. (2005). Leisure activities, alcohol, and nicotine consumption in people with a hypomanic/hyperthymic temperament. *Personality and Individual Differences, 38*, 701-712. doi:10.1016/j.paid.2004.05.024
- Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2006). Who drinks and why? A review of socio-demographic, personality, and contextual issues behind the drinking motives in young people. *Addictive Behaviors, 31*, 1844-1857. doi:10.1016/j.addbeh.2005.12.028
- Kwapil, T. R., Miller, M. B., Zinser, M. C., Chapman, L. J., Chapman, J., & Eckblad, M. (2000). A longitudinal study of high scorers on the Hypomanic Personality Scale. *Journal of Abnormal Psychology, 109* (2), 222-226.
- La Grange, L., Jones, T. D., Erb, L., & Reyes, E. (1995). Alcohol consumption: Biochemical and personality correlates in a college student population. *Addictive Behaviors, 20*, 93-103.
- LaBrie, J. W., Kenney, S. R., Napper, L. E., & Miller, K. (2014). Impulsivity and alcohol-related risk among college students: Examining urgency, sensation seeking and the moderating influence of beliefs about alcohol's role in the college experience. *Addictive Behaviors, 39*, 159-164. doi:10.1016/j.addbeh.2013.09.018
- Lewis, M., Scott, J., & Frangou, S. (2009). Impulsivity, personality, and bipolar disorder. *European Psychiatry, 24*, 464-469. doi:10.1016/j.eurpsy.2009.03.004
- Linehan, M. M., Dimeff, L. A., Reynolds, S. K., Comtois, K. A., Welch, S. S., Heagerty, P., & Kivlahan, D. R. (2002). Dialectical behavior therapy versus comprehensive validation

- therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. *Drug and Alcohol Dependence*, 67, 13-26.
- Linehan, M. M., Schmidt, H., Dimeff, L. A., Craft, J. C., Kanter, J., & Comtois, K. A. (1999). Dialectical behavior therapy for patients with borderline personality disorder and drug-dependence. *The American Journal on Addictions*, 8, 279-292.
- Littlefield, A. K., Stevens, A. K., & Sher, K. J. (2014). Impulsivity and alcohol involvement: Multiple, distinct constructs and processes. *Current Addiction Reports*, 1, 33-40.
doi:10.1007/s40429-013-0004-5
- Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales*, (2nd ed.). Sydney, Australia: Psychology Foundation of Australia.
- MacKillop, J., Amlung, M. T., Few, L. R., Ray, L. A., Sweet, L. H., & Munafò, M. R. (2011). Delayed reward discounting and addictive behavior: A meta-analysis. *Psychopharmacology*, 216, 305-321. doi:10.1007/s00213-011-2229-0
- Madden, G. J., Begotka, A. M., Raiff, B. R., & Kastern, L. L. (2003). Delay discounting of real and hypothetical rewards. *Experimental and Clinical Psychopharmacology*, 11, 139 –145.
doi:10.1037/1064-1297.11.2.139
- Madden, G. J. & Johnson, P. S. (2010). A delay-discounting primer. In: Madden, G.J., Bickel, W.K. (Eds.), *Impulsivity: The Behavioral and Neurological Science of Discounting*. American Psychological Association, Washington, DC, pp. 11–37.
- Madden, G. J., Raiff, B. R., Lagorio, C. H., Begotka, A. M., Mueller, A. M., Hehli, D. J., & Wegener, A. A. (2004). Delay discounting of potentially real and hypothetical rewards: II. Between- and within-subject comparisons. *Experimental and clinical Psychopharmacology*, 12 (4), 251-261. doi: 10.1037/1064-1297.12.4.251

- Magid, V. & Colder, C. R. (2007). The UPPS Impulsive Behavior Scale: Factor structure and associations with college drinking. *Personality and Individual Differences, 43*, 1927-1937. doi:10.1016/j.paid.2007.06.013
- Mahmood, T. & Silverstone, T. (2001). Serotonin and bipolar disorder. *Journal of Affective Disorders, 66*, 1-11.
- Martin, L. E. & Potts, G. F. (2004). Reward sensitivity in impulsivity. *Cognitive Neuroscience and Neuropsychology, 15 (9)*, 1519-1522.
- Maser, J. D., Akiskal, H. S., Schettler, P., Scheftner, W., Mueller, T., Endicott, J.,...Clayton, P. (2002). Can temperament identify affectively ill patients who engage in lethal or near-lethal suicidal behaviour? A 14-year prospective study. *Suicide and Life-Threatening Behavior, 32 (1)*, 10-32.
- Mason, L., El-Derey, W., & Bentall, R. (2011). Reward dysfunction in mania: Neural correlates of risk and impulsivity in individuals vulnerable to bipolar disorder. *International Clinical Psychopharmacology, 26*, e46. doi:10.1097/01.yic.0000405710.14232.bf
- Mason, L., O'Sullivan, N., Blackburn, M., Bentall, R., & El-Derey, W. (2012). I want it now! Neural correlates of hypersensitivity to immediate reward in hypomania. *Biological Psychiatry, 71*, 530-537. doi:10.1016/j.biopsych.2011.10.008
- Mazur, J. E. (1987). An adjusting procedure for studying delayed reinforcement. In M. L. Commons, J. E. Mazur, J. A. Nevin, & H. Rachlin (Eds.), *Quantitative analyses of behavior: Vol. 5. The effect of delay and of intervening events on reinforcement value* (pp. 55-73). Hillsdale, NJ: Erlbaum.
- McCrae, R. R., & Costa, P. T. Jr (1990). *Personality in adulthood*. New York: Guilford.

- McGue, M. (1999). Behavioral genetic models of alcoholism and drinking. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism*. New York: Guilford Press.
- McGuffin, P., Rijdsdijk, F., Andrew, M., Sham, P., Zatz, R., & Cardno, A. (2003). The heritability of bipolar affective disorder and the genetic relationship to unipolar depression. *Archives of General Psychiatry*, *60*, 497-502.
- Merikangas, K. R., Akiskal, H. S., Angst, J., Greenberg, P. E., Hirschfeld, R. M. A., Petukhova, M.,...Kessler, R. C. (2007). Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey replication. *Archives of General Psychiatry*, *64* (5), 543-552.
- Meyer, B., Rahman, R., & Shepherd, R. (2007). Hypomanic personality features and addictive tendencies. *Personality and Individual Differences*, *42*, 801–810.
doi:10.1016/j.paid.2006.08.024
- Meyer, T. D. (2002). The Hypomanic Personality Scale, the Big Five, and their relationship to depression and mania. *Personality and Individual Differences*, *32*, 649–660.
doi:10.1016/S0191-8869(01)00067-8
- Meyer, T. D. & Wolkenstein, L. (2010). Current alcohol use and risk for hypomania in male students: Generally more or more bingeing? *Comprehensive Psychiatry*, *51*, 171-176.
doi:10.1016/j.comppsy.2009.05.002
- Miller, J., Flory, K., Lynam, D., and Leukefeld, C. (2003). A test of the four-factor model of impulsivity-related traits. *Personality and Individual Differences*, *34*, 1403-1418.

- Mitchell, J. M., Fields, H. L., D'Esposito, M., & Boettiger, C. A. (2005). Impulsive responding in alcoholics. *Alcoholism: Clinical and Experimental Research*, *29* (12), 2158-2169.
doi:10.1097/01.alc.0000191755.63639.4a
- Mitchell, S. H. (2011). The genetic basis of delay discounting and its genetic relationship to alcohol dependence. *Behavioral Processes*, *87*, 10 – 17.
doi:10.1016/j.beproc.2011.02.008
- 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*, 1783-1793.
- Moeller, F. G., Barratt, E. S., Fischer, C. J., Dougherty, D. M., Reilly, E. L., Mathias, C. W., & Swann, A. C. (2004). P300 event-related potential amplitude and impulsivity in cocaine-dependent subjects. *Neuropsychobiology*, *50*, 167-173. doi:10.1159/000079110
- Myerson, J., Green, L., Hanson, J. S., Holt, D. D., & Estle, S. J. (2003). Discounting delayed and probabilistic rewards: Processes and traits. *Journal of Economic Psychology*, *24*, 619-635. doi:10.1016/S0167-4870(03)00005-9
- Najt, P., Perez, J., Sanches, M., Peluso, M. A.M., Glahn, D., & Soares, J. C. (2007). Impulsivity in bipolar disorder. *European Neuropsychopharmacology*, *17*, 313-320.
doi:10.1016/j.euroneuro.2006.10.002
- National Institute of Health. (2003). *Task force on recommended alcohol questions: National council on alcohol abuse and alcoholism recommended sets of alcohol consumption questions*. Retrieved September 6, 2007, from NIH Website:
<http://www.niaaa.nih.gov/Resources/ResearchResources/TaskForce.htm>

- O'Donnell, B. F., Vohs, J. L., Hetrick, W. P., Carroll, C. A., & Shekhar, A. (2004). Auditory event-related potential abnormalities in bipolar disorder and schizophrenia. *International Journal of Psychophysiology*, *53*, 45-55. doi:10.1016/j.ijpsycho.2004.02.001
- Palmgreen, P., Donohew, L., Lorch, E. P., Hoyle, R. H., & Stephenson, M. T. (2001). Television campaigns and adolescent marijuana use: Tests of sensation seeking targeting. *American Journal of Public Health*, *91*, 292-296.
- Palmgreen, P., Donohew, L., Lorch, E. P., Rogus, M., Helm, D., & Grant, N. (1991). Sensation seeking, message sensation value, and drug use as mediators of PSA effectiveness. *Health Communication*, *3*, 217-227.
- Palmgreen, P., Lorch, E. P., Stephenson, M. T., Hoyle, R. H., & Donohew, L. (2007). Effects of the office of national drug control policy's marijuana initiative campaign on high-sensation-seeking adolescents. *American Journal of Public Health*, *97*, 1644-1649.
- Parsons, O. A., Sinha, R., & Williams, H. L. (1990). Relationships between neuropsychological test performance and event-related potentials in alcoholic and nonalcoholic samples. *Alcoholism: Clinical and Experimental Research*, *14*, 746-755.
- Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*, *51*, 768-774.
- Peluso, M. A., Hatch, J. P., Glahn, D. C., Monkul, E. S., Sanches, M., Najt, P.,... Soares, J. C. (2007). Trait impulsivity in patients with mood disorders. *Journal of Affective Disorders*, *100*, 227-31. doi:10.1016/j.jad.2006.09.037
- Perlman, G., Johnson, W., & Iacono, W. G. (2009). The heritability of P300 amplitude in 18 year-olds is robust to adolescent alcohol use. *Psychophysiology*, *46*, 962-969. doi:10.1111/j.1469-8986.2009.00850.x

- Petry, N. M. (2001). Substance use, pathological gambling, and impulsiveness. *Drug and Alcohol Dependence*, 63, 29-38.
- Pfefferbaum, A., Ford, J. M., White, P. M., & Mathalon, D. (1991). Event-related potentials in alcoholic men: P3 amplitude reflects family history but not alcohol consumption. *Alcoholism: Clinical and Experimental Research*, 15 (5), 839-850.
- Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118, 2128-2148. doi:10.1016/j.clinph.2007.04.019
- Polich, J. & Kok, A. (1995). Cognitive and biological determinants of P300: An integrative review. *Biological Psychology*, 41, 103-146.
- Polich, J., Pollock, V. E., Bloom, F. E. (1994). Meta-analysis of P300 amplitude from males at risk for alcoholism. *Psychological Bulletin* 115 (1), 55- 73.
- Porjesz, B. & Begleiter, H. (1990). Event-related potentials in individuals at risk for alcoholism. *Alcohol*, 7, 465-469.
- Porjesz, B., Begleiter, H., Reich, T., Van Eerdewegh, P., Edenberg, H. J., Foroud, T.,...Rohrbaugh, J. (1998). Amplitude of visual P3 event-related potential as a phenotypic marker for a predisposition to alcoholism: Preliminary results from the COGA project. *Alcoholism: Clinical and Experimental Research*, 22, 1317-1323.
- Porjesz, B., Rangaswamy, M., Kamarajan, C., Jones, K. A., Padmanabhapillai, A., & Begleiter, H. (2005). The utility of neurophysiological markers in the study of alcoholism. *Clinical Neurophysiology*, 116, 993-1018. doi:10.1016/j.clinph.2004.12.016
- Preacher, K. J. & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, 40 (3), 879-891.

- Preuss, U. W. & Wong, W. M. (2000). Comorbidity. In G. Zernig, A. Saria, M. Kurz, & S. S. O'Malley (Eds.), *Handbook of Alcoholism* (pp. 287-303). Boca Raton, FL: CRC Press LLC.
- Rachlin, H., Raineri, A., & Cross, D. (1991). Subjective probability and delay. *Journal of the Experimental Analysis of Behavior*, 55, 233–244. doi:10.1901/jeab.1991.55-233
- Ramachandran, G., Porjesk, B., Begleiter, H., & Litke, A. (1996). A simple auditory oddball task in young adult males at high risk for alcoholism. *Alcoholism: Clinical and Experimental Research*, 20 (1), 9-15.
- Regier, D. A., Farmer, M. E., Rae, D. S., Locke, B. Z., Keith, S. J., Judd, L. L., & Goodwin, F. K. (1990). Comorbidity of mental disorders with alcohol and other drug abuse: Results from the Epidemiologic Catchment Area (ECA) Study. *Journal of the American Medical Association*, 264, 2511–2518.
- Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences*, 40, 305-315.
- Richards, J. B., Zhang, L., Mitchell, S. H., & de Wit, H. (1999). Delay or probability discounting in a model of impulsive behavior: Effect of alcohol. *Journal of the Experimental Analysis of Behavior*, 71 (2), 121-143.
- Robinson, L. J., Thompson, J. M., Gallagher, P., Goswami, U., Young, A. H., Ferrier, I., N., & Moore, P. B. (2006). A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. *Journal of Affective Disorders*, 93, 105-115. doi: 10.1016/j.jad.2006.02.016

- Rosenbloom, T. (2003). Risk evaluation and risky behavior of high and low sensation seekers. *Social Behavior and Personality, 31*, 375-386.
- Russo, P. M., De Pascalis, V., Varriale, V., & Barratt, E. S. (2008). Impulsivity, intelligence and P300 wave: An empirical study. *International Journal of Psychophysiology, 69*, 112-118. doi:10.1016/j.ijpsycho.2008.03.008
- Ryu, V., An, S. K., Jo, H. H., & Cho, H. S. (2010). Decreased P3 amplitudes elicited by negative facial emotion in manic patients: Selective deficits in emotional processing. *Neuroscience Letter, 281*, 92-96. doi:10.1016/j.neulet.2010.06.059
- Schalet, B. D., Durbin, C. E., & Revelle, W. (2011). Multidimensional structure of the Hypomanic Personality Scale. *Psychological Assessment, 23* (2), 504-522. doi:10.1037/a0022301
- Schulze, K. K., Hall, M.-H., McDonald, C., Marshall, N., Walshe, M., Murray, R. M., & Bramon, E. (2008). Auditory P300 in patients with bipolar disorder and their unaffected relatives. *Bipolar Disorders, 10*, 377-386.
- Schwarz, R. M., Burkhart, B. R., & Green, S. B. (1978). Turning on or turning off: Sensation seeking or tension reduction as motivational determinants of alcohol use. *Journal of Consulting and Clinical Psychology, 46*, 1144-1145.
- Selzer, M. L. (1971). The Michigan Alcohol Screening Test: the quest for a new diagnostic instrument. *American Journal of Psychiatry, 127*, 1653-1658.
- Selzer, M. L., Vinokur, A., van Rooijen, M. A. (1975). A self-administered Short Michigan Alcoholism Screening Test (SMAST). *Journal of Studies on Alcohol, 36*, 117-126.

- Shen, I-H., Lee, D-S., & Chen, C-L. (2014). The role of trait impulsivity in response inhibition: Event-related potentials in a stop-signal task. *International Journal of Psychophysiology*, *91*, 80-87. doi:10.1016/j.ijpsycho.2013.11.004
- Shin, S. H., Hong, H. G., & Jeon, S.-M. (2012). Personality and alcohol use: The role of impulsivity. *Addictive Behaviors*, *37*, 102-107. doi:10.1016/j.addbeh.2011.09.006
- Shin, Y.-W., Krishnan, G., Hetrick, W. P., Brenner, C. A., Shekhar, A., Malloy, F. W., O'Donnell, B. F. (2010). Increased temporal variability of auditory event-related potentials in schizophrenia and schizotypal personality disorder. *Schizophrenia Research*, *124*, 110-118. doi:10.1016/j.schres.2010.08.008
- Simpson, C. A. & Vuchinich, R. E. (2000). Reliability of a measure of temporal discounting. *The Psychological Record*, *50*, 3-16.
- 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 impulsive-like traits. *Assessment*, *14*, 155-170. doi:10.1177/1073191106295527
- Smoller, J. W. & Finn, C. T. (2003). Family, twin, and adoption studies of bipolar disorder. *American Journal of Medical Genetics*, *123C*, 48-58. doi:10.1002/ajmg.c.20013
- Sonne, S. S., Brady, K. T., & Morton, W. A. (1994). Substance abuse and bipolar affective disorder. *The Journal of Nervous and Mental Disease*, *182* (6), 349-352.
- Stautz, K. & Cooper, A. (2013). Impulsivity-related personality traits and adolescent alcohol use: A meta-analytic review. *Clinical Psychology Review*, *33*, 574-592. doi:10.1016/j.cpr.2013.03.003
- Steinhauer, S. R., Hill, S. Y., & Zubin, J. (1987) Event-related potentials in alcoholics and their first-degree relatives. *Alcohol*, *4*, 307-314.

- Strakowski, S. M. & DelBello, M. P. (2000). The co-occurrence of bipolar and substance use disorders. *Clinical Psychology Review, 20* (2), 191-206.
- Strakowski, S. M., Fleck, D. E., DelBello, M. P., Adler, C. M., Shear, P. K., Kotwal, R., & Arndt, S. (2010). Impulsivity across the course of bipolar disorder. *Bipolar Disorders, 12*, 285-297. doi:10.1111/j.1399-5618.2010.00806.x
- Strakowski, S. M., Fleck, D. E., DelBello, M. P., Adler, C. M., Shear, P. K., McElroy, S. L.,...Arndt, S. (2009). Characterizing impulsivity in mania. *Bipolar Disorders, 12*, 285-297.
- Swann, A. C. (2010). The strong relationship between bipolar and substance-use disorder. *Annals of the New York Academy of Sciences, 1187*, 276-293.
- Swann, A. C., Dougherty, D. M., Pazzaglia, P. J., Pham, M., & Moeller, F. G. (2004). Impulsivity: A link between bipolar disorder and substance abuse. *Bipolar Disorders, 6*, 204-212.
- Swann, A. C., Lijffijt, M., Lane, S. D., Steinberg, J. L., Acas, M. D., Cox, B., & Moeller, F. G. (2013). Pre-attentive information processing and impulsivity in bipolar disorder. *Journal of Psychiatric Research, 47*, 1917-1924. doi:10.1016/j.jpsychires.2013.08.018
- Tellegen, A. (1982). *Multidimensional Personality Questionnaire manual*. Minneapolis, MN: University of Minnesota Press.
- Tohen, M. & Zarate, C. A. (1999). Bipolar disorder and comorbid substance use disorder. In J. F. Goldberg & M. Harrow (Eds.), *Bipolar Disorders: Clinical Courses and Outcomes* (pp. 171-184). Washington, DC: American Psychiatric Press, Inc.

- Trucco, E. M., Colder, C. R., & Wieczorek, W. F. (2011). Vulnerability to peer influence: A moderated mediation study of early adolescent alcohol use and initiation. *Addictive Behaviors, 36*, 729-736. doi: 10.1016/j.addbeh.2011.02.008
- Uslaner, J. M. & Robinson, T. E. (2006). Subthalamic nucleus lesions increase impulsive action and decrease impulsive choice – mediation by enhanced incentive motivation? *European Journal of Neuroscience, 24*, 2345-2354. doi: 10.1111/j.1460-9568.2006.05117.x
- van Beijsterveldt, C. E. M. & van Baal, G. C. M. (2002). Twin and family studies of the human electroencephalogram: A review and a meta-analysis. *Biological Psychology, 61*, 111-138.
- Venables, N. C., Patrick, C. J., Hall, J. R., & Bernat, E. M. (2011). Clarifying relations between dispositional aggression and brain potential response: Overlapping and distinct contributions of impulsivity and stress reactivity. *Biological Psychology, 86*, 279-288. doi:10.1016/j.biopsycho.2010.12.009
- Victor, S. E., Johnson, S. L., & Gotlib, I. H. (2011). Quality of life and impulsivity in bipolar disorder. *Bipolar Disorders, 13*, 303-309. doi: 10.1111/j.1399-5618.2011.00919.x
- Vollebergh, W. A. M., Iedema, J., Bijl, R. V., de Graaf, R., Smit, F., & Ormel, J. (2001). The structure and stability of common mental disorders: The NEMESIS study. *Archives of General Psychiatry, 58*, 597–603. doi:10.1001/archpsyc.58.6.597
- Vuchinich, R. E. & Simpson, C. A. (1998). Hyperbolic temporal discounting in social drinkers and problem drinkers. *Experimental and Clinical Psychopharmacology, 6* (3), 292-305.
- Whiteside, S. P. & Lynam, D. R. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*, 669-689.

- Whiteside, S. P. & Lynam, D. R. (2003). Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: Application of the UPPS Impulsive Behavior Scale. *Experimental and Clinical Psychopharmacology*, *11* (3), 210-217. doi:10.1037/1064-1297.11.3.210
- Whiteside, S. P., Lynam, D. R., Miller, J. D., & Reynolds, S. K. (2005). Validation of the UPPS Impulsive Behaviour Scale: A four-factor model of impulsivity. *European Journal of Personality*, *19*, 559-574. doi:10.1002/per556
- Whitfield, J. B., Zhu, G., Madden, P. A., Neale, M. C., Heath, A. C., & Martin, N. G. (2004). The genetics of alcohol intake and of alcohol dependence. *Alcoholism: Clinical and Experimental Research*, *28*, 1153 – 1160.
- Winstanley, C. A., Dalley, J. W., Theobald, D. E. H., & Robbins, T. W. (2004). Fractioning impulsivity: Contrasting effects of central 5-HT depletion on different measures of impulsive behavior. *Neuropsychopharmacology*, *29*(7), 1331-1343. doi: 10.1038/sj.npp.1300434
- Winstanley, C. A., Theobald, D. E. H., Dalley, J. W., Cardinal, R. N., & Robbins, T. W. (2006). Double dissociation between serotonergic and dopaminergic modulation of medial prefrontal and orbitofrontal cortex during a test of impulsive choice. *Cerebral Cortex*, *16*, 106-114. doi: 10.1093/cercor/bhi088
- Widiger, T. A. & Clark, L. A. (2000). Toward DSM-V and the classification of psychopathology. *Psychological Bulletin*, *126* (6), 946-963. doi:10.1037//0033-2909.126.6.946

- Winokur, G. (1999). Alcoholism in bipolar disorder. In J. F. Goldberg & M. Harrow (Eds.), *Bipolar Disorders: Clinical Courses and Outcomes* (pp. 185-197). Washington, DC: American Psychiatric Press, Inc.
- Woicik, P. A., Stewart, S. H., Pihl, R. O., & Conrod, P. J. (2009). The substance use risk profile scale: A scale measuring traits linked to reinforcement-specific substance use profiles. *Addictive Behaviors, 34*, 1042-1055. doi:10.1016/j.addbeh.2009.07.001
- Yi, R., Mitchell, S. H., & Bickel, W. K. (2010). Delay discounting and substance abuse-dependence. In: Madden, G.J., Bickel, W.K. (Eds.), *Impulsivity: The Behavioral and Neurological Science of Discounting*. American Psychological Association, Washington, DC, pp. 191–211.
- York, J. L., Welte, J., Hirsch, J., Hoffman, J. H., & Barnes, G. (2004). Association of age at first drink with current alcohol drinking variables in a national general population sample. *Alcoholism: Clinical and Experimental Research, 28*, 1379-1387.
- Zalla, T., Joyce, C., Szöke, A., Schürhoff, F., Pillon, B., Komano, O., ...Leboyer, M. (2004). Executive dysfunction as potential markers of familiar vulnerability to bipolar disorder and schizophrenia. *Psychiatry Research, 121*, 207-217. doi: 10.1016/S0165-1781(03)00252-X
- Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking*. Cambridge: Cambridge University Press.
- Zuckerman, M. (2007). *Sensation seeking and risky behavior*. Washington, DC: American Psychological Association.

Appendix 1

Ethnicity Differences

Due to the large number of Study 1 participants endorsing East Asian ethnicity, post-hoc analyses examined mediation models in subsamples of those endorsing the two largest ethnicity groups: European/European descent and East Asian/East Asian descent. Overall, both subsamples were quite similar to the full sample. For the European only group (n = 572), either or both Negative Urgency and Sensation Seeking mediated the relationship between HPS total score and all measures of alcohol use. With regard to the HPS subscales, Sensation Seeking partially mediated the relationships between Social Vitality and quantity x frequency and alcohol use frequency and fully mediated the relationship between Social Vitality and lifetime maximum number of drinks within a 24 hour period. Negative Urgency partially mediated the relationship between Mood Volatility and alcohol related problems. For the East Asian group (n = 414), Sensation Seeking partially mediated the relationships between HPS total score and quantity x frequency, quantity of alcohol use, and frequency of alcohol use, and fully mediated the relationship between HPS total score and lifetime maximum number of drinks within a 24 hour period and binge drinking frequency. Both Sensation Seeking and Positive Urgency were significant in the full mediation of the relationship between HPS total score and alcohol related problems. With regard to the HPS subscales, Sensation Seeking partially mediated the relationships between Social Vitality and quantity x frequency, quantity of alcohol use, frequency of alcohol use, lifetime maximum number of drinks within a 24 hour period, and binge drinking frequency. Whereas Positive Urgency partially mediated the relationship between Mood Volatility and alcohol related problems. Negative Urgency was not a significant mediator in the East Asian subsample. Therefore, it appears that the involvement of Negative Urgency is

due to Europeans, whereas the involvement of Positive Urgency is due to East Asians. Sensation Seeking continued to be an important mediator in both subsamples. This suggests the possibility that alcohol related problems occur in the context of drinking in response to negative emotions for Europeans, but in response to positive emotions for East Asians.