PERSONALITY TO SUBSTANCE MISUSE IN YOUNG PEOPLE: HOW DOES WHO YOU ARE AFFECT WHAT AND WHY YOU USE?

by

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ABSTRACT

My dissertation sought to better understand the ways in which personality confers vulnerability for substance misuse. Four traits have been reliably implicated: anxiety sensitivity, hopelessness, sensation seeking, and impulsivity. I focused on the developmental periods of adolescence and emerging adulthood, as they are characterized by increased risk for substance use and abuse. I also focused on alcohol and prescription drug misuse. To date, alcohol is the substance that has been studied most extensively. Prescription drug misuse, however, is an emerging issue – that is now considered epidemic. Thus, I validated my model with alcohol before applying it to prescription drugs. Study 1 expanded the extant literature by testing chained mediation from anxiety sensitivity and hopelessness to alcohol misuse (through specific emotional disorder symptoms and coping drinking motives) in $N = 1,883$ university students. Anxiety sensitivity and hopelessness predicted hazardous alcohol use and drinking harms via symptoms of anxiety/depression and drinking to cope with anxiety/depression. Studies 2 and 3 were novel in that they successively applied this model to prescription drug misuse. Specifically, Study 2 tested theoretical pathways from personality to distinct prescription drug classes and patterns of use in $N = 1,755$ university students. AS predicted the use and medically-sanctioned use of sedatives/tranquilizers and was marginally associated with sedative/tranquilizer misuse. Hopelessness predicted the use and medically-sanctioned use of opioids. Sensation seeking predicted the use and misuse of stimulants. Impulsivity predicted the following: sedative/tranquilizer use and misuse; opioid misuse; and stimulant use, medically-sanctioned use, and misuse. Finally, Study 3 tested whether specific sets of mental health symptoms mediated these observed personality to prescription drug misuse paths in high school students followed over one year (from Grade 9; $n = 3,024$ to Grade 10; $n = 2,869$). Anxiety sensitivity predicted sedative/tranquilizer misuse via anxiety symptoms and hopelessness predicted opioid misuse via depressive symptoms. Sensation seeking was marginally associated with stimulant misuse. Impulsivity predicted stimulant misuse via attention-deficit hyperactivity disorder symptoms. Impulsivity also predicted sedative/tranquilizer, opioid, and stimulant misuse via conduct disorder symptoms. Impulsivity, however, was not directly associated with the misuse of any of these prescription drugs. Taken together, my studies suggest that personality exerts its influence on alcohol and prescription drug misuse through mental health symptoms and substance use motives. These paths vary by trait, and map onto established etiological models of addiction. My dissertation supports the ongoing use (re: alcohol) and the development (re: prescription drugs) of personality-matched prevention and intervention efforts.

LIST OF ABBREVIATIONS AND SYMBOLS USED

AUDIT	Alcohol Use Disorders Identification Test
ACHA	American College Health Association
APA	American Psychiatric Association
ANOVA	Analysis of variance
AS	Anxiety sensitivity
ADHD	Attention-deficit hyperactivity disorder
CSTADS	Canadian Student Tobacco, Alcohol, and Drugs Survey
CUSP	Canadian Underage Substance Abuse Prevention Trial
χ^2	Chi-square
CFI	Comparative fit index
CD	Conduct disorder
CI	Confidence interval
α	Cronbach's alpha
DSM-5	Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition
DMQ	Drinking Motive Questionnaire
DCWA	Drinking to cope with anxiety
DCWD	Drinking to cope with depression
F	F statistic
GAD	Generalized anxiety disorder
HOP	Hopelessness
H	Hypothesis
IMP	Impulsivity
INCB	International Narcotics Control Board
K10	Kessler Psychological Distress Scale
M	Mean
Mdn	Median
p	P-value
PD	Prescription drug
PPP	Posterior predictive p-value
RCT	Randomized controlled trial
RMSEA	Root mean square error of approximation
N or *n*	Sample size

SS	Sensation seeking
SD	Standard deviation
SRMR	Stand root mean square residual
SPSS	Statistical Package for the Social Sciences
SAMHSA	Substance Abuse and Mental Health Services Administration
SURPS	Substance Use Risk Profile Scale
TLI	Tucker-Lewis index

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I will conclude my acknowledgements with this. As I prepared for internship interviews this past Christmas, my mom stumbled across my 2018 horoscope. It read: "Understand that you are in the process of making a big personal transition that could change your mindset to where you will enjoy nearly every component of living. You just

aren't there yet. The passage is lengthy, but worthwhile. You will have to jump through some hoops, but afterward you will emerge like a child who is seeing his or her world for the very first time. You will be amazed by something as simple as the beauty of a rose. Avoid being too serious in 2018. Pop a bottle of champagne…, and remember this moment. If you do, you might even see a long-term dream come to fruition. Just stay on course." So, in closing, I would like to thank my lucky stars. Here's to degrees being earned – and to lofty dreams finally coming true.

CHAPTER 1. INTRODUCTION

My dissertation examines the role of personality in the substance use behaviour of young people (i.e., high school and university students). It includes three publication-style manuscripts. The first tested paths from personality to alcohol outcomes in emerging adults. Chained mediation was used to examine the sequential effects of mental health symptoms and drinking motives. The second compared three models of personality to prescription drug (PD) use in emerging adults: overall PD use, medically-sanctioned PD use, and PD misuse. The third tested whether mental health symptoms mediated the relations between personality and PD use over time in adolescents. Emerging adulthood was operationally defined as 18-25 years (Arnett, 2000) and adolescence as 13-17 years (Santrock & Curl, 2003). Before presenting these studies' findings, the following will be introduced: the problem of alcohol and PD misuse during these developmental periods, the model of personality vulnerability to alcohol and PD misuse that informs my dissertation, and the objectives of my research.

Adolescence and Emerging Adulthood

The teens to thirties are characterized by a dissymmetry between the more developed subcortical limbic brain regions (implicated in affect, motivation, memory, and reward) and the still-developing prefrontal cortex (implicated in behavioural inhibition, planning, and higher-order cognitive tasks; Bergman, Kelly, Nargiso, & McKowen, 2016). Risk for substance use and abuse is disproportionately higher in adolescence and emerging adulthood as a result of these underdeveloped fontal, cortical areas; a general lack of experience with alcohol and other drugs; and higher levels of psychological distress (relative to other age groups; Substance Abuse and Mental Health Services Administration [SAMHSA], 2009). In the U.S., it is estimated that 5% of

adolescents and 17% of emerging adults meet criteria for a substance use disorder (SAMHSA, 2014). Globally, substance abuse accounts for a majority of the disability-adjusted life years lost by 15-24 year-olds (Gore *et al.*, 2011).

The *Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition* (DSM-5; American Psychiatric Association [APA], 2013) suggests that intoxication often begins in adolescence and precedes the misuse that is characteristic of addictive disorders. It is in adolescence that the majority of substance users first experiment with both licit and illicit substances (SAMHSA, 2015). On average, Canadians are 13-14 years old the first time they try alcohol (Government of Canada, 2016) and over 50% of first-time marijuana, inhalant, and hallucinogen users are teens (Anderson & Teicher, 2009; SAMHSA, 2014). This is clinically significant; early experimentation and hazardous, adolescent drinking or drug-taking are risk factors for long-standing adult addiction issues (e.g., dependence, chronic disease, and mental illness; Canadian Centre on Substance Abuse, 2007). After 10 years, for example, 14% of those who started drinking at ages 11-14 years met criteria for alcohol abuse and 9-16% for alcohol dependence. This compared to 2% and 1%, respectively, for those who started drinking at age 19 years or older (de Wit, Adlaf, Offord, & Ogborne, 2000). Substance abuse in adolescence is also associated with a number of negative outcomes and trajectories (e.g., neurocognitive impairment, academic difficulty, risky sexual behaviour, and criminality; Aebi, Giger, Plattner, Metzke, & Steinhausen, 2014; Esch *et al.*, 2014).

The *DSM-5* (APA, 2013, p. 487) further suggests that "individuals aged 18-24 years have relatively high prevalence rates for the use of virtually every substance". Emerging adults, for example, endorse the highest rates of binge drinking (five or more

drinks on one occasion during the past month), heavy drinking (five or more drinks on five occasions during the past month), illicit drug use, and PD misuse of any age group (SAMHSA, 2014). For several reasons, the emerging adult developmental period is thought to confer particular vulnerability for alcohol and drug misuse (Bergman *et al.*, 2016). First, it is characterized by exciting but stressful transitions (e.g., separation and individualization from one's parents, admission to college, entrance into the workforce, and increased time and emotional connection with peers or partners). Second, parental monitoring decreases as exposure to alcohol and other drugs increases. It is therefore unsurprising that emerging adults constitute a disproportionately large share of the overall health and economic burdens of harmful substance use and addiction (Rehm *et al.*, 2014).

In summary, young people are more likely than other age groups to engage in risky substance use and to experience related harms (Adlaf, Begin, & Sawka, 2005). My dissertation focuses on the alcohol and PD use and misuse patterns of high school and university students. I have included recent Canadian rates pertaining to these substances and ages below. These statistics serve to reinforce the acute, developmental risk of both substance use initiation and substance use disorder onset that is incurred from adolescence through to emerging adulthood.

Alcohol Use and Misuse

The Canadian Centre on Substance Abuse (2007), in partnership with the Student Drug Use Surveys working group, derived Canadian estimates of high school students' alcohol use. They used data collected in 2007-2008 from nine regularly occurring provincial surveys and one national survey (i.e., the Youth Smoking Survey; Young *et*

al., 2011). Participants were in Grades 7, 9, 10, and 12. In their lifetime, 52-70% had consumed alcohol (18-35% in Grade 7 vs. 77-91% in Grade 12). In the past year, 46-62% had used alcohol at least once (8-28% in Grade 7 vs. 75-83% in Grade 12). In the past month, 19-30% had consumed five or more drinks on a single occasion (3-4% in Grade 7 vs. 41-55% in Grade 12).

The Canadian Student Tobacco, Alcohol, and Drugs Survey (CSTADS) reports similar adolescent alcohol use rates (Government of Canada, 2016). In 2014-2015, 42,094 students in Grades 6-12 were surveyed. On average, respondents were 13.5 years old the first time they tried alcohol. Forty percent of students in Grades 7-12 had consumed alcohol that year; 24% had consumed five or more drinks on one occasion. When the students were asked how difficult they thought it would be to get alcohol if they wanted it, 67% indicated that it would be "very" or "fairly" easy.

Finally, the National College Health Assessment examined alcohol use on college and university campuses (American College Health Association [ACHA], 2016). The Canadian sample included 43,780 students and was collected in 2016. In the past 30 days, 69% had used alcohol. The average number of drinks consumed the last time students had "partied" or socialized was 5.06 (*Mdn* = 4.00). Almost half (48%) had consumed five or more drinks the last time they had "partied" or socialized. Within the last two weeks, 35% of students had engaged in binge drinking at least once. In descending order, they endorsed the following past-year alcohol-related harms: 38% did something they later regretted, 29% forgot where they were or what they did, 24% had unprotected sex, 18% had physically injured themselves, 5% had seriously considered suicide, 2% had physically injured another person, 2% had been the victim of non-consensual sex, 2% had

gotten in trouble with the police, and 1% had been the perpetrator of non-consensual sex. Fifty-five percent reported experiencing at least one such harm and 5% indicated that their own alcohol use had affected their academic performance. Research further suggests that 71% of students experience second-hand harms (Thompson, Davis-MacNevin, Teehan, Stewart, & the Caring Campus Team, 2017a). Second-hand harms are alcohol-related harms experienced by someone other than the drinker (Giesbrecht, Cukier, & Stevens, 2010). They include both strains (e.g., interrupted sleep) and threats (e.g., being harassed or insulted). Second-hand harms are associated with higher levels of anxiety and depression and with poorer subjective well-being in victims (Thompson *et al.*, 2017a).

PD Use and Misuse

Psychoactive PDs (e.g., sedatives, tranquilizers, opioids, and stimulants) are essential medications for the treatment of pain, insomnia, anxiety, attention-deficit hyperactivity disorder (ADHD), and other psychiatric disorders (Olfson, Blanco, Wang, & Greenhill, 2013a; Olfson, Crystal, Iza, Wang, & Blanco, 2013b). Management of these medications is complicated by their liability for abuse and dependence (Martins *et al.*, 2012). PDs are said to be misused when they are taken without a physician's prescription, in greater amounts or more often than prescribed, via non-intended routes, for non-prescribed reasons, and/or with contraindicated substances (Barrett, Meisner, & Stewart, 2008; Haydon, Monga, Rehm, Adlah, & Fischer, 2006). PD misuse rates use are highest in Canada and the U.S. – compared to low, middle, and other high-income countries (Holmes, 2012). Canada, for instance, has the highest per capita rates of prescription opiate use and misuse in the world (International Narcotics Control Board, 2015). It is ranked second for benzodiazepine use and within the top 15 for prescription stimulant use

(International Narcotics Control Board [INCB], 2004). Prescription opioid-related deaths are now a leading cause of premature mortality in Canada (Fischer, Keates, Bühringer, Reimer, & Jürgen, 2014; McCabe & Teter, 2007). Compared to other psychoactive drugs, the burden of PD misuse is exceeded only by tobacco and alcohol abuse (Fischer & Argento, 2012). In 2014, for example, opioids accounted for 9% of the total cost (i.e., lost productivity, healthcare, criminal justice, and other direct costs) of substance use in Canada ($3.5 billion of $38.4 billion; Canadian Substance Use Costs and Harms Scientific Working Group, 2018).

According to the 2014-2015 CSTADS (Government of Canada, 2016), high school students in Grades 9-12 preferentially misused alcohol, cannabis, and then psychoactive pharmaceuticals third. Four percent had taken prescribed sedatives, tranquilizers, pain relievers, or stimulants to get high. Opioids were misused most frequently (3%). In the past year, 1% of students had misused oxycodone, >1% fentanyl, and 2% other opioids (e.g., morphine, codeine, or Tylenol 3). When students were asked how difficult it would be to get PDs if they wanted them, 37% indicated it would be "very" or "fairly" easy to get opioids. Thirty percent reported the same for prescription stimulants used to treat ADHD. When students were asked about the potential risks of using PDs to get high, 59% saw no "great risk" for such misuse "once in a while". Thirty percent said the same of such misuse "on a regular basis".

As stated earlier, emerging adults endorse the highest rates of PD misuse (Silvestri, Knight, Britt, & Correia, 2015). According to the 2016 National College Health Assessment (ACHA, 2016), 11% of Canadian college or university students had

taken a PD that was not prescribed to them within the last 12 months. More specifically, 6% had misused opioids in this manner, 5% stimulants, and 2% sedatives/tranquilizers.

Despite young people's perceptions to the contrary, PD misuse can be risky. Physiological harms include an increased risk of negative drug interactions, withdrawal, physical dependence, organ damage, cardiovascular risk, injury related to intranasal use, and other concerning health risks (Hartung *et al.*, 2013; Holloway, Bennett, Parry, & Gorden, 2014; Teter, Falone, Cranford, Boyd, & McCabe, 2010). Psychological harms include psychological dependence and symptoms of general distress, depression, and anxiety (Cohen, 1992; Holloway *et al.*, 2014). Social harms include family problems, interpersonal issues, academic issues, misuse of other drugs, polysubstance abuse, and engagement in risky/illegal activities (Brandt, Taverna, & Hallock, 2014; Hartung *et al.*, 2013; Holloway *et al.*, 2014). PD misuse is one of the most common problems for young people who are seeking treatment for a substance use disorder, attesting to their inherent risk for dependence (Gonzales, Brecht, Mooney, & Rawson, 2011).

Personality as a Predictor of Substance Misuse

Research suggests that personality is one of the many biological, environmental, and psychological factors implicated in substance misuse onset and development (Castellanos-Ryan & Conrod, 2012). The traits most commonly associated with alcohol and drug abuse can be dichotomized into those involving inhibition vs. disinhibition. These domains correspond with the main behavioural action tendencies: avoidance vs. approach (Conway, Swendsen, Rounsaville, & Merikangas, 2002; Matthews & Gilliland, 1999). They are further related to a liability for negative vs. positive affect (Sutton & Davidson, 1997). The inhibited traits that are most consistently associated with addiction

are anxiety sensitivity (AS) and hopelessness (HOP). The disinhibited traits that are most consistently associated with addiction are sensation seeking (SS) and impulsivity (IMP). Pihl & Peterson (1995) included all four traits in their model of personality vulnerability to addiction. Individual variation and vulnerability in four psychobiological systems, they argued, is manifested in specific personality traits and increased susceptibility to initiation and maintenance of drug use and abuse. Pihl & Peterson's (1995) model has been used to predict the substance use behaviour of both adolescents and emerging adults (Krank *et al.*, 2011; Woicik, Stewart, Pihl, & Conrod, 2009). Woicik *et al.* (2009) also developed and validated the Substances Use Risk Profile Scale (SURPS), which includes AS, HOP, SS, and IMP subscales. Most recently, Castellanos-Ryan & Conrod (2012) reviewed and expanded the four-factor model's underlying theory. Their work is summarized later (in the section entitled "Etiological models of personality to substance misuse").

Inhibited Traits

Neuroticism is a broad personality construct that is characterized by behavioural inhibition, anxiety, and negative emotionality (Barlow, 2000). As such, neuroticism is associated with many forms of psychopathology, including anxiety, depression, and substance use disorders (Adan, Navarro, & Forero, 2016). Research on its structure suggests that neuroticism is a higher-order factor subsuming fear and low positive affect (Clark & Watson, 1991). This tripartite model is relevant for understanding how neurotic or inhibited traits confer risk for substance misuse. Neurotic young people, for instance, tend to drink more frequently and heavily in negative situations (Cooper, Agocha, & Sheldon, 2000; Elkins, McGue, Malone, & Iacono, 2004). Negative emotionality,

specifically, predicts alcohol and drug misuse (Elkins *et al.*, 2004; Jackson & Sher, 2003; Krueger, Caspi, & Moffitt, 2000; Sher, Bartholow, & Wood, 2000; Wills, Sandy, & Shinar, 1999). AS (which resembles the tripartite model's "fear") and HOP (which resembles the tripartite model's "low positive affect") consistently predict certain aspects of addiction.

AS is the fear of anxiety-related physical sensations due to an unrealistic expectation that they will lead to "catastrophic" consequences like physical illness or loss of mental control. High-AS youth censure themselves socially, to avoid displaying these sensations (Reiss, Peterson, Gursky, & McNally, 1986). Increased levels of AS are associated with: high drinking levels (Stewart, Finn, & Pihl, 1995); alcohol misuse (Stewart, Karp, Pihl, & Peterson, 1997); drinking problems (Conrod, Pihl, & Vassileva, 1998); and anxiolytic misuse (Conrod, Pihl, Stewart, & Dongier, 2000a). Because AS is an arousal-accelerator, it is further related to: (1) substance use motives consistent with the self-medication of anxiety (Kushner, Thuras, Abrams, Brekke, & Stritar, 2001; Stewart & Kushner, 2001); (2) pharmacological sensitivity to the arousal-dampening properties of alcohol and benzodiazepines (Conrod *et al.*, 1998; MacDonald, Baker, Stewart, & Skinner, 2000); and (3) acute and chronic withdrawal (Bakhshaie *et al.*, 2018). Findings from clinical (Kushner *et al.*, 2001) and high-risk (Mackie, Castellanos-Ryan, & Conrod, 2011) samples suggest that the relationship between AS and substance use is mediated by anxiety symptoms. AS promotes fear which, in turn, stimulates drinking and drug use. Thus, AS represents a specific risk profile – whereby substances are misused to cope with feared arousal-related sensations brought on by a variety of stressors (e.g., trauma, negative life circumstances, and even substance withdrawal;

Norton, 2001; Stewart, Samoluk, & MacDonald, 1999; Stewart, Conrod, Samoluk, Pihl, & Dongier, 2000; Zvolensky, Feldner, Eifert, & Brown, 2001). Of note, AS does not always predict increased alcohol consumption (DeMartini & Carey, 2011). For example, Novak, Burgess, Clark, Zvolensky, and Brown (2003) found that AS was not related to levels of alcohol consumption and Malmerg *et al*. (2013) concluded that AS was not predictive of later, disordered alcohol use. Taken together, these results suggest that AS' relationships with substance misuse may be more complex than other personality risk factors (Mackinnon, Kehayes, Clark, Sherry, & Stewart, 2014; Stewart & Kushner, 2001). For example, it has been suggested that these discrepant results may be a function of age. Young people who are frequently drinking for coping-related reasons now may be at an increased risk for heavy drinking later (Novak *et al.*, 2003). Another key feature that may distinguish risk or protection for high AS drinkers is expectancies about the effects of alcohol use (O'Connor, Farrow, & Colder, 2008).

HOP, on the other hand, involves a low expectation of desirable events and a high expectation of aversive events (Abramson, Metalsky, & Alloy, 1989). Conrod *et al.* (2000a) found that HOP predicted a substance misuse profile best characterized by comorbid depression and opioid dependence. Because opioids have analgesic effects (Carpenter, Chapman, & Dickenson, 2000; Gray, 1982), Conrod *et al.* (2000a) concluded that high-HOP misusers were numbing their painful experiences and memories. Other studies have similarly linked HOP to both depression (Mackie *et al.*, 2011) and substance use motives consistent with the self-medication of depression (Woicik *et al.*, 2009). At certain doses, alcohol has analgesic effects that resemble opioids' (T. Thompson, Oram, Correll, Tsermentseli, & Stubbs, 2017b). HOP is related to alcohol use, heavy episodic

drinking, alcohol-related problems, and drinking to cope with depression (Krank *et al.*, 2011; Mackinnon *et al*., 2014). Thus, HOP represents a similar risk profile – whereby substances are misused to cope with depressive symptoms.

Disinhibited Traits

Disinhibition involves the inability to plan, control, or regulate. Disinhibited behaviours are therefore associated with undue risk and negative consequences (Evenden, 1999; Iacono, Malone, & McGue, 2008). The literature describes disinhibited individuals as "impulsive", "under-controlled", "excitement seeking", acting "without premeditation", acting "without planning", and having a "low tolerance for boredom" (Depue & Collins, 1999; Whiteside & Lynam, 2001). Krueger and colleagues' research (2002, 2005, 2007) supports a hierarchical model of disinhibition: a latent externalizing factor and two lower-order factors (drug use and severe aggressive behaviour). Other factor analyses have supported two to four sub-components (Eysenck, Pearson, Easting, & Allsopp, 1985; Patton, Stanford, & Barratt, 1995). These include: lack of planning, lack of persistence, urgency (or acting rashly when distressed or upset), and SS (Smith *et al.*, 2007; Whiteside & Lynam, 2001). Personality and behavioural researchers seem to agree on at least two clear sub-dimensions of disinhibited personality: SS and IMP (Reynolds, Ortengren, Richards, & de Wit, 2006; Reynolds, Penfold, & Patak, 2008).

SS involves the need for stimulation. It is characterized by both an intolerance for boredom and a willingness to take risks in order to have novel and varied experiences (Baumeister & Vohs, 2004; Schalling, 1978). SS has been robustly and longitudinally associated with substance misuse (Conrod, Castellanos, & Mackie, 2008; Krank *et al.*, 2011). In adolescents and emerging adults, it predicts heavy episodic drinking (Conrod *et*

al., 2000a), illicit stimulant use, and non-medical prescription stimulant misuse (Low & Gendaszek, 2002). Individuals who are high in SS are sensitive to the rewarding properties of substances (Castellanos-Ryan & Conrod, 2012). As such, they are likely misusing alcohol and stimulants to increase their positive states or affects (i.e., for enhancement motives; Cooper, 1994; Simons, Gaher, Correia, Hansen, & Christopher, 2005).

Finally, IMP is associated with deficits in behavioural inhibition, reflectiveness, and planning (Baumeister & Vohs, 2004; Schalling, 1978). It plays a prominent role in addictive behaviour (Castellanos-Ryan & Conrod, 2012). It is associated with early experimentation, increased quantity and frequency of drug use, and enhanced prospective risk of substance misuse (Gerevich, Bácskai, & Rózsa, 2002; Masse & Tremblay, 1997). IMP has been linked to a motivationally undefined pattern of substance use – whereby availability best predicts abuse (Hecimovic, Barrett, Darredeau, & Stewart, 2014). Thus, high-IMP young people tend to be polysubstance users (Moody, Franck, Hatx, & Bickel, 2016).

Etiological Models of Personality to Substance Misuse

My dissertation focuses on personality as a risk factor for alcohol and PD use and misuse among adolescents and emerging adults. Previous research supports several etiological addiction models: affect regulation, pharmacological vulnerability, deviance proneness, and physiological dysregulation (Castellanos-Ryan & Conrod, 2012). Personality's role in each is discussed, in turn, below.

Affect Regulation Model

Adolescents' and emerging adults' motivations are important predictors of their substance use and misuse patterns (Kuntsche, Knibbe, Gmel, & Engels, 2005; Prendergast, 1994). Motivational models contend that: (1) substance use is motivated by a desire for a specific benefit or outcome and (2) this provides a decisional framework for consumption (Cooper, 1994; Cooper, Kuntsche, Levitt, Barber, & Wolf, 2016; Cox & Klinger, 1988).

Drinking motives. Cox & Klinger (1988) proposed a preliminary drinking motives model. They suggested that young people drink to achieve expected mood changes. Cooper (1994) then expanded this model by classifying the motives based on their valence (i.e., individuals drink to achieve a positive reward or to avoid a negative outcome) and source (i.e., individuals drink to change their internal state or external environment). Crossing these two dimensions produced four distinct motives: (1) enhancement (internally motivated to increase a positive state); (2) coping (internally motivated to reduce a negative state); (3) social (externally motivated to increase a positive state); and (4) conformity (externally motivated to reduce a negative state). Personality predicts motive endorsement (Cooper, Frone, Russell, & Mudar, 1995). From adolescence (Cooper, 1994) through emerging adulthood (Simons *et al.*, 2005), drinking motives then predict alcohol use and alcohol-related problems. Strong endorsement of any motive is associated with increased alcohol consumption (Cooper *et al.*, 2016). Certain motives are riskier, though; coping motives are associated with the poorest outcomes (Cooper, 1994; Cooper, Russell, Skinner, & Windle, 1992).

Most recently, research has suggested that anxiety and depression engender different motivational states with distinct paths to alcohol misuse (e.g., Grant, Stewart, & Mohr, 2009). As such, Grant, Stewart, O'Connor, Blackwell, & Conrod's (2007b) five-factor model subdivided the global coping motive described above (Cooper, 1994). They proposed two correlated, but separable coping motives: drinking to cope with anxiety (DCWA) and drinking to cope with depression (DCWD). DCWA and DCWD are associated with different outcomes. They predict substance-related responses to specific mood induction (Grant & Stewart, 2007; Grant, Stewart, & Birch, 2007a). Grant *et al.*'s (2007b) five-factor model has better structural fit than the original four-factor one in assessing drinking motives.

PD motives. Motives for non-medical use of PDs, on the other hand, have been dichotomized into self-treatment vs. recreational use (McCabe, Boyd, & Teter, 2009b). Self-medicating misusers take PDs for their medically-intended purpose – but without a prescription or not as prescribed (e.g., taking someone else's opioids to relieve pain or taking a higher dose of sedatives than was prescribed to fall asleep). Recreational misusers take PDs for non-medical reasons (e.g., taking a stimulant to continue drinking). McCabe *et al.* (2009b) found that 39% of undergraduate PD users fell into the self-treatment subtype, 13% into the recreational subtype, and 48% into a mixed subtype.

Personality's role. The affect regulation model suggests that individuals use substances to regulate their emotional states. Negative affect regulation involves drinking or using PDs to relieve stress, depressed mood, or anxiety (Greeley & Oei, 1999; Sher, 1987). Inhibited substance users tend to endorse such coping and self-medication motives. AS, for instance, is associated with misuse motivated by the desire to manage

one's negative emotions (Comeau, Stewart, & Loba, 2001; Conrod *et al.*, 1998; Kushner *et al.*, 2001; O'Connor, Farrow, & Colder, 2008; Stewart & Kushner, 2001). High-AS young people tend to endorse motives that are consistent with reducing anxiety (i.e., DCWA; Chandley, Luebbe, Messman-Moore, & Ward, 2014) and avoiding social rejection (i.e., conformity; Mushquash, Stewart, Mushquash, Comeau, & McGrath, 2014). HOP reflects a sensitivity to punishment. It has been linked to the analgesic suppression of depressive symptoms (Conrod *et al.*, 2000a) and to DCWD motives (Mackinnon *et al.*, 2014).

Positive affect regulation, on the other hand, involves drinking or using PDs for positive reinforcement. Substances are known to neuropharmacologically affect the brain centres involved in basic reward (Koob, 2000; Leyton, 2002). They stimulate mesolimbic dopamine activity, which is implicated in incentive salience and drug "wanting" (Berridge & Robinson, 2016). As such, many drugs are taken recreationally to increase positive mood (Cooper *et al.*, 1992). Disinhibited substance users tend to endorse such recreational and enhancement motives. SS, for example, has been consistently associated with positive reinforcement and enhancement motives (Comeau *et al.*, 2001; Cooper *et al.*, 1995; Simons *et al.*, 2005; Woicik *et al.*, 2009).

Pharmacological Vulnerability Model

This model (Sher, 1991; Sher, Grekin, & Williams, 2005) suggests that young people differ in their response to the effects of alcohol and PDs. This can put them at risk in one of two ways (Castellanos-Ryan & Conrod, 2012). First, some individuals are especially sensitive to the reinforcing effects of substances. This means that they experience greater pharmacological effects, which makes them more prone to misuse.

Second, some individuals are relatively insensitive to the reinforcing effects of substances. They must therefore consume larger amounts to achieve the desired effects, which puts them at risk of physiological dependence and other secondary harms.

Personality is relevant to this model, as inhibited users are more sensitive to certain substances' stress response-dampening effects (Conrod, Peterson, & Pihl, 1997). Here, AS is implicated more than HOP. High-AS drinkers display increased electrodermal activity in response to threat cues when they are sober – and reduced levels of reactivity after they have consumed moderate quantities of alcohol (Stewart & Pihl, 1994). Similarly, when presented with aversive stimuli after drinking, high-AS men display decreased electrodermal response and decreased heart rate compared to those lower in AS (Conrod *et al.*, 1998). Disinhibited users, on the other hand, are thought to be more sensitive to drug-induced rewards (Brunelle *et al.*, 2004; Conrod *et al.*, 1997; Erblich & Earleywine, 2003; Sher *et al.*, 2000; Zuckerman & Kuhlman, 2000). Here, SS is implicated more than IMP. Post-alcohol intoxication, high-SS drinkers have faster heart rates and report more positive, psycho-stimulating feelings (e.g., excited, stimulated, euphoric; Brunelle *et al.*, 2004). Heightened heart rate in response to alcohol is, in turn, related to greater risk of substance misuse (Sher, 1991). Reward sensitivity also mediates the path between SS and binge drinking in adolescence (Castellanos-Ryan, Rubia, & Conrod, 2011). Finally, SS is associated with greater ventral striatum amphetamine-induced dopamine release and drug wanting (Leyton, 2002).

Deviance Proneness Model

This model (Sher, 1991; Sher & Slutske, 2003) conceptualises substance use as a deviant behaviour that begins in childhood as a result of poor socialization. Longitudinal

research supports paths from inadequate parenting, childhood antisocial behaviour, childhood attentional difficulties, poor academic achievement, and poor interpersonal relationships to substance abuse (Patock-Peckman, Cheong, Balhorn, & Nagoshi, 2001; Zucker, Fitzgerald, & Moses, 1995). Problem behaviour theory suggests that personality interacts with perceived environmental systems to cause deviance (Jessor, Donovan, & Costa, 1991; Windle & Davies, 1999). Thus, while deficient socialization is emphasized as the major risk factor in this model, temperament and personality factors are also thought to play a role (Petraitis, Flay, & Miller, 1995).

Of the four-factor model traits (Pihl & Peterson, 1995), IMP is most reliably implicated in deviant behaviour. IMP is associated with comorbid antisocial and addictive behaviour (Castellanos-Ryan & Conrod, 2011). High-IMP adolescents are susceptible to increased alcohol use via conduct disorder (CD) symptoms (Mackie *et al.*, 2011). IMP is associated with conduct problems (López-Romero, Romero, & Andershed, 2015), which are, in turn, related to non-medical stimulant misuse (Van Eck, Markle, & Flory, 2012)

Psychological Dysregulation Model

This model is closely related to the deviance proneness model (presented above). Both models attribute the development of behavioural problems to an interplay of individual and environmental factors. However, the deviance proneness model is based on socio-psychological theory, while the psychological dysregulation model is rooted in psychobiological theory.

The psychological dysregulation model integrates genetic, neuropsychiatric, and environmental research. It suggests that a genetically predisposed phenotype (which is

passed from parent to child) increases one's susceptibility for substance misuse. Difficult or adverse environmental factors then trigger the development of alcohol and PD use disorders in those with this liability (Tarter *et al.*, 1999, 2003). Neurobiological disinhibition or psychological dysregulation (i.e., a cognitive, behavioural, or emotional inability to adapt to environmental challenges) are thought to be early indicators of behavioural and addiction issues (Tarter *et al.*, 1999; Thatcher & Clark, 2008). Cognitive dysfunction, irritability, and externalizing problems in childhood, for example, predict affective disorders, externalizing problems, and substance misuse in adolescence (Clark, Cornelius, Kirisci, & Tarter, 2005; Krueger *et al.*, 2002; Tarter *et al.*, 2003).

Molecular genetic studies also suggest that a significant portion of the genetic contribution to early-onset addiction is mediated by personality (Laucht, Becker, Blomeyer, & Schmidt, 2007; McGue, Iacono, Legrand, Malone, & Elkins, 2001). Disinhibition, generally, and IMP, specifically, are common endophenotypes for substance misuse and problem behaviour. ADHD, for example, is an externalizing disorder characterized by dysregulation and IMP (APA, 2013). In adolescence and emerging adulthood, ADHD tends to co-occur with substance use disorders (Wilens, Carrellas, & Biederman, 2018). Poor response inhibition is a partial mediator of the relationship between IMP and externalizing behaviours (Castellanos-Ryan *et al.*, 2011).Youth with ADHD also struggle with inhibitory control (Coutinho, Reis, de Silva, Miranda, & Malloy-Diniz, 2018). They are impulsive in social contexts, have difficulty perceiving their inadequate responses, and have trouble over-riding ongoing actions toward more appropriate ones.

Summary

All four of the etiological models described above have explanatory value, as the onset and development of alcohol and PD misuse is multi-determined. Many risk factors have been identified, including: age, genes, sensitivity to the reinforcing effects of substances, deviant peers, internalizing problems, and externalizing problems (Castellanos-Ryan & Conrod, 2012). We also know, though, that some of the adolescents and emerging adults facing these factors go on to misuse substances – while others do not. It is *here* that psychological factors may come into play. Personality traits, for example, affect the onset of substance use and misuse, development of substance use disorders, and co-occurring psychological symptoms. AS, HOP, SS and IMP may be related to substance misuse through different motivational processes – and are likely associated with different patterns of alcohol and PD use and misuse.

Castellanos-Ryan & Conrod (2012) have suggested the following paths (depicted in Figure 1.1; copyright permission from Springer Nature in Appendix A). AS exerts its effects via negative affect regulation and psychopharmacological vulnerability. It is associated with anxiety disorders and substance misuse by means of increased sensitivity to the stress-dampening effects of alcohol, sedatives, and tranquilizers. HOP exerts its effects via negative affect regulation. It is associated with mood disorders, alcohol abuse, opiate misuse, and coping motives. High-HOP users are likely self-medicating their low positive affect and high negative affect. SS exerts its effects via positive affect regulation and psychopharmacological vulnerability. Its association with alcohol abuse and stimulant misuse is mediated by heightened reward sensitivity and enhancement motives. Finally, IMP exerts is effects via deviance proneness and psychological dysregulation. It

is related to poor response inhibition, emotional reactivity, externalizing problems, and polysubstance use.

Dissertation Aims

My dissertation's primary goal was to test tenets of the theories presented above, to better understand how personality affects young people's alcohol and PD use and misuse. Because they represent periods of increased vulnerability, I focused on the developmental stages of adolescence and emerging adulthood. More specifically, each study sought to accomplish the following:

Study 1

Entitled "*Neurotic personality traits and risk for adverse alcohol outcomes: Chained mediation through emotional disorder symptoms and drinking to cope*", Study 1 examined the effects of Pihl & Peterson's (1995) inhibited traits (AS and HOP) on hazardous alcohol use and drinking-related harms. This study was cross-sectional and sampled emerging adult university students. The affect regulation model suggests that AS and HOP are related to anxiety and depression, respectively, and to drinking to cope with their symptoms. As such, we hypothesized that inhibited personality would increase the risk of alcohol use and misuse via (1) emotional disorder symptoms and then (2) specific coping drinking motives. Results from a prior study by Allan, Albanese, Norr, Zvolensky, & Schmidt (2014) supported chained mediation from AS to alcohol problems through two specific pathways: AS-generalized anxiety-coping motives-alcohol problems and AS-depression-coping motives-alcohol problems. Study 1 was an extension of theirs. We tested whether emotional disorder symptoms (anxiety, depression) and specific coping motives (DCWA, DCWD) sequentially mediated the relationships between

internalizing personality (AS, HOP) and alcohol outcomes (hazardous alcohol use, drinking harms).

Study 2

Entitled "*Personality and prescription drug use/misuse among first year undergraduates*", Study 2 examined whether personality predicted PD involvement. Three models were compared: overall PD use, medically-sanctioned PD use, and PD misuse. This study was cross-sectional and sampled emerging adult university students. It addressed the limitations of previously published studies (i.e., their focus on use vs. misuse, inconsistent operationalisation of misuse, collapsing of drug classes, and small sample sizes). We ran multivariate models that included the following paths: AS to sedatives/tranquilizers, HOP to opioids, SS to stimulants, and IMP to all three PD classes. The AS and SS hypotheses were informed by the affect regulation and pharmacological vulnerability models. The HOP hypothesis was informed by the affect regulation model. The IMP hypotheses were informed by the deviance proneness and psychological dysregulation models.

Study 3

Entitled "*Personality to prescription drug misuse in adolescents: Testing affect regulation, psychological dysregulation, and deviance proneness pathways*", Study 3 examined whether the relationships between personality and PD misuse were mediated by specific sets of mental health symptoms. This study was semi-longitudinal and sampled adolescent high school students. We tested a multivariate model that included the following paths: AS to sedative/tranquilizer misuse via anxiety symptoms; HOP to opioid misuse via depressive symptoms; SS to stimulants directly;

IMP to stimulant misuse via ADHD symptoms; IMP to sedative/tranquilizer, opioid, and stimulant misuse via CD symptoms; and IMP to sedative/tranquilizer, opioid, and stimulant misuse directly. These hypotheses were informed by all four substance misuse models: affect regulation, pharmacological vulnerability, deviance proneness, and psychological dysregulation.

Outline

Each of these studies are presented, in turn, in the upcoming chapters. Study 1 can be found in Chapter 2, Study 2 in Chapter 4, and Study 3 in Chapter 6. Chapters 3 and 5 provide transitions between studies. Chapter 7 is an integrative discussion of my entire dissertation's findings, including the theoretical and clinical implications that emerge from my work.

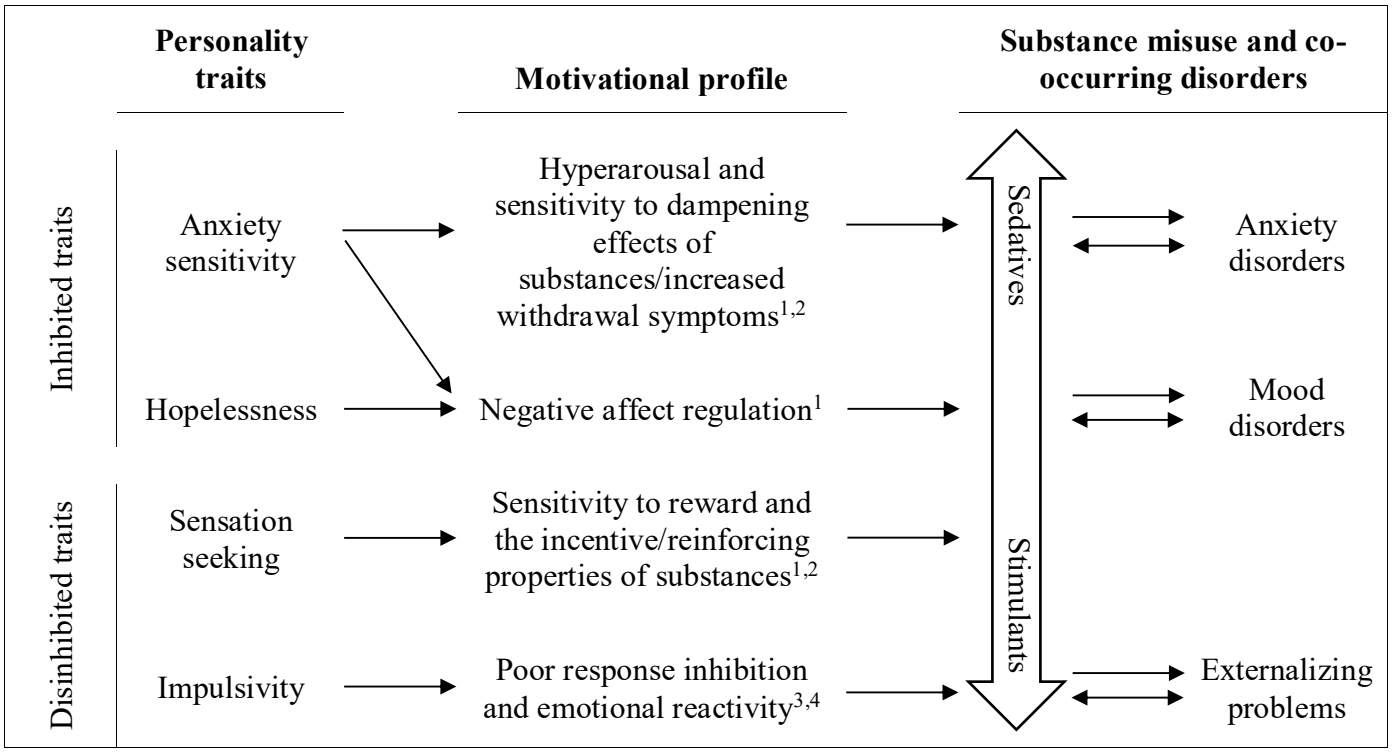

Figure 1.1. Castellanos-Ryan & Conrod's (2012) model of four distinct personality pathways to substance misuse and comorbid psychopathology. Reprinted with permission (see Appendix A). [1]offers support for the affect regulation model, [2]offers support for the psychopharmacological vulnerability model, [3]offers support for the deviance proneness model, and [4]offers support for the psychological dysregulation model.

CHAPTER 2. STUDY 1: NEUROTIC PERSONALITY TRAITS AND RISK FOR ADVERSE ALCOHOL OUTCOMES: CHAINED MEDIATION THROUGH EMOTIONAL DISORDER SYMPTOMS AND DRINKING TO COPE

The manuscript prepared for this study is presented below. Readers are advised that Annie Chinneck, under the supervision of Dr. Sherry Stewart, was responsible for developing the research questions and hypotheses, preparing the dataset for analyses, conducting some analyses, and interpreting the study findings. Annie wrote the initial draft of the manuscript; she received and incorporated feedback from her co-authors. The manuscript underwent peer-review. Annie led the response to both rounds of revision. The manuscript was accepted to *Substance Use and Misuse* on January 22, 2018. See Appendix B for copyright permission from the publisher (Taylor & Francis). The full reference is as follows:

Chinneck, A., Thompson, K., Dobson, K. S., Stuart, H., Teehan, M., Stewart, S. H., & The Caring Campus Team. (2018). Neurotic personality traits and risk for adverse alcohol outcomes: Chained mediation through emotional disorder symptoms and drinking to cope. *Substance Use & Misuse, 53,* 1730-1741.

Abstract

Rates of alcohol abuse are high on Canadian postsecondary campuses. Individual trait differences have been linked to indices of alcohol use and misuse, including neurotic traits like anxiety sensitivity and hopelessness. We know little, though, about *how* these traits confer vulnerability. Anxiety sensitivity and hopelessness are related to anxiety and depression, respectively, and to drinking to cope with symptoms of those disorders. Neurotic personality may therefore increase risk of alcohol abuse via (1) emotional disorder symptoms and/or (2) coping drinking motives. Allan *et al.* (2014) found chained mediation through anxiety sensitivity-generalized anxiety-coping motives-alcohol problems and anxiety sensitivity-depression-coping motives-alcohol problems. We sought to expand their research by examining the sequential effects of more specific predictors and mediators. We tested chained mediation from internalizing personality traits (anxiety sensitivity, hopelessness) to emotional disorder symptoms (anxiety, depression) to specific coping motives (drinking to cope with anxiety, depression) to alcohol outcomes (hazardous alcohol use, drinking harms) in university students. This study used cross-sectional data collected in Fall 2014 as part of the Movember-funded Caring Campus Project (N = 1,883). The resulting model was partially specific. Anxiety sensitivity and hopelessness were both related to hazardous alcohol use and drinking harms via emotional disorder symptoms and, in turn, coping motives. All of the indirect pathways that incorporated all four variables were statistically significant, suggesting that both mediators are necessary. The study's results have important implications for personality-matched interventions for addictive disorders.

Keywords: neurotic traits; emotional disorder symptoms; coping drinking motives; alcohol misuse; chained mediation.

Introduction

In their first year of university, students are exposed to a culture where excessive alcohol consumption is common and encouraged. For example, 69% of the 43,000 Canadian students who responded to the National College Health Assessment (ACHA, 2016) reported past-month drinking. The majority (55%) also endorsed at least one alcohol-related harm (such as feeling guilty or experiencing memory loss), and 5% reported that their drinking had negatively affected their academic performance. In light of these inflated risky drinking rates, an examination of the risk factors associated with undergraduate alcohol consumption is warranted.

Personality and Alcohol Use

Individual trait differences have been consistently linked to various indices of alcohol use/misuse (Ruiz, Pincus, & Dickinson, 2003). For instance, a meta-analysis of 20 studies (Malouff, Thorsteinsson, Rooke, & Schutte, 2007) concluded that drinking was significantly related to low conscientiousness, low agreeableness, and high neuroticism. Of these variables, neuroticism has the most robust associations with psychopathology, including addictions (Ruiz *et al.*, 2003). Characterized as being excitable or easily upset, high-neuroticism individuals are more likely to experience negative emotions. They have temperaments that are sensitive to negative stimuli (Tellegen, 1985), making them more susceptible to fear and sadness (Watson & Clark, 1984). In fact, neuroticism is an established vulnerability factor for the development of anxiety and depression (Zinbarg *et al.*, 2016). High-neuroticism individuals tend to further endorse the negatively reinforcing drinking motives (i.e., they use alcohol to relieve these aversive states; Woicik *et al.*, 2009). This is noteworthy because drinking to

cope has a particularly strong relationship with alcohol-related problems (Martens, Cox, & Beck, 2003). Among young adults, neuroticism has been linked to both sub-clinical and disordered drinking (Grant *et al.*, 2007b). More specifically, five of its six facets (angry hostility, depression, self-consciousness, impulsiveness, and vulnerability) predict alcohol-related problems (Ruiz *et al.*, 2003).

Anxiety sensitivity and hopelessness. The five factor model of personality (Costa & McCrae, 1997) is often used to describe *normal* personality. It involves several personality domains linked to alcohol use/misuse, including neuroticism. Pihl & Peterson (1995) developed a more specific model, upon which Castellanos-Ryan & Conrod (2012) elaborated. These models outline the *pathological* traits most closely related to alcohol abuse risk; they describe personality vulnerabilities associated with earlier drinking onset, heavy episodic drinking, and other alcohol problems (Castellanos-Ryan, O'Leary-Barrett, Sully, & Conrod, 2013; Krank *et al.*, 2011). Each trait corresponds to a particular biological underpinning, pattern of drinking motives, propensity for alcohol misuse, and associated mental health comorbidity. Two of the four traits are neurotic: AS and HOP. Both are associated with alcohol misuse, even after controlling for the theoretical overlap of Costa & McCrae's (1997) five factor model (which includes neuroticism; Woicik *et al.*, 2009).

AS is the fear of anxiety-related sensations, due to an unrealistic expectation that they could lead to catastrophic consequences. High-AS individuals worry that their symptoms will cause physical illness, social embarrassment, and/or loss of control (Taylor, 2014). They have dysfunctional fear motivation systems, likely as a result of heredity and learning experiences that involved arousal sensations (Taylor, MacKinnon,

& Tein, 2008; Watt, O'Connor, Stewart, Moon, & Terry, 2008). This makes them more vulnerable to anxiety disorders (McLaughlin, Stewart, & Taylor, 2007). AS is also associated with increased alcohol abuse risk. In a two-year prospective study, it was identified as a risk factor for alcohol use disorder development (Schmidt, Buckner, & Keough, 2007). High-AS individuals are more susceptible to the arousal- (Stewart & Pihl, 1994) and fear-dampening (MacDonald *et al.*, 2000) effects of alcohol. They are vulnerable to its negatively reinforcing properties and report alcohol use motives that reflect a desire to self-medicate (Woicik *et al.*, 2009). Specifically, high-AS individuals may be relying on the anxiolytic and stress-response dampening properties of alcohol to control the arousal and anxiety-related sensations they so fear (Kuntsche, Knibbe, Gmel, & Engels, 2006).

In contrast, HOP is characterized by a low expectation of desirable events, a high expectation for aversive events, and the belief that the likelihood of these outcomes cannot be changed (Abramson *et al.*, 1989). High-HOP individuals are sensitive to the threat of punishment (Castellanos-Ryan & Conrod, 2012), likely as a result of a deficient endogenous opiate system (Stanley *et al.*, 2010). This makes those high in HOP more vulnerable to mood disorders (Joiner, 2000). HOP is also associated with an increased likelihood of alcohol use, heavy episodic drinking, and alcohol-related problems (Krank *et al.*, 2011). Studies show that individuals who feel hopeless after a significant life stressor are more likely to drink to cope with their negative affect. Moreover, among college students, depression and self-consciousness (both lower-order facets of HOP-related neuroticism) predict the use of negatively reinforcing drinking motives (Stewart & Devine, 2000). It is therefore likely that high-HOP individuals are trying to manage,

reduce, or self-medicate their depressive symptoms by misusing alcohol (Woicik *et al.*, 2009).

How Personality Confers Vulnerability for Alcohol Misuse

Pihl & Peterson's (1995) model is well-supported in the alcohol literature (see also Castellanos-Ryan & Conrod, 2012); its traits have been consistently linked to negative outcomes (Krank *et al.*, 2011; Woicik *et al.*, 2009). Few studies have tested its underlying theory, though, so we know little about *how* the neurotic personality traits confer vulnerability for alcohol misuse. AS and HOP are related to anxiety and depression, respectively, and to drinking to cope with the symptoms of those disorders. Neurotic personality may therefore increase risk of alcohol use/abuse via (1) emotional disorder symptoms and/or (2) coping drinking motives.

Emotional disorders. Previous research confirms that personality is related to emotional disorders. Neuroticism, for example, is a vulnerability factor for the development of anxiety and mood disorders (Zinbarg *et al.*, 2016). AS is linked to anxiety (McLaughlin *et al.*, 2007) and HOP to depression (Joiner, 2000). Studies also show that emotional disorder symptoms precede alcohol abuse. Among college students, for example, having generalized anxiety disorder (GAD), panic disorder, agoraphobia, and/or social phobia quadruples the risk of alcohol dependence three to six years later (Kushner, Sher, & Erickson, 1999). Childhood depression is associated with (1) earlier drinking onset, risk of intoxication, and alcohol-related problems in late adolescence and (2) alcohol dependence in young adulthood (Crum *et al.*, 2008).

Coping drinking motives. Motivational models of alcohol use/misuse contend that individuals drink to achieve a desired outcome (Cooper *et al.*, 2016; Cox & Klinger,

1988). Drinking motives predict undergraduate alcohol use/abuse (Simons *et al.*, 2005) and can be categorized based on their source (internal vs. external) and valence (positive vs. negative reinforcement). By crossing these two dimensions, Cooper (1994) developed a four-factor drinking motives model. The internal, negatively reinforcing motive predicts alcohol-related problems most reliably (Cooper *et al.*, 2016). Here, one drinks to reduce or avoid negative emotions (i.e., to *cope*).

Different personality traits are associated with different self-reported reasons for drinking. Loukas, Krull, Chassin, & Carle (2000) found that neuroticism initially correlated with all four of Cooper's (1994) drinking motives (enhancement, coping, social, and conformity). After controlling for the other motives, however, only the neuroticism-to-coping relationship remained significant. Thus, Loukas *et al.* (2000) concluded that high-neuroticism drinkers were predominantly using alcohol to alleviate their anxiety and sadness. In their review, Cooper *et al.* (2016) also discuss personality as a predictor of motives. They note that drinking to cope is the motive most strongly related to increased alcohol-related problems/harms – even when enhancement, coping, social, and conformity motives are controlled. Drinking to cope may therefore mediate the relationship between neurotic personality and alcohol use/abuse. Stewart, Zvolensky, & Eifert (2001) previously demonstrated that coping motives mediated the relationship between AS and both (1) weekly drinking frequency, and (2) yearly excessive drinking frequency, in undergraduate students.

Research, however, suggests that anxiety and depression are different affective states with distinct links to alcohol misuse (Grant *et al.*, 2009). As such, the global coping motive described above was subdivided into two correlated but separable coping motives.

The resulting five-factor model (Grant *et al.*, 2007b) differentiates DCWA vs. DCWD. In a sample of undergraduate drinkers, this revised model predicted prospective alcohol outcomes and had better structural fit than the original four-factor one. Both DCWA and DCWD are associated with alcohol-related problems (Kuntsche *et al.*, 2005). Mackinnon *et al.* (2014) also demonstrated that HOP is a longitudinal, indirect predictor of alcohol problems through DCWD motives.

Chained mediation. AS is related to anxiety (e.g., McLaughlin *et al.*, 2007). Drinkers who are high in anxiety are more likely to endorse drinking to cope, than those lower in anxiety (Grant *et al.*, 2007b). DCWA motives are associated with alcohol problems (Kuntsche *et al.*, 2005). HOP, on the other hand, is related to depression (Joiner, 2000). Depressed drinkers use alcohol to cope with their low mood (Grant *et al.*, 2007b). DCWD motives are associated with hazardous alcohol use (Krank *et al.*, 2011). Neurotic personality traits (AS, HOP) may therefore lead to problematic drinking via emotional disorder symptoms (anxiety, depression) and specific coping motives (DCWA, DCWD). Using chained mediation, the sequential and respective effect of multiple mediators can be examined (Taylor *et al.*, 2008).

For example, Allan *et al.* (2014) tested the effect of AS on alcohol problems, through (1) anxiety or depression and, in turn, (2) each of Cooper's (1994) drinking motives (conformity, social, enhancement, and coping). They found chained mediation through AS-*generalized anxiety*-coping motives-alcohol problems and through AS-*depression*-coping motives-alcohol problems. However, the following limitations are noted. Allan *et al.* (2014) examined only one neurotic vulnerability, so their results cannot speak to whether AS predicts alcohol problems within the context of other

established neurotic personality risk factors (like HOP). Similarly, they assessed drinking to cope with negative affect globally, but did not examine the more specific DCWA vs. DCWD motives. The present study is an expansion; it examines how neurotic traits (Castellanos-Ryan & Conrod, 2012; Pihl & Peterson, 1995) might confer vulnerability for alcohol misuse. Specifically, we investigated whether emotional disorder symptoms (anxiety, depression) and specific coping motives (DCWA, DCWD) sequentially mediated the AS/HOP to hazardous alcohol use/drinking harms relationships among university students.

The Present Study

Personality was hypothesized to influence alcohol use/abuse via chained mediation (Figure 2.1). We predicted that neurotic personality would increase the likelihood of emotional disorder symptoms, which would then increase coping motives and, in turn, influence adverse drinking outcomes. More specifically it was hypothesized that: (1) the positive relationship between AS and drinking (hazardous alcohol use and drinking harms) would be specifically mediated by anxiety and, in turn, DCWA; and (2) the positive relationship between HOP and our alcohol outcomes would be specifically mediated by depression and, in turn, DCWD.

While some of this work will be a replication (e.g., of Allan *et al.*, 2014), all of the articulated factors had yet to be entered together into a single model. Our study also took advantage of psychometric advances in the measurement of drinking motives (Grant *et al.*, 2007b) by distinguishing DCWA vs. DCWD. We measured both hazardous alcohol use and drinking harms (as opposed to just alcohol problems). Finally, most of the existing literature has looked at adolescents (Battista, Pencer, McGonnell, Durdle, &

Stewart, 2013) or adults (Crutzen, Kuntsche, & Schelleman-Offermans, 2013). A renewed focus on undergraduates (who are approaching or have reached legal drinking age, who have moved away from home, and who have lost important social networks) is therefore warranted – given their elevated risk for problematic drinking (ACHA, 2016).

Methods

The present study used data from the Caring Campus Project, a Movember-funded research study (Stuart *et al.*, In press). Phase I involved collecting survey data from students at three Canadian universities. Students' alcohol and substance use patterns and their perceptions of on-campus drinking were assessed. The present study, which is archival, used data collected from Wave 1 (Fall 2014).

Participants

Participants were in first-year at an Eastern (N = 870), Eastern-Central (N = 577), and Western (N = 436) Canadian university. Analyses were based on 1,883 students who were, on average, 18.3 years old (SD = 1.1). The total sample was evenly distributed across genders (50% female, 50% male, and >1% identified as "other"). Nine percent were international students, and 70% lived on campus.

Procedure

Ethical approval was independently granted by each institution's Research Ethics Board. Of note, the following site-specific recruitment strategies were employed. At the Eastern university, all first-year undergraduates were invited to participate by email. In keeping with their youth advisory team's decision and the funder's (i.e., Movember's) mandate to focus on male mental health and substance use, the Eastern-Central university emailed only their first-year males. The Western university advertised in various

electronic newsletters, so undergraduates of any year could participate. For consistency, analyses were restricted to first years. All three sites also used secondary recruitment strategies like on-campus posters and social media advertising. The survey was administered between October and November of 2014. Participants received modest compensation in the form of gift cards or class credit. They could also donate the cash value to support Movember-sponsored mental health and alcohol harm reduction activities on campus. Response rates were 32% for the Eastern and Eastern-Central universities. This is comparable with other Canadian surveys conducted with University populations (e.g., ACHA, 2016). The response rate at the Western university could not be calculated, as their recruitment strategy did not allow for determination of the *unique* number of people approached to complete the survey.

Measures

The survey included approximately 55 questions and took approximately 20 minutes to complete.

Neurotic personality (see Appendix C). Assessed using the SURPS (Woicik *et al.*, 2009). This 23-item self-report questionnaire contains four subscales, which map onto the four-factor model (Castellanos-Ryan & Conrod, 2012; Pihl & Peterson, 1995). Based on our stated interest in *neurotic* vulnerability, only the AS (5 items) and HOP (7 items) subscales were administered. Based on item content and face validity, the SURPS AS subscale assesses physical concerns (more so than cognitive or social concerns; Taylor *et al.*, 2007). The SURPS HOP subscale assesses the degree to which a respondent holds negative expectations about themselves, their world, and their future (Beck, Weissman, Lester, & Trexler, 1974). Participants responded using a 5-point Likert scale

(1 *strongly disagree* to 5 *strongly agree*). Subscale scores were generated by summing component items. They ranged from 5-25 for AS and 7-35 for HOP, with higher scores representing greater levels of that trait. Both subscales have good internal consistency, test-retest reliability, and factorial validity when used with adolescents and emerging adults (Castellanos-Ryan *et al.*, 2013; Krank *et al.*, 2011; Woicik *et al.*, 2009). In our sample, both were reliable (AS $\alpha = .76$; HOP $\alpha = .85$).

Emotional disorder symptoms (see Appendix D). Assessed using the Kessler Psychological Distress Scale (K10; Kessler *et al.*, 2002). It is a 10-item self-report questionnaire. Participants were asked how often (in the last 30 days) they had experienced a given symptom; a 5-point Likert scale (1 *none of the time* to 5 *all of the time*) was used to quantify their responses. The K10 has good concurrent validity; scores are positively associated with past-year mental health consultations (Andrews & Slade, 2001). A confirmatory factor analysis conducted by Brooks, Beard, & Steel (2006) supported two second-order factors: Anxiety (nervousness and agitation) and Depression (negative affect and fatigue). Factors scores were therefore generated by summing component items. They ranged from 4-20 for Anxiety (4 items) and 6-30 for Depression (6 items), with higher scores representing greater symptomology. In our sample, both factors were reliable (Anxiety $\alpha = .81$; Depression $\alpha = .90$).

Coping drinking motives (see Appendix E). Assessed using an adapted version of the Drinking Motive Questionnaire (DMQ) Revised Short Form (Kuntsche & Kuntsche, 2009). The 12-item DMQ Revised Short Form was adapted from the original 20-item DMQ Revised (Cooper, 1994). We added three items that reflected Grant *et al.*'s (2007b) reconceptualization of the coping motive. Our subscales were consistent with the

Modified DMQ Revised (Grant *et al.*, 2007b): enhancement, social, conformity, DCWA, and DCWD. Participants reported how often in the past term they had used alcohol for the specified reason. A 5-point Likert scale (1 *almost never/never* to 5 *almost always/always*) was used to quantify their responses. For the purposes of this study, only the DCWA (3 items) and DCWD (3 items) subscales were scored by averaging component items. Higher scores indicate greater motive endorsement. The DMQ Revised Short Form is reliable and valid (Kuntsche & Kuntsche, 2009). When used with undergraduates, the Modified DMQ Revised DCWA and DCWD subscales also have good internal consistency, test-retest reliability, and factorial validity (Grant *et al.*, 2007b). In our sample, both were reliable (DCWA $\alpha = .77$; DCWD $\alpha = .84$).

Drinking outcomes. Hazardous drinking was assessed using the short form Alcohol Use Disorders Identification Test-3 (AUDIT-3; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993; see Appendix F). This 3-item self-report questionnaire measures frequency of drinking, quantity of alcohol consumed, and frequency of binge drinking. Responses were scored using a 5-point Likert scale: 0 *never* to 4 *four or more times a week* (for frequency) or 0 *one or two drinks* to 4 *10 or more drinks* (for quantity). The AUDIT-3 total was calculated by summing all three items, yielding a total score of 0-12. Higher scores indicate more hazardous drinking. The AUDIT-3 has good concurrent and discriminant validity; it is useful for early detection of hazardous or harmful drinking (Bohn, Babor, & Kranzler, 1995). In our sample, the AUDIT-3 had good internal consistency ($\alpha = .83$).

Alcohol-related harms were also assessed by asking participants if they had experienced 27 harms as a result of their drinking (e.g., passing out, being hungover, or

having an argument) in the past term (see Appendix G). Responses were scored dichotomously (0 *no* and 1 *yes*) and summed to create a 0-27 total. Higher scores indicate that the respondent experienced more drinking harms. In our sample, it was reliable (α = .95).

Statistical Analyses

The K10 (Kessler *et al.*, 2002) is often used as a generic measure of emotional distress. Because we planned to separate anxious vs. depressive symptoms, this distinction needed validation. As discussed, Brooks *et al.* (2006) concluded that the K10 had four factors that loaded onto two second-order factors: anxiety (nervousness and agitation) and depression (negative affect and fatigue). This structure was stable across two waves of prospective data (n = 1,407 community adults) and was cross-validated using the Australian National Survey of Mental Health of Well-Being (n = 10,641 community adults). To test whether this factor structure held in Canadian emerging adults (n = 1,883 first-year undergraduates), we used confirmatory factor analysis.

Chained mediation (Taylor *et al.*, 2008) was then used to examine the relationship between neurotic personality (AS, HOP) and adverse drinking outcomes (Hazardous Alcohol Use, Drinking Harms), with emotional disorder symptoms (Anxiety, Depression) and coping drinking motives (DCWA, DCWD) as chained mediators. All models were fit in MPlus 7.11 (Muthén & Muthén, 1998-2012). Outcome distributions were as follows: Anxiety skew = .81 and kurtosis = .42, Depression skew = .93 and kurtosis = .40, DCWA skew = .97 and kurtosis = .16, DCWD skew = 1.34 and kurtosis = 1.06, Hazardous Alcohol Use = .07 and kurtosis = -1.26, and Drinking Harms skew = 1.31 and kurtosis 2.31. A maximum likelihood (ML) estimator was used, as it is well-equipped to handle

non-normally distributed data (Wall, Guo, & Amemiya, 2012). Full-information maximum likelihood estimation was used to handle missing data. Path models were fitted to continuous item-level data. Gender differences were also tested using a multiple-group model. A comparison of model fit between constrained and unconstrained models revealed no significant difference ($\Delta\chi^2$ (4) = 3.31, p = .50), suggesting that parameter estimates did not differ significantly by gender. Thus, we did not compare male and female results. Data collection Site and Age were controlled, by regressing them onto the mediating and outcome variables (i.e., onto all but personality). See Appendix H (Table H.1) for a supplementary correlation table that includes all of the covariates and other variables of interest. The overall model fit was assessed using χ^2 and other incremental fit indices. Standard indices were used to assess model fit. RMSEA/SRMR ≤ 0.05 and CFI/TLI ≥ .95 indicate good fit. RSMEA/SRMR ≤ 0.08 and CFI/TLI ≥ .90 indicate adequate fit (Hu & Bentler, 1999). Of note, chi-square values are often significant when the sample size is large (Curran, West, & Finch, 1996). Thus, given our large sample size, we have reported chi-square but have not interpreted it as a stand-alone fit statistic. Significant effects were detected at a 95% confidence interval. Bootstrapped confidence intervals were used to determine the significance of the mediated effects.

Results

Confirmatory Factor Analysis

The predicted K10 structure (Brooks *et al.*, 2006) was upheld in our emerging adult sample (see Figure 2.2). All factor loadings were significant (p < .001) and the model fit well ($\chi^2(30) = 426.91$, p < .001; RMSEA = .08, 90% CI [.08, .09]; CFI = .96; TLI = .94, SRMR = .03). In the interest of model parsimony, the four second-order

Anxiety items and the six second-order Depression items were summed to create the two emotional disorder symptom subscales used in the analyses below (as opposed to using factor scores).

Chained Mediation

Descriptive statistics and correlations are displayed in Table 2.1. The hypothesized path model (see Figure 2.3) provided good fit to the data ($\chi^2(8) = 13.83$, $p = .09$; RMSEA = .04, 90% CI [.00, .04]; CFI = .99; TLI = .99; SRMR = .01). As hypothesized, chained mediation occurred from AS, through Anxiety and then DCWA, to both Hazardous Alcohol Use and Drinking Harms. There was also evidence, however, that the association between AS and our alcohol outcomes was mediated by Depression and DCWD. A software-specific command (i.e., the NEW parameter option in Mplus) was used to test the significance of the difference between these various mediation effects (Lau & Cheung, 2010). The hypothesized AS-Anxiety-DCWA-Hazardous Use path was significantly stronger than the AS-Anxiety-*DCWD*-Hazardous Use path (95% CI [.002, .03]), indicating partial specificity.

As hypothesized, chained mediation also occurred from HOP, through Depression and then DCWD, to both Hazardous Alcohol Use and Drinking Harms. The association between HOP and our alcohol outcomes was also mediated, however, by Anxiety and DCWA. The NEW parameter option showed that the hypothesized HOP-Depression-DCWD-Hazardous Use path was significantly stronger than the HOP-*Anxiety*-DCWD-Hazardous Use path (95% CI [.003, .03]). The hypothesized HOP-Depression-DCWD-Drinking Harms path was also significantly stronger than: (1) the HOP-*Anxiety*-DCWD-

Drinking Harms path (95% CI [.01, .09]) and (2) the HOP-*Anxiety*-*DCWA*-Drinking Harms path (95% CI [.003, .08]). Again, this suggests partial specificity.

All of the indirect pathways incorporating only one of two mediators (e.g., AS-*Anxiety*-Hazardous Alcohol Use) were non-significant (see Table 2.2). All of the pathways incorporating both mediators (e.g., AS-*Anxiety*-*DCWA*-Hazardous Alcohol Use) were significant. After accounting for the mediators, AS and HOP were both directly associated with *less* Hazardous Alcohol Use. HOP was associated with *less* Drinking Harms. AS was not directly associated with Drinking Harms.

Discussion

Allan *et al.* (2014) found chained mediation from AS to alcohol problems through anxiety/depression and then drinking to cope with generic, negative affect. They concluded that AS was indirectly related to alcohol abuse via these intervening variables. Our results are both a replication and extension of theirs.

Direct Effects

Descriptive statistics (displayed in Table 2.1) suggest that personality, emotional disorder symptoms, and coping drinking motives are all directly predictive of alcohol outcomes. All correlations were significant at $p < .05$, save for anxiety to hazardous alcohol use.

Indirect Effects

Our model supported both Anxiety/Depression and DWCA/DCWD (which represent more specific coping motives) as mediators in the AS-to-Hazardous Alcohol Use/Drinking Harms relationships. Unlike Allan *et al.* (2014), we also tested the chained mediation of a second neurotic personality risk factor: HOP. Like with AS, our model

supported both Anxiety/Depression and DWCA/DCWD as mediators in the HOP-to-Hazardous Alcohol Use/Drinking Harms relationships. HOP is therefore indirectly related to alcohol misuse via the same intervening variables, namely emotional disorder symptoms and coping drinking motives. In summary, AS and HOP were related to increased anxiety and depression. Both were indirectly associated with drinking to cope with these symptoms. Because we included all eight variables in a single model, we can further conclude that AS and HOP are related to alcohol problems in the context of (and while controlling for) the other.

Evidence of specificity. While *all* the indirect pathways with both mediators (emotional disorder symptoms and coping drinking motives) were significant, there was some evidence of specificity. DCWA motives were more strongly associated with Anxiety than Depression for AS-Anxiety-DCWA-drinking outcomes. This is consistent with Grant *et al.*'s (2007b) five-factor drinking motives model. It suggests that, in AS-targeted treatments, clinicians should focus on the tendency to drink to cope with anxiety when panicked or worried. Castellanos & Conrod (2006), for example, found that personality-targeted cognitive-behavioural interventions reduced relevant psychological problems in youth. Compared to a no-intervention control, the AS-targeted intervention moderately reduced alcohol misuse and concurrent panic attacks or school avoidance. Similarly, for HOP-Depression-DCWD-drinking outcomes, depression was more strongly associated with HOP than AS. This is consistent with the idea that HOP makes one more vulnerable to mood disorders (Joiner, 2000). Castellanos and Conrod's (2006) negative thinking-targeted intervention for high-HOP youth also moderately and specifically reduced alcohol misuse and concurrent depression levels.

Evidence of lack of specificity. Many of the observed associations were not as specific as theory would suggest. The association between AS and Hazardous Alcohol Use/Drinking Harms was mediated not only by Anxiety and DCWA, but by Depression and DCWD. This is not altogether surprising. Previous research supports a dual mediator model, whereby both depression and problematic coping indirectly affect the AS-to-alcohol dependence relationship (Lechner *et al.*, 2014). The association between HOP and drinking was also mediated not only by Depression and DCWD, but by Anxiety and DCWA. This lack of specificity may be due to overlapping anxiety and mood disorders (Engels *et al.*, 2010), as evidenced by their high current/lifetime comorbidity rates (Barlow, 2004). It may also be reflective of the ongoing debate about whether coping motives should be measured globally or specifically.

As previously stated, the original four-factor drinking motives model (Cooper, 1994) includes a generic drinking to cope with negative affect factor. The five-factor model differentiates DCWA vs. DCWD (Grant *et al.*, 2007b). While it is logical to assume that drinking to cope with a specific negative affect (i.e., anxiety or depression) should only mediate the relationships between that affect and alcohol-related outcomes – our results failed to support this. So did result by Bravo & Pearson (2017). Using two independent samples of college drinkers, they tested the effects of anxiety/depression on alcohol-related problems via: (1) DCWA only; (2) DWCD only; (3) a global drinking to cope with negative affect factor; and (4) both DCWA and DCWD. In all four models, the total indirect effects of anxiety and depression on alcohol-related problems were significant. The indirect effect was slightly larger in the third model, which included the global coping factor. In their discussion, Bravo & Pearson (2017) therefore suggested that

the more inclusive motive categories (like drinking to cope with negative affect) applied to a wider range of instances (e.g., drinking to cope with other negative feelings, like shame). They also noted that clinical recommendations are best built from within-subjects analyses, which *have* supported distinguishing affective states. In Grant *et al.*'s (2009) daily diary study, for example, DCWA alone positively moderated the daily anxiety-to-alcohol consumption relation and DCWD alone positively moderated the daily depression-to-alcohol consumption one.

Our results are also mixed. Some of the predicted paths were stronger (e.g., for AS-Anxiety-DCWA-drinking outcomes, DCWA motives were more strongly associated with Anxiety than Depression). This suggests that the coping motives should be split. But this specificity was not consistent throughout our model (e.g., for HOP-Depression-DCWD-drinking outcomes, DCWD motives were not more strongly associated with Depression). Rather than questioning if coping motives should be measured generally or specifically (i.e., DCWA vs. DCWD), future research might better ask: under what circumstances is splitting the coping motive useful? Researchers might also compare the magnitude of chained mediation for generic vs. specific coping motives, using the models tested herein. Further, daily diary studies (e.g., Grant *et al.*, 2009) have supported distinct event-level motivational pathways, while between-subject studies (like ours) have not consistently supported distinct trait-like ones. This may be because daily diary studies measure specific instances of drinking (vs. overall proclivities). For this reason, future studies may wish to also incorporate diaries to answer this question more conclusively.

Mediated Effects

Once the direct effects/mediated pathways were accounted for, DCWA and DCWD continued to predict hazardous alcohol use and drinking harms – while anxiety and depression did not. Finally, in our model, AS and HOP were associated with less hazardous alcohol use and drinking harms. Preliminary correlations (see Table 2.1) show that AS and HOP were significantly negatively correlated with hazardous alcohol use at the bivariate level. However, AS and HOP were significantly *positively* related to drinking harms at the bivariate level. Thus, our model's latter effect suggests that (after accounting for their associations with emotional disorder symptoms and coping drinking motives) something about AS and HOP protects against alcohol-related harm. For example, high-AS individuals may engage in fear-mediated avoidance – while those high in HOP may experience depression-mediated withdrawal. In other words, neuroticism may deter students from being in the social contexts in which problematic student drinking typically occurs (Peterson, Morey, & Higgins, 2005) and where harms are likely to ensue. Additionally, the negative associations of AS and HOP with hazardous alcohol use are consistent with prior mixed findings. Neurotic traits (particularly AS) are not always associated with greater drinking and may even be associated with less hazardous alcohol. Again, this underscores the complexity of the associations between these neurotic traits and alcohol outcomes like ours (DeMartini & Carey, 2011; Malmerg *et al*. 2013; Zvolensky, & Brown, 2003).

Clinical Implications

Previous research has supported the use of personality-matched interventions for addictive disorders. What we do not yet know is if high-AS patients would benefit from

HOP-targeted interventions, and vice versa. Conrod *et al.*'s (2000b) randomized controlled trial (RCT) indicated that motivation-matched interventions were more effective than motivation-mismatched ones. The former significantly reduced the frequency and severity of problematic alcohol use and prevented the use of multiple medical services at six months. The latter involved targeting a theoretically different personality profile, which included but was not limited to targeting AS in high-HOP individuals and vice versa. On the one hand, our partially specific results suggest that focusing on AS vs. HOP is beneficial. However, our observed lack of *full* specificity suggests that non-specific protocols (e.g., the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders; Barlow *et al.*, 2011) may be as effective as personality-targeted interventions. Unlike Conrod *et al.* (2000b), our model cannot speak to the efficacy of matched vs. mismatched intervention within the neurotic spectrum. High-AS participants, for example, might fare as well as high-HOP drinkers (i.e., matched drinkers) in the HOP-targeted intervention. Given our equivocal results, these research questions require future study. Identifying "causal" pathways in correlational research allows for the preliminary identification of potential personality-to-alcohol outcomes mechanisms. Our results could therefore inform personality-matched treatment, once re-tested in a RCT that includes non-specific, matched, and mismatched intervention groups.

Limitations

While we followed Grant *et al.*'s (2007b) lead and subdivided global coping into DCWA vs. DCWD, we did not measure AS and HOP's lower order factors. AS, for instance, has three sub-factors: fear of somatic sensations, cognitive dyscontrol, and

socially observable anxiety symptoms (Zinbarg, Molman, & Hong, 1999). Tapping into these constructs would require using a measure like the Anxiety Sensitivity Index-3 (Taylor *et al.*, 2007). This is clinically relevant as Lechner *et al.* (2014) found that only AS-total and AS-physical concerns' relationships with alcohol dependence were mediated by depression. Second, in the interest of model parsimony, we did not control for the other less theoretically-relevant drinking motives (enhancement, coping, social, and conformity). Given that the motives are all inter-correlated (Cooper, 1994), future studies may want to control them to examine the unique contributions of coping. Third, while our confirmatory factor analysis supported the use of the K10's Anxiety and Depression subscales, future studies may wish to reduce multicollinearity by using more specific scales. Relatedly, some of the SURPS and K10 items are overlapping (e.g., the K10's fourth item assesses HOP content). Fourth, our survey did not query ethnicity. As such (even though age and gender were evenly distributed), we cannot be certain that our sample demographics are representative of all Canadian university students. Finally, this study was cross-sectional. A true test of chained mediation requires longitudinal data with, in our case, at least four waves. It is also possible that our variables could be ordered in a different causal chain or that mediation could occur in the reverse direction (i.e., motives predict alcohol-related outcomes via personality). However, theory (Cox & Klinger, 1988) and past empirical tests (Cooper *et al.*, 2016) suggest that this is not the case.

Conclusion

In summary, the present study extended prior work by clarifying how neurotic personality might confer vulnerability for alcohol misuse. Specifically, chained mediation

was observed from AS and HOP to emotional disorder symptoms (anxiety, depression) to coping drinking motives (DCWA, DCWD) to adverse alcohol outcomes (hazardous alcohol use, drinking harms). However, the observed pathways were only partially specific. As such, it remains unclear whether personality-matched interventions for AS and HOP would perform better than a generic intervention for neurotic traits in terms of impacts on alcohol misuse.

Table 2.1. Means, Standard Deviations, and Correlations.

N = 1,883	*M*	*SD*	1	2	3	4	5	6	7	8
1. AS	12.34	2.98	1.00	**.24**	**.20**	**.16**	**.39**	**.38**	**- .11**	**.12**
2. HOP	13.38	3.54		1.00	**.29**	**.29**	**.47**	**.66**	**- .13**	**.14**
3. DCWA	2.09	1.05			1.00	**.67**	**.36**	**.32**	**.22**	**.35**
4. DCWD	1.84	1.04				1.00	**.32**	**.36**	**.19**	**.39**
5. Anxiety	8.97	3.31					1.00	**.70**	- .05	**.24**
6. Depression	13.04	5.38						1.00	**- .08**	**.22**
7. Hazardous Alcohol Use	4.37	3.39							1.00	**.48**
8. Drinking Harms	6.03	5.40								1.00

Note. AS is Anxiety Sensitivity (range = 5-15) and HOP is Hopelessness (range = 7-35). Both were measured using the SURPS (Woicik *et al.*, 2009). Anxiety (range = 4-20) and Depression (range = 6-30) were measured using the K10 (Kessler *et al.*, 2002). DCWA is Drinking to Cope with Anxiety (range = 3-15) and DCWD is Drinking to Cope with Depression (range = 3-15). Both were measured using the DMQ Revised Short Form (Kuntsche & Kuntsche, 2009). Hazardous Alcohol Use (range = 0-12) was measured using the AUDIT-3 (Saunders *et al.*, 1993). Drinking Harms included 27 potential harms (range = 0-27). Bold correlations are significant at $p < .05$.

Table 2.2. Indirect Effects of Neurotic Personality on Drinking through Internalizing Disorders and Coping Drinking Motives.

Predictor	Mediator 1	Mediator 2	Outcome	Indirect Effect	95% Confidence Interval
AS	Depression		Hazardous Use	- .01	[- .04, .01]
AS	Anxiety		Hazardous Use	- .01	[- .04, .01]
AS	Depression	DCWD	Hazardous Use	.01	[.01, .02]*
AS	Anxiety	DCWD	Hazardous Use	.01	[.00, .01]*
AS	Depression	DCWA	Hazardous Use	.01	[.01, .02]*
AS	**Anxiety**	**DCWA**	**Hazardous Use**	**.02**	**[.01, .03]***
HOP	Depression		Hazardous Use	- .03	[- .07, .02]
HOP	Anxiety		Hazardous Use	- .01	[- .04, .01]
HOP	**Depression**	**DCWD**	**Hazardous Use**	**.02**	**[.01, .04]***
HOP	Anxiety	DCWD	Hazardous Use	.01	[.00, .02]*
HOP	Depression	DCWA	Hazardous Use	.02	[.01, .04]*
HOP	Anxiety	DCWA	Hazardous Use	.02	[.01, .07]*
AS	Depression		Drinking Harms	.02	[- .01, .08]
AS	Anxiety		Drinking Harms	.02	[- .01, .09]
AS	Depression	DCWD	Drinking Harms	.02	[.02, .05]*
AS	Anxiety	DCWD	Drinking Harms	.01	[.01, .04]*
AS	Depression	DCWA	Drinking Harms	.01	[.01, .02]*
AS	**Anxiety**	**DCWA**	**Drinking Harms**	**.01**	**[.01, .04]***
HOP	Depression		Drinking Harms	.04	[- .02, .16]
HOP	Anxiety		Drinking Harms	.03	[- .01, .10]
HOP	**Depression**	**DCWD**	**Drinking Harms**	**.04**	**[.04, .10]***
HOP	Anxiety	DCWD	Drinking Harms	.01	[.01, .04]*
HOP	Depression	DCWA	Drinking Harms	.02	[.01, .05]*
HOP	Anxiety	DCWA	Drinking Harms	.02	[.02, .05]*

Note. AS is Anxiety Sensitivity; HOP is Hopelessness. DCWD is Drinking to Cope with Depression; DCWA is Drinking to Cope with Anxiety. Bolded pathways were theorized. Asterisks represent significance at $p < .05$.

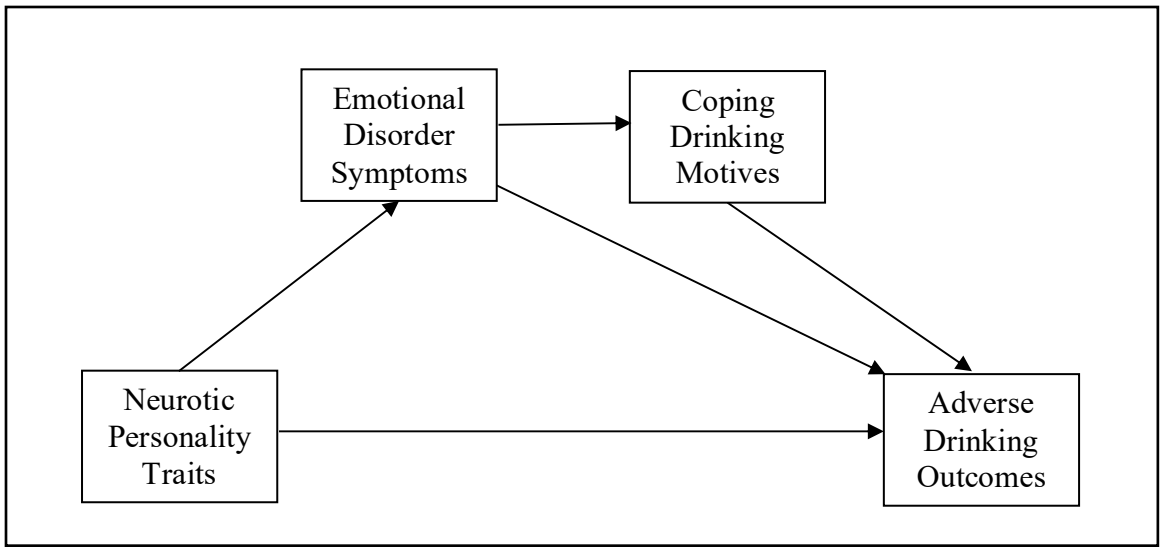

Figure 2.1. Chained mediation model from personality to adverse drinking outcomes, through emotional disorder symptoms and coping drinking motives. Rectangles represent measured variables; arrows represent paths.

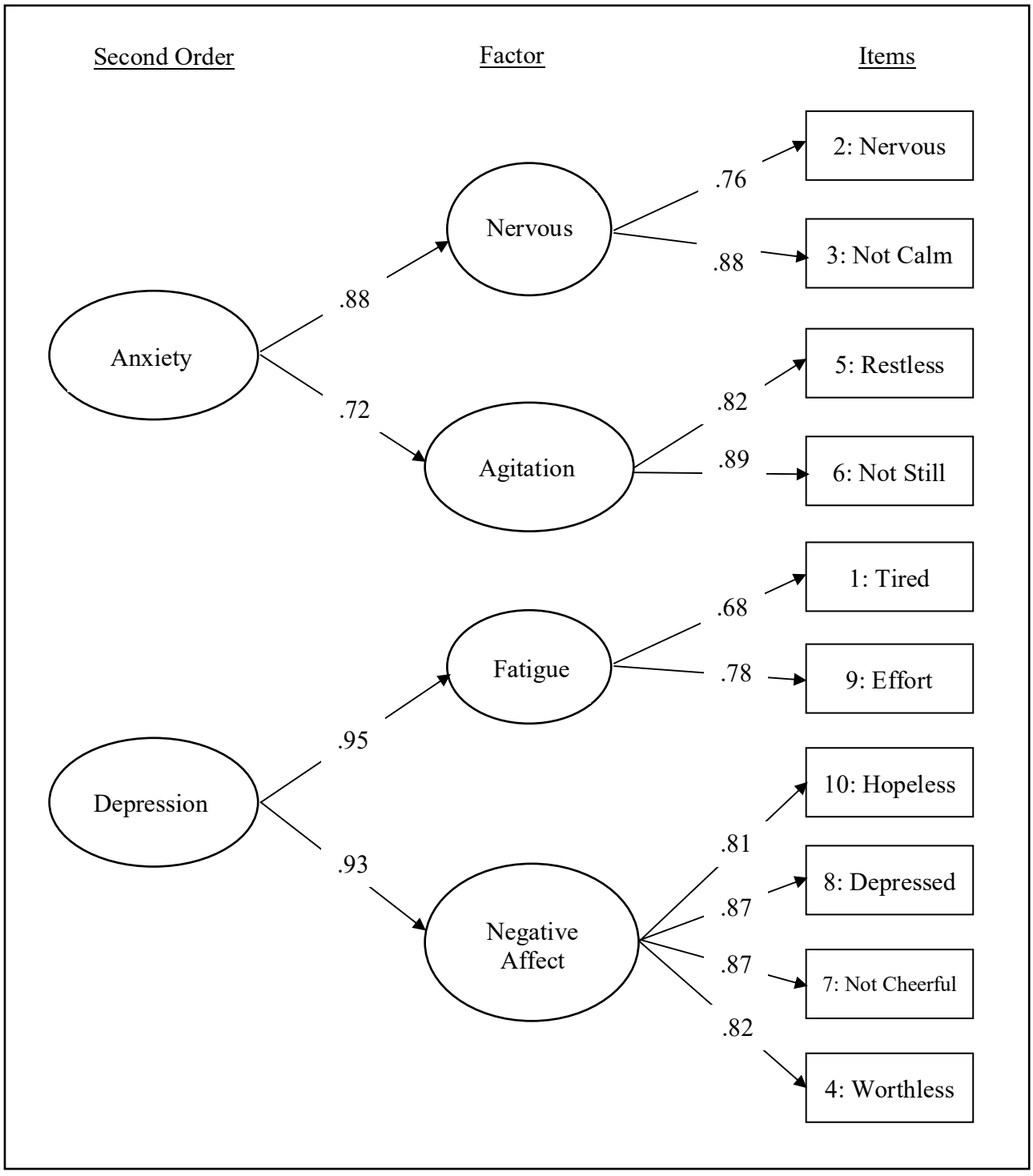

Figure 2.2. Path model of the confirmatory factor analysis of the K10's (Kessler *et al.*, 2002) second-order factor structure. Rectangles represent measured variables; ovals represent latent variables. Single-headed arrows represent paths. All pathways were significant at $p < .001$.

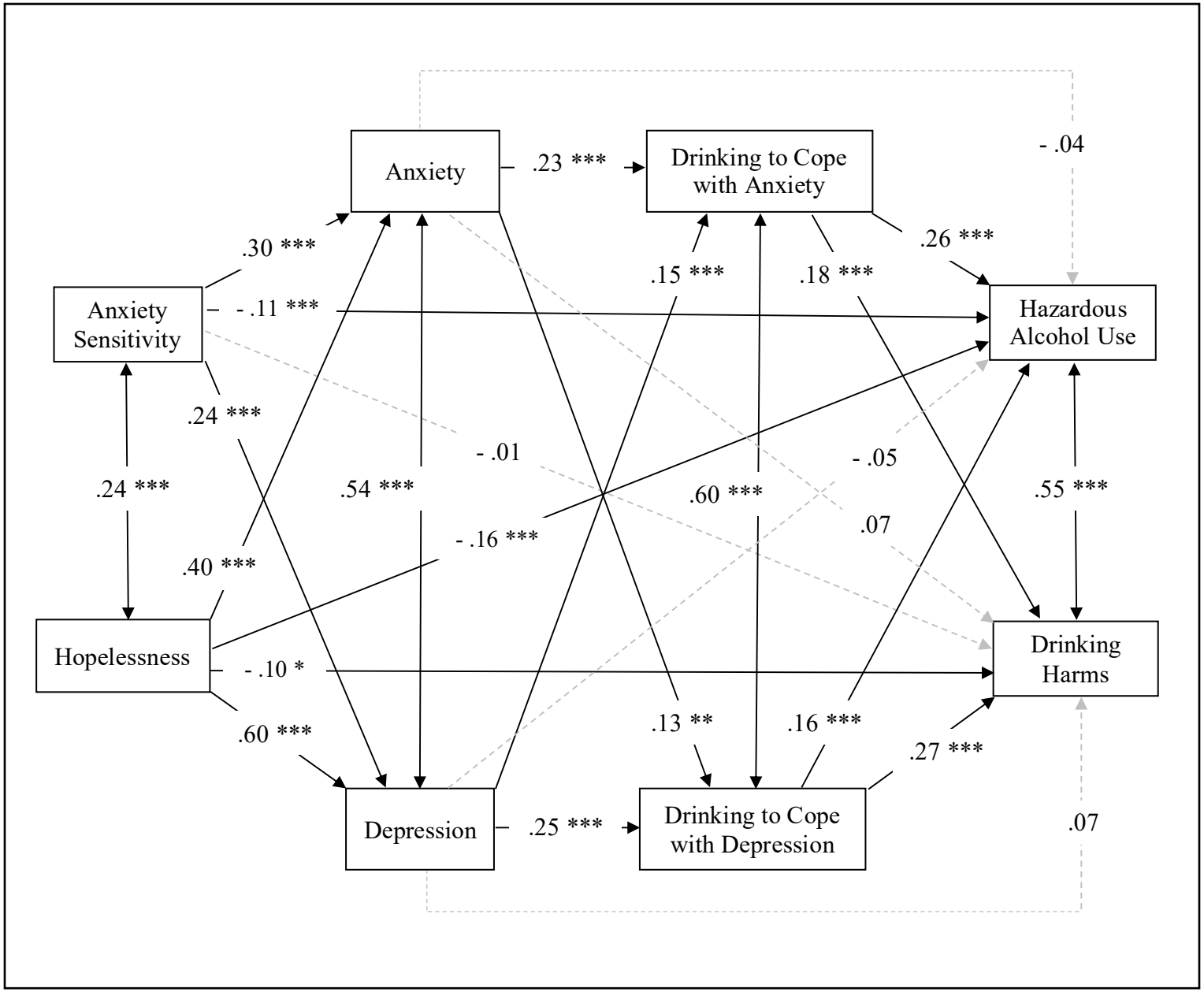
Anxiety
.23 ***
Drinking to Cope with Anxiety
- .04
.30 ***
.15 ***
.18 ***
.26 ***
Anxiety Sensitivity
- .11 ***
Hazardous Alcohol Use
.24 ***
- .01
- .05
.24 ***
.54 ***
.60 ***
.55 ***
.07
- .16 ***
.40 ***
Hopelessness
- .10 *
Drinking Harms
.60 ***
.13 **
.16 ***
.27 ***
Depression
.25 ***
Drinking to Cope with Depression
.07

Figure 2.3. Chained mediation of neurotic personality traits to (1) hazardous alcohol use and (2) drinking harms, through emotional disorder symptoms and coping drinking motives. Solid arrows represent statistically significant pathways; dotted arrows represent non-significant pathways. Numbers represent standardized coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$.

CHAPTER 3. TRANSITION FROM STUDY 1 TO 2

As established in the introductions to this dissertation (Chapter 1) and to Study 1 (Chapter 2), emerging adults endorse the high levels of alcohol use and misuse. This is particularly true of 18-25 year-olds (Arnett, 2000) who attend university (ACHA, 2016). Nearly 80% of undergraduates drink alcohol (Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2015). Heavy drinking is also normative in university (Skidmore, Kaufman, & Crowell, 2016). For example, most students cite "getting drunk" as their primary reason for drinking (Jessor, Costa, Krueger, & Turbin, 2006). Because alcohol is legal, its consumption is often regarded as "safe" (van Amsterdam, Opperhuizen, Koeter, & van den Brink, 2010). This perception exists despite the fact that alcohol is the largest contributor to student morbidity and mortality (Borsari, Murphy, & Barnett, 2007). Finally, Kypri & Langley (2015) concluded that most students overestimate the incidence of heavy drinking among their peers. The extent of one's overestimation also strongly predicts one's own heavy drinking frequency (Kypri & Langley, 2015), presumably because students feel pressured to emulate perceived drinking norms.

Alcohol is considered by researchers, university staff, and university students alike as the substance most pervasively misused on campus (Perkins, 2002). Because alcohol is consistently cited as a problem (for emerging adults and, particularly, university students), it has been studied more extensively than other substances (Skidmore *et al.*, 2016). Recent reports, however, suggest that "the defining drug problem of the new century will be the nonmedical use of prescribed controlled substances" (DuPont, 2011; p. 127). Currently, in North America, PD misuse has reached epidemic proportions (Hernandez & Nelson, 2017). PDs' sedative, analgesic, anxiolytic, anesthetic, or stimulant properties have led to a penchant for abuse and a state of emergency.

Rates of PD misuse are highest in Canada and the U.S. compared to low, middle, and other high-income countries (Holmes, 2012). Canada, for instance, has the highest per capita rates of prescription opiate use and misuse (INCB, 2015). It is ranked second for benzodiazepine use and within the top 15 for prescription stimulant use (INCB, 2004). In North America, PD misuse is now the second highest category of non-medical drug use (after cannabis; SAMHSA, 2012). In both Canada and the U.S., prescription opioid-related deaths are a leading cause of premature adult mortality (McCabe & Teter, 2007). Compared to other psychoactive drugs, the burden of PD misuse is exceeded only by alcohol and tobacco abuse in these countries (Fischer & Argento, 2012). In the U.S., for example, poisoning-related deaths (90% of which are caused by drug overdose) have become the leading cause of death by injury (above motor vehicle accidents; Warner, Chen, Makuc, Anderson, & Minino, 2011). In addition to increased mortality, PD misuse is associated with morbidity and other harms.

Physiological harms of PD misuse include an increased risk of negative drug interactions, withdrawal, physical dependence, organ damage, cardiovascular risk, injury related to intranasal use, and other health risks (Hartung *et al.*, 2013; Holloway *et al.*, 2014; McCabe & Teter, 2007; Teter *et al.*, 2010). Between 2004 and 2009, the number of American emergency room visits involving PD misuse increased by 98% (Holloway *et al.*, 2014). Benzodiazepine misuse has been identified as a specific risk factor for overdose, particularly if they are taken while drinking (Sergeev, Karpets, Sarang, & Tikhonov, 2003). Mixing opiates and alcohol can also be fatal (Coffin *et al.*, 2003). In fact, the insurgence of opioid-misuse related deaths has prompted the development of naloxone education and distributions programs, which teach at-risk users and bystanders

how to prevent, recognize, and respond to an overdose (Walley *et al.*, 2013). Psychological harms of PD misuse include psychological dependence, and symptoms of general distress, anxiety, and depression (Cohen, 1992; Holloway *et al.*, 2014). PD misuse is one of the most common problems for young people enrolled in drug treatment programs, attesting to their inherent risk for dependence (Gonzales *et al.*, 2011). Social harms include a lower grade point average, family problems, interpersonal consequences, misuse of other drugs, polysubstance abuse, engagement in other risky behaviours, and engagement in illegal activities (Brandt *et al.*, 2014; Garnier-Dykstra, Caldeira, Vincent, O'Grady, & Arria, 2012; Hartung *et al.*, 2013; Holloway *et al.*, 2014; McCabe & Teter, 2007). PD misuse also has substantial negative economic impacts. The cost of prescription opioid misuse alone, in the U.S., is in the tens of billions of dollars (Birnbaum *et al.*, 2011).

Of any age group, emerging adults are the mostly likely to take PDs non-medically. Nearly one-third of 18-25-year-olds endorse lifetime PD misuse (DuPont, 2011). High and rising rates have also been reported by university students (Arria *et al.*, 2008a; Babcock & Byrne, 2000; Teter, McCabe, Boyd, & Guthrie, 2003). Like alcohol, undergraduates tend to view PDs as non-harmful and widely misused. Because PDs have been approved for medical use and can be accessed more safely, their misuse is thought to be more socially acceptable than that of illicit drugs (Friedman, 2006; Hernandez & Nelson, 2017; McCabe & Boyd, 2005). Most undergraduates also overestimate the prevalence of opioid (70%) and stimulant (70%) misuse on campus (McCabe, 2008). Actual on-campus estimates peak at 22% for opioids and 17% for stimulants (Holloway *et al.*, 2014). Arria, Caldeira, Vincent, O'Grady, & Wish (2008b) concluded that students

with low perceived PD harmfulness were 10 times more likely to engage in non-medical PD use, compared to those with high perceived harmfulness. Again, despite student perceptions to the contrary, PD misuse is associated with acute adverse effects, risk for dependence, and contraindications (Arria *et al.*, 2008b).

While PD misuse is well-documented and has caused public concern, it is a relatively new field of study (Arria *et al.*, 2008b). Early work has focused on PD diversion sources, routes of administration, and motives (McCabe, Boyd, & Cranford, 2009a; McCabe *et al.*, 2009b; McCabe, Teter, & Boyd, 2006). But researchers have yet to examine the processes that underlie young people's propensity for PD misuse. In Studies 2 and 3 of this dissertation, I will apply models that have been robustly studied with and validated for alcohol misuse and illicit drug use to the emerging issues of PD involvement. Models like the one tested in Study 1 (i.e., that incorporate personality, mental health symptoms, and substance use motives) predict hazardous alcohol use and drinking harms. But PDs are fundamentally different than other abuseable substances because they are designed for specific purposes, have obvious health benefits when taken as prescribed, and can be "misused" in a myriad of ways (Barrett *et al.*, 2008; Compton & Volkow, 2006).

As a first step, Study 2 will examine the continued applicability of Pihl & Peterson's (1995) four-factor model of personality vulnerability to addiction. Non-medical PD misuse encompasses a variety of drug classes, each with different purported reasons for use and consequences (Arria *et al.*, 2008b). Thus, I will test the following theory-driven paths: AS to sedatives/tranquilizers, HOP to opioids, SS to stimulants, and IMP to all three (i.e., to unconstrained PD-taking). I will also explore whether these

personality-to-specific PD class paths apply to freshmen's overall PD use, medically-sanctioned use, and/or misuse.

CHAPTER 4. STUDY 2: PERSONALITY AND PRESCRIPTION DRUG USE/MISUSE AMONG FIRST YEAR UNDERGRADUATES

The manuscript prepared for this study is presented below. Readers are advised that Annie Chinneck, under the supervision of Dr. Sherry Stewart, was responsible for developing the research questions and hypotheses, preparing the dataset for analyses, conducting some analyses, and interpreting the study findings. Annie wrote the initial draft of the manuscript; she received and incorporated feedback from her co-authors. The manuscript underwent peer review. Annie led the response to revisions. The manuscript was accepted to Addictive behaviours on July 2, 2018. See Appendix I for copyright permission from the publisher (Elsevier). The full reference is as follows:

Chinneck, A., Thompson, K., Mahu, I. T., Davis-MacNevin, P., Dobson, K., & Stewart, S. H. (2018). Personality and prescription drug use/misuse among first year undergraduates. *Addictive Behaviours, 87,* 122-130.

Abstract

Emerging adults (18-25 year-olds) endorse the highest rates of prescription drug misuse. Attending college or university may confer additional risk. Previous research suggests that personality is an important predictor of many addictive behaviours. Four traits have been consistently implicated: anxiety sensitivity, hopelessness, sensation seeking, and impulsivity. Published studies on personality as a predictor of prescription drug abuse are limited, however, by their primary focus on overall prescription drug use, inconsistent operationalisation of misuse, and failure to control for alcohol use. Sample sizes have been small and non-specific. We sought to better understand how personality predicted the overall use, the medically-sanctioned use, and the misuse of prescription sedatives/tranquilizers, opioids, and stimulants. A large (N = 1,755) sample of first year Canadian undergraduate students (mean age = 18.6 years; 69% female) was used. We predicted that: anxiety sensitivity would be related to sedatives/tranquilizers, hopelessness to opioids, sensation seeking to stimulants, and impulsivity to all three. Save for the impulsivity to opioid use path, predictions were fully supported in our "any use" model. For medically-sanctioned use: anxiety sensitivity predicted sedative/tranquilizers, hopelessness predicted opioids, and impulsivity predicted stimulants. For misuse: anxiety sensitivity (marginally) predicted sedatives/tranquilizers, sensation seeking predicted stimulants, and impulsivity predicted all three. Our models support using personality-matched interventions. Specifically, results suggest targeting anxiety sensitivity for sedative/tranquilizer misuse, sensation seeking for stimulant misuse, and impulsivity for unconstrained prescription drug misuse. Interventions with

early coping skills that pertain to all four traits might be useful for preventing prescription drug uptake and later misuse.

Keywords: undergraduate students; personality vulnerability; prescription drug use; medically-sanctioned prescription drug use; prescription drug misuse.

Introduction

PDs are misused when they are taken without a physician's prescription, in greater amounts or more often than prescribed, via non-intended routes, for non-prescribed reasons, and/or with contraindicated substances (Haydon *et al.*, 2006). Physiological harms of PD misuse include increased risk of negative drug interactions, withdrawal, physical dependence, injury related to intranasal use, organ damage, cardiovascular risk, accidental overdose, and death (Hartung *et al.*, 2013; Holloway *et al.*, 2014; Teter *et al.*, 2010). Psychological harms include psychological dependence, distress, depression, and anxiety (Cohen, 1992; Holloway *et al.*, 2014). Social harms include antisocial behaviour, academic issues, family problems, and interpersonal issues (Brandt *et al.*, 2014; Hartung *et al.*, 2013; Holloway *et al.*, 2014). Despite these consequences, young people continue to misuse PDs at an alarming rate. Reported lifetime PD misuse rates among American university students, for example, have exceeded 50% (McCabe *et al.*, 2006).

Personality as a Predictor

Pihl & Peterson (1995) developed a model, upon which Castellanos-Ryan & Conrod (2012) elaborated, that outlines four substance misuse vulnerabilities. It is well-supported in the literature (e.g., Castellanos-Ryan & Conrod, 2012; Mackinnon *et al.*, 2014). The first two traits are internalizing. They are characterized by internal processes, constraint, inhibition, and over-control. AS is the fear of anxiety-related sensations, due to the unrealistic expectation that these sensations will have catastrophic consequences (e.g., physical illness, social embarrassment, or loss of control; Reiss, 1991; Taylor, 2014). HOP is expecting aversive events, and not expecting desirable ones (Abramson *et*

al., 1989). Both AS and HOP are associated with coping motives (Woicik *et al.*, 2009). Undergraduates who are high in these traits tend to self-medicate with depressants (Conrod *et al.*, 2000a; Woicik *et al.*, 2009). Specifically, among treatment-seeking adult substance abusers, AS predicts anxiolytic dependence and HOP predicts opioid dependence (Conrod *et al.*, 2000a).

The remaining two traits are externalizing. They are characterized by external actions, lack of constraint, disinhibition, and under-control. SS is the desire for novel experiences (Zuckerman, 1994). Individuals high in SS are sensitive to the rewarding properties of substances (Castellanos-Ryan & Conrod, 2012). In undergraduates, SS is associated with illicit and prescription stimulant misuse (Low & Gendaszek, 2002). IMP is the tendency to act without careful deliberation (Dawe & Loxton, 2004). It is associated with a motivationally undefined pattern of substance use, whereby availability predicts misuse (Hecimovic *et al.*, 2014). Related deficits in response inhibition mean that young people who are high in IMP are more susceptible to early experimentation and to later, compulsive use (Castellanos-Ryan *et al.*, 2014). These students tend to engage in heavier, unconstrained drug use (Woicik *et al.*, 2009).

Student Misusers

Emerging adults (Arnett, 2000) endorse the highest PD misuse rates. Nearly 15% of 18-25 year-old undergraduates report past-year PD abuse (Silvestri *et al.*, 2015). Between 1989 and 2002, these rates increased from 7% to 22% (SAMHSA, 2003). Further, most North American emerging adults are enrolled in university. In the Youth in Transition Survey, 81% of participating Canadians aged 26-28 years had attended a post-secondary institution (Shaienks & Gluszynski, 2009). This is important, as university

represents a time of heightened risk for PD misuse. Students are under academic strain and are facing multiple simultaneous stressors (e.g., pressure to succeed, competition with peers, financial strain, and concerns about the future). They are also nearing the age of onset for use of many drugs of abuse (Holloway *et al.*, 2014; Tavolacci *et al.*, 2013). McCabe, West, Morales, Cranford, & Boyd (2007), for example, identified the following as the mean ages of onset for lifetime PD misuse: 18.9 for stimulants (*Mdn* = 18, *SE* = 0.1); 22.7 for tranquilizers (*Mdn* = 20, *SE* = 0.3); 23.1 for sedatives (*Mdn* = 30, *SE* = 0.4); and 23.2 for opioids (*Mdn* = 20, *SE* = 0.3).

Quintero, Peterson, & Young (2006) interviewed 52 PD misusers. Many associated studenthood with drug abuse; they acknowledged that they could not misuse PDs in the same way or at the same rate post-graduation. Thus, university may represent a "time-out period", in which responsibility is suspended and experimentation encouraged (Côté & Allahar, 1996). Respondents further indicated that PD misuse was socially acceptable. Unlike illicit drugs, they noted that PDs were government-approved, subjected to extensive laboratory testing, manufactured by professionals, advertised, known to produce dose-dependent effects, and associated with listed side effects. They categorized PDs as "soft" drugs (i.e., those facilitating pleasure and performance).

Quintero *et al.*'s students (2006) endorsed three PD misuse motives. First, they used PDs to self-medicate affective states and physical conditions (e.g., stress, pain, and being overweight). Second, they took PDs recreationally (e.g., to have fun or get high). Third, they used PDs to more effectively fulfill their role demands (e.g., to study, focus, or concentrate). Brandt *et al.* (2014) linked these motives to specific PD classes. Sedatives/tranquilizers are misused to relax, decrease another drug's side effects, or

increase its high. Opioids are misused to self-medicate pain or get high. Stimulants are misused to get high or as a study aid. A systematic review (Holloway *et al.*, 2014) listed undergraduate lifetime misuse rates as: 4-9% for sedatives/tranquilizers, 12-22% for opioids, and 2-17% for stimulants. Compared to pre-university, by second year university, lifetime misuse rises 103% for sedatives/tranquilizers, 86% for opioids, and 319% for stimulants (Arria *et al.*, 2008a).

The Present Study

The only study to examine how the four-factor personality model (Castellanos-Ryan & Conrod, 2012; Pihl & Peterson, 1995) predicted undergraduate PD use was conducted by Woicik *et al.* (2009). AS was associated with depressant use, SS with stimulant and polysubstance use, and IMP with stimulant use. The present study extends Woicik *et al.*'s (2009) in several ways.

First, Woicik *et al.* (2009) broadly examined any PD "use". To address this limitation, we examined relations between personality and: (1) any PD use, (2) medically-sanctioned PD use, and (3) PD misuse. Students most commonly misuse sedatives/tranquilizers, opioids, and stimulants (Colliver, Kroutil, Dai, & Gfroerer, 2006). Personality may differentially predispose a student to either take or misuse a given PD class. As such, we also compared these three drug classes. Second, Woicik *et al.* (2009) failed to include an important covariate: alcohol use. University students tend to use alcohol with other substances, including PDs (McCabe, West, Schepis, & Teter, 2015). To address this limitation, our models control for alcohol dependence. Third, Woicik *et al.*'s (2009) study was underpowered ($N = 162$). They had to combine sedatives, tranquilizers, and opioids to form a single depressant drug category. This may have

obscured specific, theorized personality-to-PD paths. For example, Woicik *et al.* (2009) found that AS predicted depressant use but HOP did not. However, using a clinical sample of 293 substance misusers, Conrod *et al.* (2000a) substantiated the specific AS-to-anxiolytic dependence and HOP-to-opioid dependence pathways. Using a sample of 22,783 undergraduates, Zullig & Divin (2012) further concluded that high-HOP students were 1.18-1.43 times more likely than others to use prescription opioids. To address this limitation of Woicik *et al.*'s (2009; i.e., to increase power), we used a large sample of undergraduates. Finally, Woicik *et al.* (2009) sampled all undergraduates, regardless of year of study. First year represents a time of particular vulnerability. Freshmen have moved away from home, have lost important social networks, and are under new academic strain (Holloway *et al.*, 2014). Compared to freshmen, more senior students have lower odds of past-year non-medical PD use (Lanier & Farley, 2011). To address this limitation, our sample was restricted to freshmen.

Hypotheses. The current study predicted that: AS would be related to sedative/tranquilizer use, HOP to opioid use, SS to stimulant use, and IMP to use of all three. See Figure 4.1 for our hypothesized model. Barrett *et al.* (2008) suggest that the PD literature is limited by a lack of consensus about what constitutes misuse and by the inconsistent use of operational criteria. Thus, in addition to our Any Use model, we also ran Medically-Sanctioned Use and Misuse models. In so doing, we hoped to extend the extant research by examining whether personality was similarly related to all three operationalizations. We believe that any use, medically-sanctioned use, and misuse are all clinically-important constructs. Any PD use carries risk (Cohen, 2001) and use tends to

precede misuse (Maisto, Galizio, & Connors, 1991). This second research question (i.e., comparing use vs. medically-sanctioned use vs. misuse) was exploratory in nature.

Methods

This study used data from Phase I of the Movember-funded Caring Campus Project (Stuart *et al.*, In press), which involved surveying Canadian undergraduates. The present study is archival, using data collected mid-Winter 2015 (Cohort 1) and mid-Fall 2015 (Cohort 2) at a single site.

Participants

A total of 1,755 freshmen from a mid-sized Eastern Canadian university participated (Cohort 1 n = 714, Cohort 2 n = 1,041). Students ranged in age from 17-25 years (M = 18.59, SD = 1.07). They were predominantly female (69%), Canadian (91%), in a Science program (40%), living in residence (58%), and unemployed (74%). Average disposable income was $59.94 per week ($SD$ = $170.22); students spent a mean of $15.67 per week on alcohol/drugs (SD = $17.18). Average self-reported GPA was in the B range (M = 1.90, SD = 0.78). See Table 4.1 for remaining sample demographic information. Most variables did not differ between Cohorts 1 and 2. Between-group ANOVAs were significant for any sedative/tranquilizer use ($F(1, 1573) = 4.00$, $p < .05$); any opioid use ($F(1, 1301) = 7.45$, $p < .01$); and medically-sanctioned opioid use ($F(1, 1301) = 4.30$, $p < .05$). Sedative/tranquilizer rates were higher in Cohort 2; opioid rates were higher in Cohort 1.

Procedure

Ethical approval was granted by the university's Research Ethics Board. All freshmen were emailed a link to the survey. Secondary recruitment strategies included

on-campus posters and social media advertising. Participation was voluntary. Participants received modest compensation (a $5.00 CDN gift card or partial course credit). Response rates were 32% for Cohort 1 and 38% for Cohort 2. These rates are comparable to other Canadian undergraduate surveys (e.g., Thompson *et al.*, 2017a). They are higher than the national average (i.e., 19%; ACHA, 2016).

Measures

Personality (see Appendix C). The 23-item SURPS (Woicik *et al.*, 2009) was used to assess personality. It has four subscales: AS (5 items; "It is frightening to feel dizzy or faint"), HOP (7 items; "I feel that I'm a failure"), SS (6 items; "I like doing things that frighten me a little"), and IMP (5 items; "I usually act without stopping to think"). Participants responded using a 5-point Likert scale (1 *strongly disagree* to 5 *strongly agree*). Following any reverse scoring, subscale scores were generated by summing component items. The SURPS has good internal consistency, test-retest reliability, and factorial validity (Castellanos-Ryan *et al.*, 2013). In our sample, the subscales were internally consistent (AS $\alpha = .84$, HOP $\alpha = .93$, SS $\alpha = .87$, and IMP $\alpha = .85$).

PD involvement (see Appendix J). A screener question asked: "Have you used any PDs at all this term?" If the participant said "yes", they were asked a series of more specific questions. First, they were asked how often they had taken sedatives/tranquilizers, opioids, and/or stimulants that term. To deal with zero-inflation, these questions were scored dichotomously (i.e., 1 if the student *had used that PD class* and 0 if they *had not*). They were operationalized as "Any Use".

If the participant reported using a PD class, they were presented with a list of ways they might have taken it. The following options were presented; respondents were asked to check all that applied. (1) As prescribed by my doctor to treat a medical condition; (2) have a prescription but sometimes do not use it as prescribed; (3) without a prescription to treat a medical condition; (4) take PDs and then drink alcohol; (5) to get high; and (6) as a study aid.

For each class, Medically-Sanctioned Use was operationally defined as checking "as prescribed by my doctor" (#1). This variable was scored dichotomously (i.e., 1 if the student *had used that PD class appropriately* and 0 if they *had not*). If any of the remaining list items (#2-6) were checked, the student was assigned a 0. In other words, Medically-Sanctioned Use meant *exclusively* using a PD as prescribed.

Finally, for each class, a Misuse score was calculated. Respondents were given a 1 if they endorsed *any* of the remaining list items (#2-6). Of note, "as a study aid" (#6) is misuse. Even prescribed stimulants (e.g., Adderall and Ritalin) are meant to be taken regularly (Svetlov, Kobeissy, & Gold, 2007). Peterkin, Crone, Sheridan, & Wise (2011) found that 87% of college ADHD stimulant misusers do so for academic reasons. This variable was scored dichotomously (i.e., 1 if the student *had misused that PD class* and 0 if they *had not)*.

Alcohol dependence (see Appendix F). The 10-item AUDIT (Saunders *et al.*, 1993) was used to assess alcohol dependence. There are a number of ways to score and interpret the AUDIT (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). The dependence score is calculated by summing questions 4-6, which ask about impaired control over drinking, increased salience of drinking, and morning drinking. Participants

responded using a 5-point Likert scale (0 *never* to 4 *daily or almost daily*). A subtotal of 4-12 on these questions suggests the possibility of alcohol dependence. As such, this variable was dichotomized (i.e., 1 if the student was *dependent on alcohol* and 0 if they *were not*). The AUDIT has been validly and reliably used to assess undergraduates' alcohol use and misuse (O'Hare & Sherrer, 1999). It accurately detects alcohol dependence in university students (Fleming, Barry, & MacDonald, 1991).

Statistical Analyses

Frequencies were calculated in SPSS 20.0. Multivariate path models (where all specified paths are estimated simultaneously) were run in MPlus 7.11 (Muthén & Muthén, 1998-2012). Because our PD outcomes were categorical, they were modeled using the default probit regression and Bayesian estimation (ESTIMATOR = BAYES). Bayesian estimators are best when the dependent variables are dichotomous and inter-correlated (Muthén, 2010). Because Bayesian models do not produce standard fit statistics, posterior predictive *p*-values (PPP) are reported (Gelman, 2013). PPP are legitimate and informative probability statements (Chambert, Rotella, & Diggs, 2014; Gelman & Shalizi, 2013). PPP > .05 indicates good fit; PPP = .5 indicates excellent fit (Gelman *et al.*, 2014). Because most between-group ANOVAs were non-significant, cohorts were combined and Cohort was controlled. Because university students tend to use alcohol with other substances, including PDs (McCabe *et al.*, 2015), Alcohol Dependence (as measured by the AUDIT; Babor *et al.*, 2001) was also controlled. Finally, we controlled for Age and Gender, given their known effects on PD use (Boyd, Cranford, & McCabe, 2016). All of these covariates were regressed onto all of the outcome variables. See Appendix H (Table H.2) for a supplementary correlation table

that includes all of the covariates and other variables of interest. Outcomes are reported as standardized regression estimates.

Results

Opioids were used most frequently; most opioid use was medically-sanctioned. The most commonly endorsed misuse category was "without a prescription" (1% of the total sample; 44% of misusers). Stimulants were used second most frequently; most stimulant use constituted misuse. The most commonly endorsed misuse category was "as a study aid" (4% of the total sample; 88% of misusers). Sedatives/tranquilizers were used least frequently; most sedative/tranquilizer use was medically-sanctioned. The most commonly endorsed misuse category was "not as prescribed" (1% of the total sample; 40% of misusers). See Table 4.2 for descriptive statistics and Table 4.3 for the correlation matrix.

Model 1 (Figure 4.2) tested our Any Use hypotheses. This model had good fit (PPP = .29). As predicted, AS was significantly associated with sedatives/tranquilizers, HOP with opioids, and SS with stimulants. IMP predicted sedative/tranquilizer and stimulant use – but not opioid use. Model 2 (Figure 4.3) explored Medically-Sanctioned Use. This model had good fit (PPP = .42). AS was significantly associated with appropriate sedative/tranquilizer use, HOP with appropriate opioid use, and IMP with appropriate stimulant use. Model 3 (Figure 4.4) explored Misuse. This model had good fit (PPP = .39). SS was significantly associated with stimulant misuse, and IMP with unconstrained PD misuse. AS marginally (p = .05) predicted sedative/tranquilizer misuse.

Of note, preliminary models did not control for alcohol dependence. The models described in-text and depicted in Figures 4.2-4.4 did. Differences between the first set of

results (i.e., not controlling for alcohol dependence) and the second set of results (i.e., controlling for alcohol dependence) were as follows. Once alcohol dependence was co-varied: (1) HOP to any opioid use became non-significant, (2) IMP to medically-sanctioned stimulant use became significant, and (3) AS to sedative/tranquilizer misuse became marginally significant ($p = .05$). The significance of all other paths remained the same, regardless of whether or not alcohol dependence was controlled.

Discussion

We examined how the four-factor personality model (Castellanos-Ryan & Conrod, 2012; Pihl & Peterson, 1995) predicted freshmen's PD use. Theory predicted the following pathways: AS to sedatives/tranquilizers, HOP to opioids, SS to stimulants, and IMP to all three. Opioids were used (both overall and appropriately) most frequently. This is consistent with the current North American prescription opioid use crisis (Fischer *et al.*, 2014). Stimulants were misused most frequently. This is also consistent with previous studies (McCabe *et al.*, 2006).

Any PD Use

All of our hypothesized personality to PD pathways were significant, save for IMP to opioid use. This is consistent with previous research supporting AS, HOP, SS, and IMP as predictors of substance use (Woicik *et al.*, 2009). University students tend to concurrently use opioids and alcohol (Garnier *et al.*, 2009) and IMP is a strong predictor of polysubstance use (Moody *et al.*, 2016). It is therefore unsurprising that controlling for alcohol dependence negated the IMP to opioid use effect[1].

Medically-Sanctioned PD Use

The following pathways were significant: AS to sedatives/tranquilizers, HOP to opioids, and IMP to stimulants. Sedatives, tranquilizers, opioids, and stimulants are used to treat a number of psychiatric illnesses and sequelae (Olfson *et al.*, 2013a, 2013b). Sedatives/tranquilizers are prescribed for anxiety (Bisaga & Mariani, 2015) and AS is a risk factor for anxiety (McLaughlin *et al.*, 2007). Stimulants are used to manage ADHD (Solanto, Arnsten, & Castellanos, 2001), for which IMP is a prominent symptom (APA, 2013; Barkley, Murphy, & Fischer, 2008). We know that personality leads to substance use via mental health symptoms and motives (Allan *et al.*, 2014; Chinneck *et al.*, 2018a). As such, future research should see if mental health symptoms (e.g., anxiety or ADHD) mediate these paths.

Previous research supports a link between HOP and opioid dependence (Conrod *et al.*, 2000). The path from HOP to *medically-sanctioned* opioid use, however, is less intuitive. HOP predicts the presence of pain and pain severity (Yildirim, Sertoz, Uyar, Fadiloglu, & Uslu, 2009). Thus, pain could mediate this relationship (making it medication). Previous research supports a link between HOP and opioid dependence (Conrod *et al.*, 2000). The path from HOP to *medically-sanctioned* opioid use, however, is less intuitive. HOP predicts the presence of pain and pain severity (Yildirim, Sertoz, Uyar, Fadiloglu, & Uslu, 2009). Thus, pain could mediate this relationship (making the association between HOP and medically-sanctioned opioid use explained by medication for pain). HOP is also a precursor for depression (Joiner, 2000). Opioids are not generally prescribed to treat depressive symptoms, but chronic pain and depression tend to co-occur (Sheng, Liu, Wang, Cui, & Zhang, 2017). In their review, Amari, Rehm, Goldner,

and Fischer (2011) found that pain prevalence was elevated among non-medical opioid users (15-62% in general, treatment, and street drug-using samples). They further concluded that depression was the mental health issue most strongly related to non-medical prescription opioid use (odds ratios ranged from 1.2 to 4.3). Thus, depression could also mediate this relationship (making the association between HOP and medically-sanctioned opioid use explained by self-medication for depression). Finally, future research should examine chained mediation from pain, to HOP, to depression, to opioid misuse. While physical pain disorder is the most obvious diagnosis, it might also be useful to identify the other conditions for which opioids are being prescribed.

The remaining externalizing pathways (i.e., SS to stimulants, IMP to sedatives/tranquilizers, and IMP to opioids) were non-significant. SS is not pathological; it is not included in the diagnostic criteria of any disorder for which stimulants are prescribed (APA, 2013). Further, SS has been linked to substance misuse in the absence of other forms of psychopathology (Conrod *et al.*, 2000b). Finally, sedatives, tranquilizers and opioids are not prescribed for high-IMP disorders (e.g., disruptive, impulse-control, or conduct disorders; APA, 2013).

PD Misuse

The following pathways were significant: SS to stimulants and IMP to sedatives/tranquilizers, opioids, and stimulants. SS is associated with enhancement motives for substance use (e.g., Comeau *et al.*, 2001). Because they seek excitement (Zuckerman, 1994), high-SS students preferentially misuse stimulants. IMP is associated with severity of substance dependence (Conrod *et al.*, 2000a). It involves the inability to control one's behaviour in the face of immediate reward (Arnett, 1994). This explains

why our high-IMP students were unconstrained PD users; they have trouble controlling their behaviour in the face any immediately reinforcing PD drug effects (Moody *et al.*, 2016). Taken together, these results suggest that students high in SS are seeking to alter a particular affective state, while those high in IMP are struggling to inhibit their behaviour.

The AS to sedative/tranquilizer misuse pathway was marginally significant. This suggests that students who are high in AS are self-medicating their anxiety-related sensations by taking these PDs without a physician's prescription, in greater amounts or more often than prescribed, via non-intended routes, and/or with contraindicated substances (Haydon *et al.*, 2006). This finding, while marginal, is worth mentioning. It is consistent with both previous studies (Conrod *et al.*, 2000a) and cited theory (Khantzian, 1997).

The HOP to opioid misuse pathway was non-significant. This may have been due to our sample's generally low endorsement of past-term misuse or to overwhelming IMP effects. PD misusers tend to abuse multiple drug classes. Stimulant misusers, for example, are more likely to endorse concurrent opioid use (compared to appropriate stimulant users; Hartung *et al.*, 2013). Over 23% of PD misusers take stimulants with opioids (Chen, Crum, Starin, Martins, & Mojtabai, 2015). In our freshmen sample, IMP predicted sedative/tranquilizer, opioid, *and* stimulant misuse. Previous research (e.g., Messina *et al.*, 2014) supports the robustness of these relationships. Thus, IMP may have accounted for most of the variance for personality-related risk of opioid misuse. It is also possible that the relationships between our covariates (e.g., age, gender, and/or alcohol dependence) and IMP and/or opioid misuse are suppressing this effect. Because greater

power allows for detection of smaller effects, future studies might also benefit from even larger sample sizes (e.g., a multisite study). While this would increase the chances of a small effect becoming significant, it would not increase the size of our effect, which was very small. We also recommend re-examining these relationships with upper-class undergraduates. HOP predicted any use and medically-sanctioned use. Thus, its associations with misuse may emerge over time.

Limitations

First, our models were cross-sectional. Previous research suggests that personality is a determinant of future drug use (Hengartner, Tyrer, Adjacic-Gross, Angst, & Rössler, 2018). In the absence of longitudinal data, however, we cannot rule out bidirectional effects. It is also possible that personality change is a consequence of PD use. De Wit (2009), for example, found that drug abuse increased IMP. Second, we used self-report measures. The reliability and stability of self-reported drug use is purportedly high (O'Malley, Bachman, & Johnston, 2009). But Smith *et al.*'s (2015b) concluded that no published instruments can adequately assess inappropriate medication use events (e.g., PD abuse or misuse) or context of use. Given these measurement issues, we cannot be certain that our participants responded accurately. Third, our PD outcomes were scored dichotomously. Questions were either asked in a yes/no format or too skewed for appropriate modelling of variability in frequency of use. In order to better examine personality's effect on quantity and frequency of PD use, future research should take advantage of selective sampling and continuous variables. Fourth, observed effect sizes were modest. PD use vs. misuse is a complex phenomenon (Barrett *et al.*, 2008). There are many contributing factors, and personality is only one determinant. To account for a

larger proportion of variance, follow-up studies could examine how individual and environmental factors interact to predict the various forms and types of PD-taking. Fifth, we did not examine personality's relationship with PD use motives. It would be interesting to test if personality predicts: (1) self-medication vs. social-recreational use (Quintero *et al.*, 2006) or (2) coping, enhancement, conformity, and social substance use motives (Cooper *et al.*, 2016) as adapted to PDs. Sixth, our cohorts were assessed at different time points. The survey was administered mid-winter and mid-fall and asked about the current semester. We controlled for Cohort, but this could have influenced participants' use rates and response tendencies. Finally, we did not ask about ethnicity or socioeconomic status. As such, we cannot be certain that our sample is representative of all Canadian undergraduates. In addition to addressing these limitations, future research could examine more specific facets of the personality traits tested herein. The Anxiety Sensitivity Index-3 (Taylor *et al.*, 2007), for example, measures physical, cognitive, and social AS concerns. Barratt's Impulsiveness Scale-11 (Patton *et al.*, 1995) measures attentional, motor, and non-planning forms of IMP.

Conclusion

Previous research on the relationships between personality and PD use in university students (e.g., Woicik *et al.*, 2009) was limited by its primary focus on overall PD use, inconsistent operationalisation of misuse, and failure to control for alcohol use. Sample sizes were small and non-specific. We sought to clarify the effects of personality on PD-taking in a large sample of first year undergraduates (N = 1,755). Save for the IMP-to-opioid use path, our personality-to-Any Use predictions were fully supported. To explore how students were taking PDs and how personality was implicated, we ran

separate Medically-Sanctioned Use and Misuse models. In the former, AS, HOP, and IMP were significant predictors. In the latter, AS, SS, and IMP were significant predictors.

Our findings have important clinical implications. Risk for onset of substance use and related problems is heightened while in university (Tavolacci *et al.*, 2013). Substance use during this period and at these ages is associated with academic difficulty, interpersonal violence, high-risk sexual behaviour, immediate health problems, and mental illness (Hingson, Zha, & Weitzman, 2009; Parks, Collins, & Derrick, 2012; Squeglia *et al.*, 2012). Compared to other age groups, young adults are the most likely to abuse PDs and to be seen in emergency rooms for related symptoms (Okie, 2010). Voltmer, Kötter & Spahn (2012) concluded that while the health and wellbeing of university students is decreasing, their risk patterns are increasing. They highlighted the need for prevention and health promotion initiatives that focused on students' individual behaviour. Our findings support targeting personality vulnerability, in particular.

AS predicted the medically-sanctioned use and misuse of sedatives/tranquilizers. HOP predicted using opioids as prescribed by a doctor to treat a medical condition. Since taking these medications entails risk (Cohen, 2001), preventing the uptake of both use and misuse would be beneficial. Our models suggest that sedative, tranquilizer, and opioid use could be prevented by treating precursors for anxiety and depression. Stimulant misuse and unconstrained PD misuse, on the other hand, were linked to SS and IMP, respectively. Stimulant misuse could be prevented by offering stimulating, prosocial activities to high-SS students. PD misuse, generally, could be prevented by

promoting future-oriented, non-impulsive thinking on campus. These strategies are consistent with empirically supported addiction therapies (Herie & Watkin-Merek, 2006).

Brief, selected, skills-based interventions (Watt, Stewart, Birch, & Bernier, 2006) are also effective in reducing illicit drug use in adolescents and adults (Conrod, Castellanos-Ryan, & Strang, 2010; Conrod *et al.*, 2000b) and PD misuse in adults (Conrod *et al.*, 2000b). Personality-matched interventions are manualized and include psychoeducational, motivational, and cognitive-behavioural components. They are designed to change the way that vulnerable users cope (Conrod *et al.*, 2010). Our models support using these interventions to curb university students' PD use and misuse. Specifically, sedative/tranquilizer misuse could be treated with an AS-specific protocol, stimulant misuse with a SS one, and unconstrained PD misuse with an IMP one. Follow-up trials might also examine if HOP-matched intervention prevents uptake of opioids. If yes, this protocol could be used to pre-emptively build coping skills among high school students.

Table 4.1. Sample Demographic Information.

N = 1,755	%
Gender	
Female	68.9
Male	31.0
Non-binary	0.1
International	
Canadian	90.5
International	9.5
Program	
Science	39.9
Arts & Social Sciences	17.5
Health Professions	12.4
Engineering	9.5
Management	8.2
Other	12.5
Living arrangement	
In residence	58.2
With family	24.6
With roommates	15.0
On own	2.2
Work	
No	73.6
Yes	26.4
	M (SD)
Age (in years)	18.59 (1.07)
Grade point average (1 *A* to 5 *F*)	1.90 (0.78)
Weekly disposable income (in CAD)	59.94 (170.22)
Weekly money spent on alcohol/drugs (in CAD)	15.67 (17.18)

Table 4.2. Descriptive Statistics.

	Any Use		Medically-Sanctioned Use			Misuse		
N = 1,755	*n*	%	*n*	%	% of Use	*n*	%	% of Use
Sedatives/Tranquilizers	69	4.4	39	2.5	56.52	30	1.9	43.48
Opioids	128	9.9	92	7.1	71.88	36	2.8	28.12
Stimulants	107	6.8	22	1.4	20.56	85	5.4	79.44
	M (SD)							
Anxiety Sensitivity	12.85 (2.74)							
Hopelessness	13.42 (3.63)							
Sensation Seeking	16.47 (3.52)							
Impulsivity	10.54 (2.55)							
Alcohol Dependence	0.07 (0.25)							

Note. Personality was assessed using the SURPS (Woicik *et al.*, 2009). Subscale ranges are as follows: 5-25 for Anxiety Sensitivity, 7-25 for Hopelessness, 6-30 for Sensation Seeking, and 5-25 for Impulsivity. Alcohol Dependence was assessed using the AUDIT (Saunders *et al.*, 1993) and scored dichotomously.

Table 4.3. Correlation Matrix.

N = 1,755	2	3	4	5	6	7	8	9	10	11	12	13	14
Sedatives/Tranquilizers													
1. Any	**.74**	**.65**	**.16**	**.11**	**.12**	**.25**	**.13**	**.21**	**.11**	**.12**	.01	**.10**	**.12**
2. Medically-Sanctioned	1	-.02	**.07**	**.10**	-.03	**.15**	**.12**	**.11**	**.10**	**.08**	-.03	.05	.06
3. Misuse		1	**.16**	.04	**.22**	**.20**	**.06**	**.19**	**.06**	**.10**	.04	**.10**	**.11**
Opioids													
4. Any			1	**.84**	**.51**	**.11**	-.01	**.13**	**.08**	**.09**	-.03	**.06**	**.07**
5. Medically-Sanctioned				1	-.05	-.002	-.03	.13	**.07**	**.07**	-.05	-.003	.04
6. Misuse					1	**.20**	.03	**.21**	.03	.05	.02	**.10**	**.07**
Stimulants													
7. Any						1	**.44**	**.89**	.05	**.09**	**.14**	**.17**	**.24**
8. Medically-Sanctioned							1	-.03	**.06**	.03	.003	**.05**	.05
9. Misuse								1	.03	**.08**	**.15**	**.17**	**.24**
Personality													
10. Anxiety Sensitivity									1	**.22**	**-.21**	**.15**	.02
11. Hopelessness										1	**-.10**	**.21**	**.08**
12. Sensation Seeking											1	**.29**	**.15**
13. Impulsivity												1	**.25**
Covariate													
14. Alcohol Dependence													1

Note. Bold correlations are significant at $p < .05$.

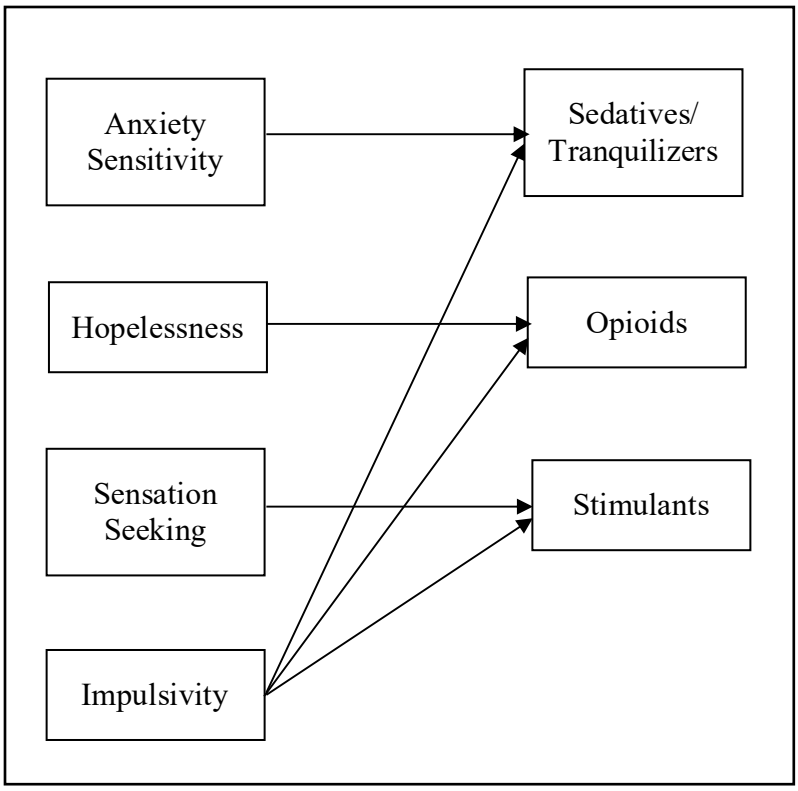

Figure 4.1. Hypothesized models. Rectangles represent observed variables. Single-headed arrows represent paths.

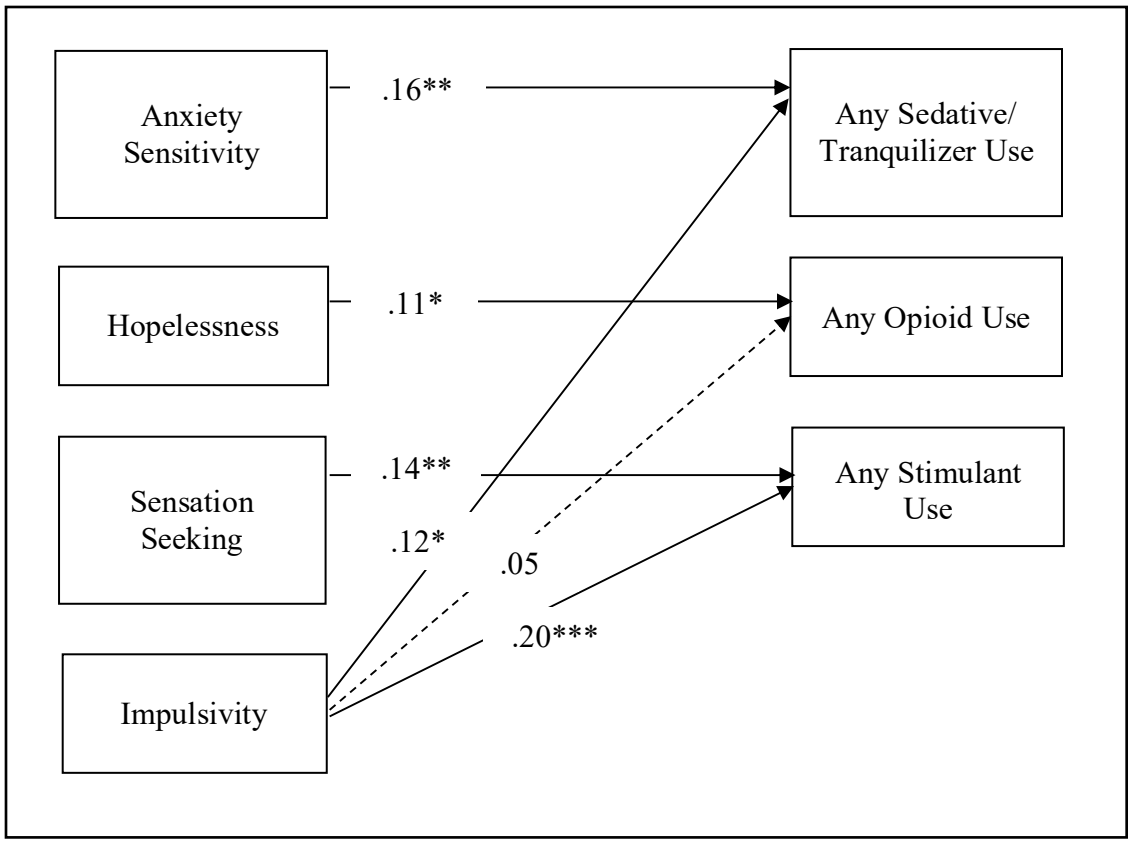

Figure 4.2. Model 1: Any PD use. Rectangles represent observed variables. Solid arrows represent statistically significant pathways. Numbers represent standardized coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$.

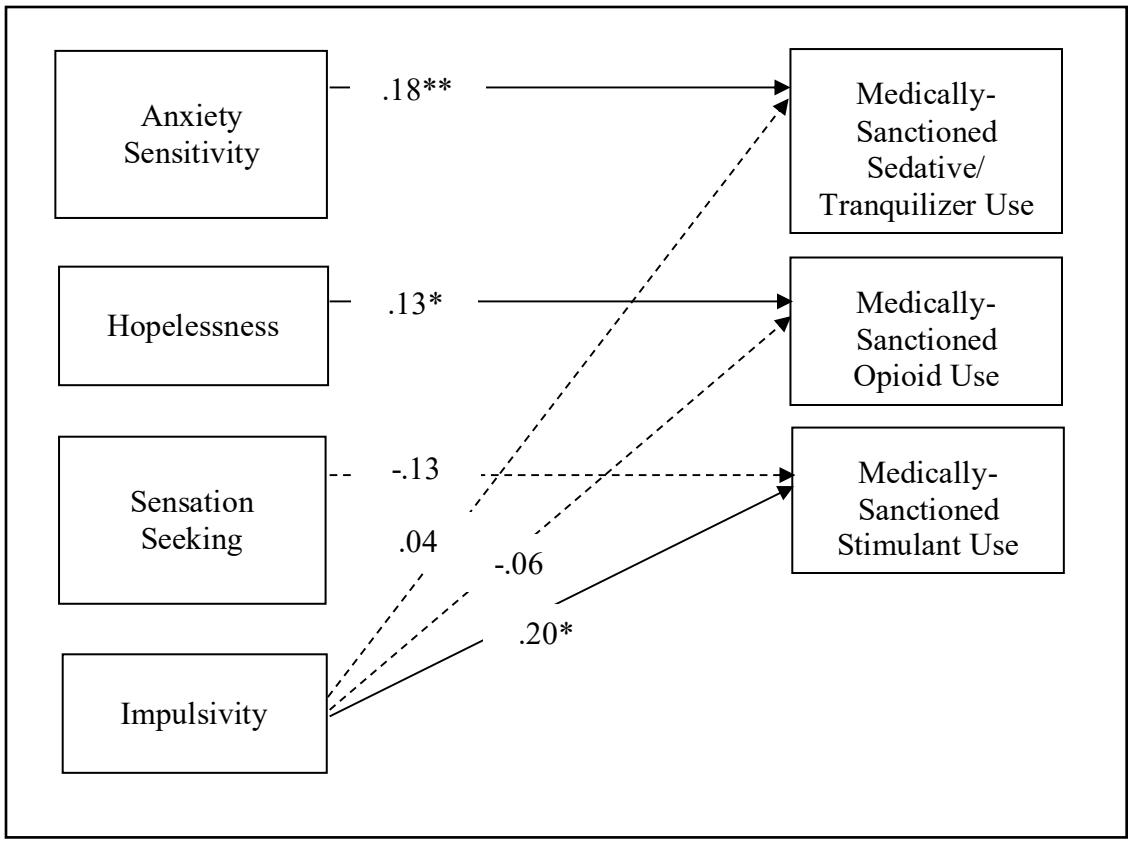

Figure 4.3. Model 2: Medically-sanctioned PD use. Rectangles represent observed variables. Solid arrows represent statistically significant pathways; dotted arrows represent non-significant pathways. Numbers represent standardized coefficients. * $p < .05$, ** $p < .01$.

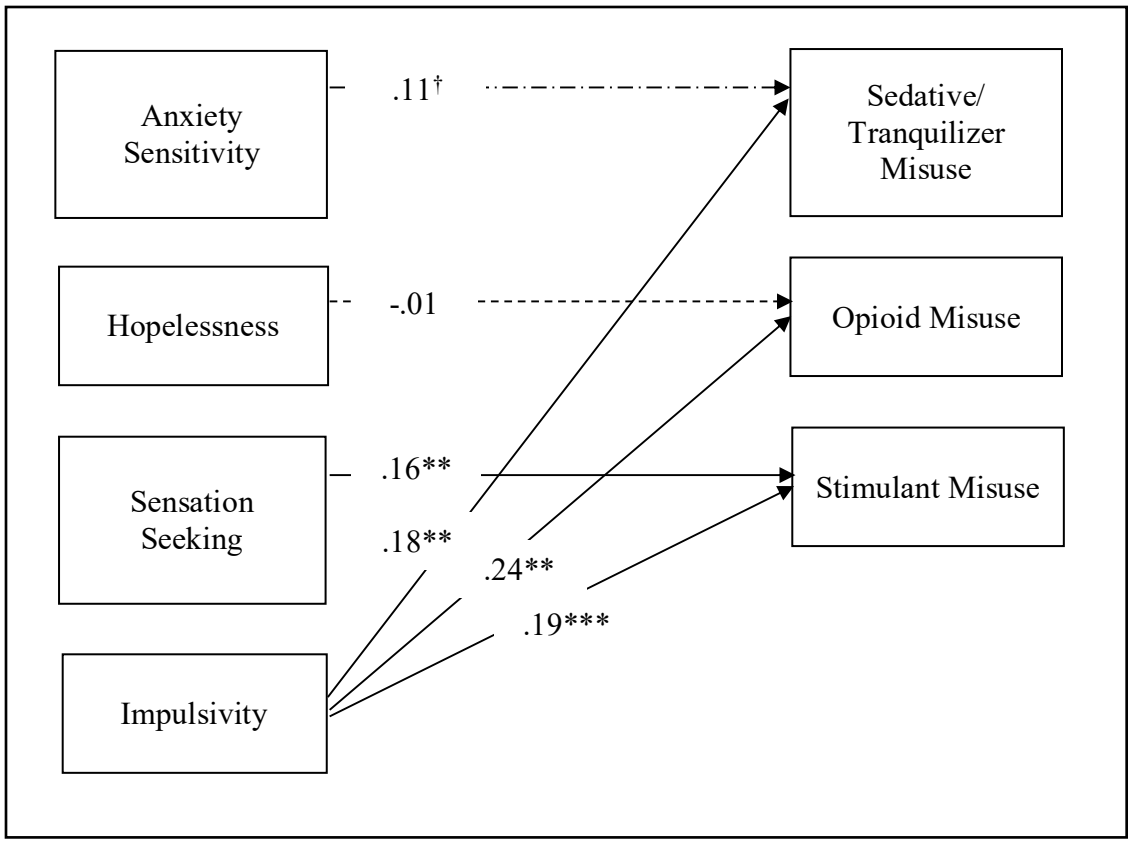

Figure 4.4. Model 3: PD misuse. Rectangles represent observed variables. Solid arrows represent statistically significant pathways; dotted arrows represent non-significant pathways. Numbers represent standardized coefficients. ** $p < .01$, *** $p < .001$. † represents marginal significance at $p = .05$.

CHAPTER 5. TRANSITION FROM STUDY 2 TO 3

Study 2 (Chapter 4) applied Pihl & Peterson's (1995) four-factor model of personality vulnerability to three forms of PD-taking: overall use, medically-sanctioned use, and misuse. Consistent with theory and hypotheses, each trait predicted the misuse of a specific class of PDs. Namely, AS (marginally) predicted sedative/tranquilizer misuse; SS predicted stimulant misuse; and IMP predicted sedative/tranquilizer, opioid, and stimulant misuse (i.e., unconstrained PD misuse). HOP also predicted general use of opioids. Study 1 (Chapter 2) showed that personality's effect on alcohol misuse was mediated by mental health symptoms and related (i.e., coping) drinking motives. There is evidence to suggest that mental health symptoms might similarly mediate my observed relations between personality and PD misuse. In young people, personality is a robust predictor of both internalizing and externalizing mental health symptoms (Adan *et al.*, 2016; Smith *et al.*, 2007). Mental health symptoms are further associated with youths' PD misuse. Generally, teens who struggle with acute psychiatric issues are at an increased risk of non-medical PD misuse (Armstrong & Costello, 2002; Brooks, Harris, Thrall, & Woods, 2002).

Internalizing symptoms, specifically, are associated with PD misuse. Experience sampling showed that dysphoria predicted PD misuse in daily life (Papp & Kouros, 2017). S. L. Stewart, Baiden, & den Dummen's (2013) multivariate model substantiated the following PD misuse predictors: having multiple psychiatric admissions, history of emotional abuse, threat or danger to self, and symptoms of depression. A recent latent class analysis (Kelly, Rendina, Vuolo, Wells, & Parsons, 2015) delineated four types of PD misusers: dabblers, primary downers, primary stimulants, and extensive regulars. The "extensive regulars" class reported the most dependence symptoms, PD misuse-related

problems, and mental health symptoms (i.e., anxiety, depression, and somatization). The "dabblers" class reported the fewest. Finally, those in the "primary downers" class reported more symptoms and problems than those in the "primary stimulants" one. Kelly *et al.* (2015) did not assess externalizing symptoms. Kenne *et al.* (2017) went on to suggest that young people who self-medicate emotional pain with PDs were too embarrassed to seek treatment. This, they argued, was due to the stigmatization of mental health (Corrigan *et al.*, 2014).

Boyd, McCabe, Cranford, & Young (2006) and Teter, McCabe, Cranford, Boyd, & Guthrie (2005) suggest that some youth are motivated to misuse prescription medications to self-medicate, while others engage in misuse to get high, experiment, or improve performance. Externalizing symptoms, specifically, are also associated with PD misuse. Conduct problems predict unconstrained PD misuse (Khoddam & Leventhal, 2016). They are related to opioid misuse (Morioka, Howard, Caldeira, Wang, & Arria, 2018), stimulant misuse (Van Eck *et al.*, 2012), and the diversion of PDs (Garnier *et al.*, 2010). After controlling for prescribed use, ADHD symptoms are still associated with stimulant misuse (Van Eck *et al.*, 2012).

As stated previously, my dissertation sought to apply models that have been robustly studied with and validated for alcohol misuse and other illicit drug use – to the emerging issue of PD misuse. Study 3 will therefore examine the mediating effects of mental health symptoms on the relationships between personality and the misuse of various classes of PDs. Specifically, I will test the following theory-driven paths: AS to anxiety to sedative/tranquilizer misuse; HOP to depression to opioid misuse; IMP to ADHD to stimulant misuse; and IMP to CD to sedative/tranquilizer, opioid, and stimulant

misuse (i.e., unconstrained PD misuse). My multivariate model will also include direct paths from SS to stimulant misuse and from IMP to sedatives/tranquilizer, opioid, and stimulant misuse.

Of note, in Study 2, HOP predicted opioid use but not misuse. The high-HOP users could have been taking opioids to medicate pain *or* to self-medicate depressive symptoms. Pain and depression tend to co-occur (Sheng *et al.*, 2017). However, because opioids are not prescribed for depression (Singh & Reece, 2014), taking them to cope with related symptoms would constitute misuse (Haydon *et al.*, 2006). Teter *et al.* (2010) and Zullig & Divin (2012) previously linked HOP to PD misuse. I am suggesting that this is because HOP predicts taking your own or someone else's opioids for a non-prescribed, non-pain-related reason. To confirm this hypothesis, a HOP to depressive symptoms to opioid misuse pathway was included in Study 3's model. Thus, an added rational for Study 3 is to gain increased clarify of Study 2's results.

Finally, while Studies 1 and 2 sampled emerging adults, Study 3 focuses on adolescents. After 18-25 year-olds, PD misuse is second highest among 12-17 year-olds (Cotto *et al.*, 2010; Maxwell, 2011). Half of Grade 7-12 students do not believe that PD misuse is risky and one third do not think PDs are addictive (Partnership for a Drug-Free America, 2009). S. L. Stewart *et al.* (2013) suggest that adolescent PD misuse rates are increasing in Canada. One in seven Ontarian students in Grades 7-12 now report past-year non-medical PD misuse. PD misuse also increases progressively with age and grade level (Paglia-Boak, Adlaf, & Mann, 2011). Thus, to determine early risk factors and to best inform prevention efforts, Study 3 sampled Grade 9 and 10 students. Across Canada, "high school" consistently includes Grades 9-12. As described in Studies 1 and 2, risk

increases as emerging adults transition from high school to university (Holloway *et al.*, 2014; Lanier & Farley, 2011). Risk similarly increases as adolescents transition from middle to high school (McIntosh, Flannery, Sugai, Braun, & Cochrane, 2008). Thus, Grade 9 represents another period of increased vulnerability for substance misuse.

CHAPTER 6. STUDY 3: PERSONALITY TO PRESCRIPTION DRUG MISUSE IN ADOLESCENTS: TESTING AFFECT REGULATION, PSYCHOLOGICAL DYSREGULATION, AND DEVIANCE PRONENESS PATHWAYS

The manuscript prepared for this study is presented below. Readers are advised that Annie Chinneck, under the supervision of Dr. Sherry Stewart, was responsible for developing the research questions and hypotheses, preparing the dataset for analyses, conducting some of the analyses, and interpreting the study findings. Annie wrote the initial draft of the manuscript; she received and incorporated feedback from her co-authors. Annie has submitted the manuscript to a special issue of *Journal of Consulting and Clinical Psychology* for peer review. Her abstract was pre-approved for their "Responding to the Opioid Crisis: Perspectives, Challenges, and Directions" call for papers. See Appendix K for copyright permission from all co-authors. The current reference is as follows:

Chinneck, A., Thompson, K., Conrod, P. J., Afzali, M. H., Nogueira-Arjona, R., Mahu, I. T., & Stewart, S. H. (Paper submitted July 2018). Personality to prescription drug misuse in adolescents: Testing affect regulation, psychological dysregulation, and deviance proneness pathways. *Journal of Consulting and Clinical Psychology*.

Abstract

Access to prescription drugs, including opioid medications, is at an all-time high. Of any age group, 15-25 year-olds are the most likely to misuse prescription drugs. Few studies have tested theoretical models of adolescent risk for prescription drug misuse, generally or by drug class. As such, we tested theory-driven mediational paths from personality to mental health to prescription drug misuse. Our hypotheses were informed by etiological models of addiction (i.e., affect regulation, psychological dysregulation, deviance proneness). We used semi-longitudinal data collected during the Co-Venture Trial. Our sample included students from 31 Canadian high schools. They were tested in Grade 9 (September 2014-May 2015; n = 3,024; mean age = 14.79) and again in Grade 10 (September 2015-May 2016; n = 2,869; mean age = 15.83). Gender composition was 51% male; students were predominantly middle-class. Personality (hopelessness, anxiety sensitivity, impulsivity, sensation seeking) was assessed in Grade 9. Mental health symptoms (depression, anxiety, attention-deficit hyperactivity disorder, conduct disorder) and prescription drug misuse (opioids, sedatives/tranquilizers, stimulants) were assessed at both time points. Consistent with negative affect regulation: hopelessness predicted opioid misuse via depressive symptoms and anxiety sensitivity predicted sedative/tranquilizer misuse via anxiety symptoms. Consistent with positive affect regulation: sensation seeking marginally predicted stimulant misuse directly. Consistent with psychological dysregulation: impulsivity predicted stimulant misuse via attention-deficit hyperactivity disorder symptoms. Consistent with deviance proneness: impulsivity predicted unconstrained prescription drug misuse via conduct disorder symptoms. Identifying youth high in personality risk may benefit targeted prevention and early

intervention efforts. Personality-matched cognitive-behavioural interventions may reduce their risk for prescription drug misuse.

Keywords: adolescents; prescription drug misuse; self-medication; psychological dysregulation; deviance proneness.

Introduction

The National Survey on Drug Use and Mental Health defines PD misuse as "use … without a prescription belonging to the respondent, or use that occurred simply for the experience or feeling the drug caused" (SAMHSA, 2008). PDs are federally reviewed and regulated. As such, many young people consider them to be less harmful than illicit drugs (Manchikanti, 2006). However, due to their potency, potential for addiction, and the ease with which overdoses can occur, PD misuse can be injurious or fatal (Levine, 2007). PD misuse accounts for more emergency room visits than all illicit substances combined (SAMHSA, 2012). Since 1999, opioid overdoses have quadrupled (Wide-Ranging Online Data for Epidemiologic Research, 2016). From 2002-2010, the number of people undergoing treatment for PD misuse doubled (SAMHSA, 2011). PD misuse is a risk factor for future dependence (McCabe *et al.*, 2007) and has been linked to North America's increasing heroin use rates (Muhuri, Gfroerer, & Davies, 2013).

Access to PDs is at an all-time high. Opioids are increasingly prescribed to manage pain, and stimulants to manage ADHD (Manchikanti, Fellows, Ailinani, & Pampati, 2010). Of any age group, 15-25 year-olds are the most likely to misuse PDs (SAMHSA, 2015). From 1992-2003, PD misuse increased 212% among Americans aged 12-17 years (National Center on Addiction and Substance Abuse at Columbia University, 2005). PDs are the drug most commonly abused by North American adolescents, after marijuana (SAMHSA, 2015). Over 8% of youth endorse past-year PD misuse and 3% report symptoms of a PD-related substance use disorder (Schepis & Krishnan-Sarin, 2008). This is clinically significant as early-onset PD misuse is related to worse sequelae

(McCabe *et al.*, 2007) and to future substance-related problems and disorders (Milner, Ham, & Zamboanga, 2014).

Previous research has established that the following factors differentiate adolescent PD misusers from non-misusers. Factors that protect against adolescent PD misuse include high parental involvement, parental disapproval of substance use, and religious beliefs (Nargiso, Ballard, & Skeer, 2015). Factors that increase the risk for adolescent PD misuse include greater access to PDs, academic issues, friends who misuse substances, favourable substance use attitudes, other substance use, antisocial behaviour, mental health symptoms, and psychiatric admissions (Nargiso *et al.*, 2015). Boyd *et al.* (2006) suggest that the demographic, behavioural, and social correlates of PD misuse may vary considerably by drug class (i.e., for opioids vs. sedatives/tranquilizers vs. stimulants). Moreover, few studies have tested theoretical models of adolescent risk for PD misuse (Ford, Reckdenwald, & Marquardt, 2014).

Personality as Risk Factor

Personality is a robust predictor of adolescent addictive behaviour. Internalizing and externalizing traits, for example, have been reliably associated with an increased susceptibility for illicit substance misuse in youth (Conrod, 2016). Pihl & Peterson (1995) developed a model, upon which Castellanos-Ryan & Conrod (2012) elaborated, that delineates four such traits. The first two are internalizing. HOP is expecting aversive events but not desirable ones (Abramson *et al.*, 1989). AS is the fear of anxiety-related sensations, due to an unrealistic expectation that they will have catastrophic consequences (e.g., physical illness, social embarrassment, loss of control; Taylor, 2014). In adolescents, both HOP and AS are associated with coping motives (Comeau *et al.*,

2001). Young people high in these traits tend to preferentially misuse depressant drugs (Conrod *et al.*, 2000a; Woicik *et al.*, 2009). In adults, HOP predicts opioid dependence and AS predicts anxiolytic dependence (Conrod *et al.*, 2000a). The remaining two traits are externalizing. IMP is the tendency to act without forethought (Dawe & Loxton, 2004). IMP has been associated with a motivationally-undefined pattern (Hecimovic *et al.*, 2014) of polysubstance use (Moody *et al.*, 2016). Related deficits in response inhibition make high-IMP teens more susceptible to early experimentation and later, compulsive use (Castellanos-Ryan *et al.*, 2014). SS is the desire for stimulation (Zuckerman, 1994). High-SS users are sensitive to the rewarding properties of drugs (Castellanos-Ryan & Conrod, 2012). Thus, they tend to misuse stimulants (Herman-Stahl, Krebs, Kroutil, & Heller, 2006) to study, stay awake, improve alertness, get high, party, and experiment (Yudko, Lozhkina, & Fouts, 2007).

Pihl & Peterson's (1995) four-factor personality vulnerability model has been used to predict: adolescent alcohol use (Stewart, McGonnell, Wekerle, & Adlaf, 2011), adolescent illicit drug use (Castellanos-Ryan *et al.*, 2013), young adult PD use (Woicik *et al.*, 2009), and adult PD use (Conrod *et al.*, 2000a). It has yet to be applied to adolescent PD misuse. Are AS, HOP, SS, and IMP risk factors at this age, for these drugs? Castellanos-Ryan & Conrod (2012) outlined several etiological models of addiction. The affect regulation, psychological dysregulation, and deviance proneness models are reviewed, in turn, below. Personality's role in each is discussed and our associated hypotheses are introduced.

Affect Regulation Model

This model posits that individuals take drugs to regulate their emotions. There are two major sources of drug reinforcement (Woicik *et al.*, 2009): negative (i.e., a drug's ability to relieve negative affect) and positive (i.e., a drug's hedonic effects). Negative affect regulation would be taking a PD to avoid/control negative feelings (e.g., to relieve stress, depressed mood, or anxiety). Positive affect regulation would be taking a PD to feel good (e.g., for pleasure, stimulation, or euphoria). This dichotomy is in keeping with McCabe *et al.*'s (2009a) motives work, which suggests that youth misuse PDs to self-medicate or for recreational purposes.

A Paths: Negative Affect Regulation and Self-Medication

PDs are prescribed to treat a number of psychiatric disorders (Olfson *et al.*, 2013a, 2013b). This practice is complicated, however, by contraindications, unpleasant side effects, and adverse drug interactions (Cohen, 2001). The liability of PD misuse and dependence (Martins *et al.*, 2012) may be due, in part, to the stigmatization of mental health, the misconception that PDs are safe, and the ease with which they can be obtained (Lo, Monge, Howell, & Cheng, 2013). Self-medication motives tend to be consistent with therapeutic indications (Boyd *et al.*, 2006). But taking PDs without a prescription, in greater amounts or more often than prescribed, via non-intended routes, for non-prescribed reasons, or with contraindicated substances is potentially harmful (Zosel, Bartelson, Bailey, Lowenstein, & Dart, 2013) and constitutes misuse (Haydon *et al.*, 2006).

***Hypothesis 1*: HOP to depressive symptoms to opioid misuse.** Adults high in HOP tend to misuse depressants, particularly opioids (Conrod *et al.*, 2000a; Woicik *et al.*,

2009). The following pathways have been substantiated in youth: HOP-to-depression and depression-to-opioid misuse. HOP predicts adolescent depression (Joiner, 2000) and depression itself increases risk of PD misuse (Ford *et al.*, 2014). Zullig & Divin (2012) found that students who endorsed HOP, depression, and suicidality were 1.18-1.43 times more likely to misuse opioids. No studies to date have tested a mediational HOP to depressive symptoms to opioid misuse pathway (*H1*) in adolescents.

***Hypothesis 2*: AS to anxiety symptoms to sedative/tranquilizer misuse.** Adults high in AS tend to misuse depressants, particularly anxiolytics (Conrod *et al.*, 2000a; Woicik *et al.*, 2009). The following pathways have been substantiated in youth: AS-to-anxiety and anxiety-to-sedative/tranquilizer misuse. Knapp, Blumenthal, Mischel, Badour, & Leen-Feldner (2015) found that AS was related to worry, anxiety, and GAD in 10-17 year-olds. McLaughlin *et al.* (2007) concluded that AS incrementally predicted anxiety disorder symptoms in youth, over and above state anxiety. GAD, agoraphobia, and panic disorder are associated with sedative/tranquilizer misuse/dependence (Becker, Fiellin, & Desai, 2007). No studies to date have tested a mediational AS to anxiety symptoms to sedative/tranquilizer misuse pathway (*H2*) in adolescents.

B Paths: Positive Affect Regulation and Recreation

Substances neuropharmacologically affect the brain centres involved in basic reward (Koob, 2000). In particular, stimulants activate mesolimbic dopamine activity (Seeman & Madras, 2002). For this reason, stimulants are often taken recreationally to increase positive mood (Cooper *et al.*, 1992).

***Hypothesis 3*: SS to stimulant misuse.** Individuals high in SS tend to misuse stimulants (Herman-Stahl *et al.*, 2006). SS is robustly related to positive reinforcement

and enhancement motives (Woicik *et al.*, 2009). High-SS youth are more sensitive to the rewarding properties of substances (Castellanos-Ryan & Conrod, 2012). Thus, SS could underlie sensitivity to stimulant reinforcement (Miller, Badger, Heil, Higgins, & Sigmon, 2015). *H3* predicts that SS will be directly related to adolescent stimulant misuse.

C Paths: Psychological Dysregulation Model

In this model (Thatcher & Clark, 2008), psychological dysregulation is defined as the cognitive, behavioural, or emotional inability to adapt to environmental challenges. Childhood cognitive dysfunction, irritability, and externalizing problems predict adolescent externalizing disorders and substance misuse (Clark *et al.*, 2005; Krueger *et al.*, 2002; Tarter *et al.*, 2003). IMP is also related to both problem behaviour and substance misuse (Castellanos-Ryan & Conrod, 2011). The relationship between IMP and externalizing behaviour is partially mediated by poor response inhibition (Castellanos-Ryan *et al.*, 2011). Moreover, IMP is heavily implicated in ADHD, an externalizing disorder that is characterized by persistent dysregulation interfering with functioning or development (APA, 2013). Youth with ADHD struggle with inhibitory control (Coutinho *et al.*, 2018). They are impulsive in social contexts, have difficulty perceiving their inadequate responses, and have trouble over-riding ongoing actions toward more appropriate ones. IMP's effect on substance misuse may be attributable, at least in part, to an inability to inhibit pre-potent responses (Logan, 1994). This is likely epitomized in youth with ADHD.

***Hypothesis 4*: IMP to ADHD symptoms to stimulant misuse.** The following pathways have been substantiated in youth: IMP-to-ADHD and ADHD-to-stimulant misuse. Individuals who are high in IMP (Conrod, 2016) or who have ADHD (Cassidy *et*

al., 2015) are more likely to misuse stimulants. Close to 4% of 10-18 year-olds endorse past-month stimulant misuse (Cottler, Striley, & Lasopa, 2013). But 14% of 4-17 year-olds with ADHD endorse past-two-week stimulant misuse (Sawyer *et al.*, 2017). Even after controlling for prescribed use, ADHD symptoms are associated with stimulant misuse (Van Eck *et al.*, 2012). The young people most likely to abuse prescription stimulants are those with markers of a possible mental health disorder but without a formal diagnosis or prescription (Arria & DuPont, 2010). No studies to date have tested a mediational IMP to ADHD symptoms to stimulant misuse pathway (*H4*) in adolescents.

D Paths: Deviance Proneness Model

This model (Sher & Slutske, 2003) categorizes substance use as a deviant behaviour. While deficient socialization is emphasized as the major risk factor in this model, personality is also thought to affect young people's substance use trajectories (Petraitis *et al.*, 1995). Of the four-factor traits, IMP has been most reliably implicated in deviant behaviour; it is associated with comorbid addictive and antisocial behaviours (Castellanos-Ryan & Conrod, 2011).

***Hypotheses 5-7*: IMP to CD symptoms to PD misuse.** The following pathways have been substantiated in youth: IMP-to-CD and CD-to-unconstrained PD misuse. IMP is the central characteristic of CD (L. L. Thompson, Whitmore, Raymond, & Crowley, 2006), while poor self-control and substance misuse are associated features (APA, 2013). López-Romero *et al.* (2015) collected prospective data from elementary school students. At baseline (i.e., ages 6-11), high IMP was associated with conduct problems. IMP also predicted students' developmental trajectories. It distinguished the Stable Low (i.e., non-problematic conduct) from the Stable High (i.e., early-onset and persistent conduct

problems). In adolescence, CD severity is associated with greater substance involvement (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Conduct problems also predict unconstrained PD misuse in adolescence (Khoddam & Leventhal, 2016) – specifically opioid misuse (Morioka *et al.*, 2018), stimulant misuse (Van Eck *et al.*, 2012), and diversion of PDs (Garnier *et al.*, 2010). No studies to date have tested these mediational IMP to CD symptoms to opioid (*H5*), sedative/tranquilizer (*H6*), and stimulant (*H7*) misuse pathways in adolescents.

The Present Study

Nargiso *et al.* (2015) reviewed the extant literature on adolescent PD misuse (N = 50 articles). They identified the following limitations. First, most studies were cross-sectional. Second, non-demographic factors (e.g., personality and mental health symptoms) were understudied. Third, there was a lack of specificity regarding patterns of misuse across PD classes. The present study sought to address these limitations by examining predictors of opioid, sedative/tranquilizer, and stimulant misuse in a large sample of Canadian adolescents, tested in Grade 9 and 10. Because adolescents who engage in high-intensity drinking are the mostly likely to co-ingest PDs (McCabe, Veliz, & Patrick, 2017), alcohol misuse was controlled.

Hypotheses. *H1-2* and *4-7* predicted that mental health symptoms would mediate personality's effect on PD misuse. These hypotheses were grounded in the negative affect regulation, psychological dysregulation, and deviance proneness models. *H3* predicted that SS would lead to stimulant misuse directly. This hypothesis was informed by the positive affect regulation model. Finally, it was hypothesized that IMP would lead to opioid (*H8*), sedative/tranquilizer (*H9*), and stimulant (*H10*) misuse directly. While HOP

and AS' effects on opioid and sedative/tranquilizer misuse were thought to be fully mediated by symptoms of depression and anxiety, respectively – IMP is known to carry risk over and above symptoms of ADHD and CD (Milner *et al.*, 2014).

Rationale. The present study's data was collected as part of the Co-Venture Trial (O'Leary-Barrett *et al.*, 2017). The Preventure Program (Newton *et al.*, 2016) examines the longer-term efficacy of personality-targeted substance abuse prevention. If we can establish prospective links between personality and various forms of PD misuse in adolescents, there would be impetus for personality-matched trials. Because HOP, AS, and IMP are also relevant to non-addictive psychopathology, personality-based approaches can effectively reduce concurrent mental health concerns (Conrod, 2016). Clients with concurrent disorders have higher rates of service, worse social outcomes, and poorer treatment response (Chen *et al.*, 2015). PD misusers with mental illness also exhibit greater levels of drug dependence, compared to their non-mentally ill counterparts (Ghandour, Martins, & Chilcoat, 2008). Thus, our results may prove useful for both personality-matched prevention and concurrent disorder treatment-planning.

Methods

Assenting students from 31 high schools (public and private; English and French) in Québec, Canada participated. Data was collected annually (during the fall and spring terms) beginning in September 2012. A web-based platform (Delosis Ltd., London, U.K.) was used to survey students during regular class times. At baseline, students were in Grade 7. The present study is archival and used data collected in Grade 9 (September 2014-May 2015) and Grade 10 (September 2015-May 2016). Across Canada, "high school" consistently includes Grades 9-12. Grade 11 data was not yet available. Ethical

approval was granted by Sainte-Justine Hospital's Research Ethics Board and each administrative school board.

Participants

Sample sizes were n = 3,024 students in Grade 9 and n = 2,869 students in Grade 10. Across this year, the attrition rate was 5%. T-tests and between-group ANOVAs suggest that the students who dropped out were older and more likely to attend certain schools. They endorsed more personality vulnerability (HOP, SS, IMP); mental health symptoms (depression, CD, ADHD); alcohol misuse; and PD misuse. The sample was evenly distributed across genders. The majority of students were middle class and of Canadian or American descent. See Table 6.1 for more details about our sample.

Measures

Personality (see Appendix C). The 23-item SURPS (Woicik *et al.*, 2009) was used to assess personality. It has four subscales: HOP (7 items; "I feel that I'm a failure"), AS (5 items; "It is frightening to feel dizzy or faint"), SS (6 items; "I like doing things that frighten me a little"), and IMP (5 items; "I usually act without stopping to think"). Participants responded using a 5-point Likert scale (1 *strongly disagree* to 5 *strongly agree*). Following any reverse scoring, subscale scores were generated by summing component items. The SURPS has strong psychometric properties in both English (Castellanos-Ryan *et al.*, 2013) and French (Castonguay-Jolin *et al.*, 2013). In our sample, the subscales were internally consistent: HOP α = .89 in Grades 9 and 10; AS α = .70 in Grade 9 and .73 in Grade 10; SS α = .70 in Grade 9 and .71 in Grade 10; IMP α = .75 in Grades 9 and 10.

Internalizing symptoms (see Appendix L). The 18-item Brief Symptom Inventory-18 (BSI-18; Derogatis, 2001) was used to assess symptoms of depression and anxiety. It measures past-week psychological distress across three subscales. In this study, only the Depression (6 items; "feeling blue") and Anxiety (6 items; "nervousness or shakiness inside") subscales were analyzed. Participants responded using a 5-point Likert Scale (0 *not at all* to 4 *extremely often*). Subscale scores were generated by summing component items. The BSI-18 has strong psychometric properties in both English (Lancaster, McCrea, & Nelson, 2016) and French (Perrudet-Badoux, 1987). In our sample, the subscales were internally consistent: Depression $\alpha = .90$ in Grades 9 and 10; Anxiety $\alpha = .90$ in Grade 9 and .89 in Grade 10.

Externalizing symptoms (see Appendix M). The 25-item Youth Self-Report Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) was used to assess symptoms of ADHD and CD. It measures six-month positive and negative symptoms across five subscales. In this study, only the Hyperactivity/Inattention (5 items; "restless, cannot sit still for long") and Conduct Problems (5 items; "often accused of lying or cheating") subscales were analyzed. Participants responded using a 3-point Likert Scale (0 *not true* to 2 *certainly true*). Following any reverse scoring, subscale scores were generated by summing component items. The SDQ has strong psychometric properties in both English (He, Burstein, Schmitz, & Merikangas, 2013) and French (Capron, Thérond, & Duyme, 2007). In our sample, the subscales were internally consistent: ADHD $\alpha = .72$ in Grade 9 and .74 in Grade 10; CD $\alpha = .62$ in Grade 9 and .64 in Grade 10. Of note, for short scales with 10 items or less, an alpha of $\geq .60$ is considered to be acceptable (Lowenthal, 2004).

Prescription drug misuse (see Appendix N). A modified and validated version of the Detection of Alcohol and Drug Problems in Adolescents (DEP-ADO; Landry, Tremblay, Guyon, Bergeron, & Brunelle, 2004) included the following question: "Have you used one or more of these substances in your lifetime and if so, how often?" The items related to PD misuse were as follows. (1) Opioids: "opiates like Codeine, Demerol, Morphine, Percodan, Methadone, Darvon, Opium, Dilaudil, or Talwin"; (2) sedatives: "non-prescribed medication like barbiturates, sedatives, downers, or sleeping pills like Seconal and Quaaludes"; (3) tranquilizers: "tranquilizers or anti-anxiety pills like Valium, Librium, or Ativan (without a prescription)"; and (4) stimulants: "stimulants, speed, methamphetamine, amphetamine, or Benzedrine". Participants responded using a 6-point Likert scale (0 *never* to 5 *every day*). To deal with zero-inflation, items were scored dichotomously (i.e., 1 if the student *had used that PD class* and 0 if they *had not*). In keeping with our previous research (Chinneck *et al.*, 2018b), sedatives and tranquilizers were collapsed. The DEP-ADO has strong psychometric properties in both English (Landry *et al.*, 2004) and French (Bernard *et al.*, 2005).

Alcohol misuse (see Appendix N). Alcohol misuse were also assessed using the DEP-ADO (Landry *et al.*, 2004). It includes 10 yes/no items that pertain to lifetime issues with: physical health, psychological health, familial relationships, intimate relationships, academics, finances, delinquency, risky behaviour, alcohol tolerance, and treatment seeking. These items were summed to create a 0-10 total alcohol harms score.

Statistical Analyses

Sample descriptive statistics were first calculated in SPSS 20.0. The hypothesized model was then run in MPlus 7.11 (Muthén & Muthén, 1998-2012). Correlations were

specified between the personality, mental health, and PD misuse variables. Because our dependent variables were categorical, a robust weighted least squares approach was used (ESTIMATOR = WLSMV; Muthén, 2010). Missing data was handled using pairwise deletion. We controlled for School and Grade 9 mental health and PD misuse. We also controlled for Age, Gender, Ethnicity, and Socioeconomic Status (Currie, Elton, Todd, & Platt, 1997), given their known effects on PD misuse (Milner *et al.*, 2014; Nargiso *et al.*, 2015). Finally, because high-intensity drinking is associated with PD misuse (McCabe *et al.*, 2017), we controlled for Alcohol Misuse. All of these covariates were regressed onto all of the outcome variables. See Appendix H (Table H.3) for a supplementary correlation table that includes all of the covariates and other variables of interest. Standard indices were used to assess model fit. RMSEA ≤ 0.05 and CFI/TLI ≥ .95 indicate good fit. RSMEA≤ 0.08 and CFI/TLI ≥ .90 indicate adequate fit (Hu & Bentler, 1999). Of note, chi-square values are often significant when the sample size is large (Curran *et al.*, 1996). As such, we have reported chi-square but have not interpreted it as a stand-alone fit statistic. Significant effects were detected at a 95% confidence interval. Bootstrapped confidence intervals were used to determine the significance of the mediated effects.

Results

In Grade 10, lifetime PD misuse rates were: 3% for opioids, 4% for sedatives/tranquilizers, and 2% for stimulants. Descriptive statistics are displayed in Table 6.1 and correlations in Table 6.2. Our hypothesized model (see Figure 6.1) fit well: $\chi^2(68) = 156.56$, $p < .001$; RMSEA = .02, 90% CI [.02, .03]; CFI = .98; TLI = .96. Indirect effects are reported in Table 6.3. The following hypotheses were supported. *H1*: HOP was related to opioid misuse via depressive symptoms; the indirect effect was

significant. *H2*: AS was related to sedative/tranquilizer misuse via anxiety symptoms; the indirect effect was significant. *H4*: IMP was related to stimulant misuse via ADHD symptoms; the indirect effect was significant. *H5-H7*: IMP was related to opioid, sedative/tranquilizer, and stimulant misuse via CD symptoms; all indirect effects were significant. *H3* was partially supported. The direct path from SS to stimulant misuse was marginally significant ($p = .06$)[1]. *H8-10* were not supported. The direct paths from IMP to opioid, sedative/tranquilizer, and stimulant misuse were non-significant ($p > .05$).

Discussion

Our sample's PD misuse rates are largely in keeping with previously-reported Canadian statistics (CAMH, 2017). Hypothesized pathways were grounded in several etiological models of addiction (Castellanos-Ryan & Conrod, 2012) and were largely supported.

Self-Medication

Personality predicts adolescent mental health problems (Castellanos-Ryan *et al.*, 2013). In keeping with self-medication theories (Khantzian, 1997), mental health problems tend to precede substance misuse (McCabe *et al.*, 2007). Young adults with mental illness, for example, are at a much greater risk of PD misuse, compared to those without (Lo *et al.*, 2013). Further, when severity of mental illness is controlled, lack of needed mental health care is related to PD misuse.

***A* Paths: Negative affect regulation.** *H1* predicted that HOP would lead to opioid misuse via depressive symptoms; *H2* predicted that AS would lead to

[1] Preliminary analyses did not control for alcohol misuse. In that model, SS to stimulant misuse was significant ($p < .05$). Once this important covariate was added (i.e., in the current model), only this path was reduced to marginal significance ($p = .06$).

sedative/tranquilizer misuse via anxiety symptoms. Both of these hypotheses were supported; the indirect effects were significant.

HOP is a precursor for depression (Joiner, 2000). Opioids are typically prescribed for physical pain (Smith *et al.*, 2015a) and not for depression (Singh & Reece, 2014). Thus, because high-HOP users are taking opioids for non-prescribed reasons, this pathway constitutes PD *misuse* (Haydon *et al.*, 2006). In their review, Amari *et al.* (2011) concluded that depression was the mental health issue most strongly related to opioid misuse (odds ratios ranged from 1.2 to 4.3). Opioids possess psychic pain-numbing properties (Smith *et al.*, 2015a), which makes them particularly attractive to high-HOP adolescents, who are prone to depression and looking to dull their psychological pain. Thus, in adolescence, HOP likely confers risk for opioid misuse via negative affect regulation and self-medication motives.

AS is a precursor for anxiety (McLaughlin *et al.*, 2007). While sedatives/tranquilizers are commonly prescribed for anxiety (Bisaga & Mariani, 2015), the relevant DEP-ADO items (Landry *et al.*, 2004) specify "non-prescribed" use "without a prescription". This suggests that high-AS adolescents are taking sedatives/tranquilizers that have not been prescribed to them (i.e., that they have obtained from their family, friends, or dealers) in an attempt to quell their anxiety symptoms. Thus, in adolescence, AS likely confers risk for sedative/tranquilizer misuse via negative affect regulation and self-medication motives.

***C* Paths: Psychological dysregulation.** *H4* predicted that IMP would lead to stimulant misuse via ADHD symptoms. This hypothesis was supported; the indirect effect was significant. This pathway likely pertains to both medical stimulant use (i.e.,

medication) and non-medical stimulant misuse (i.e., self-medication). IMP is a prominent symptom of ADHD (Barkley *et al.*, 2008) and stimulants are prescribed for ADHD (Solanto *et al.*, 2001). Prescription stimulants are classified as Schedule III under the Controlled Drugs and Substances Act. This suggests that, although prescription stimulants have been approved for medical use, they have a high potential for abuse (Kollins, 2007). For those high in IMP, availability is the best predictor of misuse (Hecimovic *et al.*, 2014). Teens who report symptoms of ADHD are more likely to have prescriptions, which they can then misuse (e.g., by taking their stimulants in greater amounts or more often than prescribed, via non-intended routes, for non-prescribed ADHD-related reasons, and/or with contraindicated substances; Haydon *et al.*, 2006). In their review, Weyandt *et al.* (2014) concluded that estimates of stimulant misuse were relatively low in general adolescent samples. Rates were much higher, however, among adolescents who: had symptoms of ADHD, had ADHD, were receiving treatment for ADHD, had prescriptions for stimulant medication, were performing worse academically, or were using other substances. Van Eck *et al.* (2012) suggest that young people misuse stimulants to cope with their ADHD-related disorganization, poor time management, forgetfulness, and distractibility. Thus, in adolescence, IMP likely confers risk for stimulant misuse (in part) via self-medication of psychological dysregulation. Because ADHD does not involve negative affect (like anxiety or depression), this type of self-medication is theoretically distinct from the negative affect regulation paths (i.e., *A* paths) described above.

B Paths: Positive Affect Regulation

H3 predicted that SS would lead to stimulant misuse. This hypothesis was partially supported. In a preliminary model (in which alcohol misuse was not co-varied), this effect was significant. In the current model (which controlled for alcohol misuse), this effect was marginally significant. It is therefore likely that the same process links SS to both excessive drinking *and* stimulant misuse.

SS is robustly associated with adolescent alcohol misuse (Krank *et al.*, 2011). McCabe *et al.* (2015) found that 48% of adolescents co-ingested alcohol and prescription stimulants. Non-medical users who took stimulants with other substances were more likely to report recreational motives and greater subjective highs – both of which are consistent with high-SS. SS is strongly related to positive reinforcement and enhancement motives (Woicik *et al.*, 2009). It predicts substance misuse (Mackie *et al.*, 2011) that is driven by a need for positive affect and psycho-stimulation (Comeau *et al.*, 2001). Finn, Sharkansky, Brandt, & Turcotte (2000) found that SS was both directly and indirectly (through alcohol use and positive alcohol expectancies) linked to alcohol problems. Castellanos-Ryan *et al.* (2011) concluded that SS's effect on binge drinking was mediated by reward response bias. Thus, in adolescence, SS likely confers risk for substance misuse (including stimulant misuse) via positive affect regulation and recreational motives. Future studies might explicitly test this; is SS's effect on stimulant misuse mediated by enhancement motives or a reward response? Existing motive questionnaires tend to be substance-specific (e.g., Modified DMQ Revised; Grant *et al.*, 2007b). However, a Clinical Substance Use Motives measure was recently developed (Blevins, Lash, & Abrantes, 2018) and includes items about stimulant use for

social/enhancement motives. Reward sensitivity, on the other hand, can be assessed using either self-report (e.g., Sensitivity to Reward and Punishment Questionnaire; Torrubia, Avila, Molto, & Caseras, 2001) or behavioural tasks (e.g., rewarded go/no-go tasks; see Castellanos-Ryan *et al.*, 2011).

D Paths: Deviance Proneness

H5-7 predicted that IMP would lead to opioid, sedative/tranquilizer, and stimulant misuse via CD symptoms. These hypotheses were supported; the indirect effects were significant. IMP's relationship with substance misuse is motivationally undefined (Woicik *et al.*, 2009). It is more reflective of a general inability to inhibit behaviour (Finn, Mazas, Jutus, & Steinmetz, 2002). Self-reported IMP, for example, is associated with deficits in response execution and inhibition (Castellanos-Ryan *et al.*, 2011). Poor response inhibition is a risk factor for both CD (Herba, Tranah, Rubia, & Yule, 2006) and substance misuse (Li, Huang, Constable, & Sinha, 2006). Furthermore, paths from IMP-to-CD and IMP-to-alcohol problems are partially mediated by deficient response inhibition (Castellanos-Ryan *et al.*, 2011; Finn *et al.*, 2000). In sum, we know that high-IMP teens struggle to regulate and inhibit their impulses and that this makes them more vulnerable to deviance (including CD and PD misuse). Thus, in adolescence, IMP likely confers risk for PD misuse via deviance proneness. This finding is in keeping with previous research with other substances. Mackie *et al.* (2011), for instance, found that IMP predicted adolescent alcohol use via CD symptoms.

H8-10 predicted that IMP would also lead to opioid, sedative/tranquilizer, and stimulant misuse directly. Unexpectedly (Milner *et al.*, 2014), these hypotheses were not supported. This suggests that IMP's risk for unconstrained PD misuse is fully mediated

by CD symptoms. Of note, in the case of stimulant misuse, the full mediation of IMP-related risk also includes ADHD symptoms. Our conceptualizations of CD and ADHD may have accounted for all of the IMP-related risk for PD misuse. Our use of the more general SURPS (Woicik *et al.*, 2009) IMP subscale could have further contributed to this discrepancy. For increased clarity, future studies could replicate our model using Barratt's Impulsiveness Scale-11 (Patton *et al.*, 1995) as it delineates attentional, motor, and non-planning forms of IMP.

Limitations

First, we measured personality in Grade 9 – and mental health symptoms and PD misuse in Grade 10. As such, our final pathways (from mental health symptoms to PD misuse) were cross-sectional. Methodologically, we set our semi-longitudinal model up in this way because *H1-3* pertain to self-medication. Measuring PD misuse in Grade 11, for example, would have meant asking students if they had misused PDs to cope with the mental health symptoms they had reported a year earlier. Grade 11 data was not yet available and we wanted to measure mental health symptoms and PD misuse in closer proximity. This meant sacrificing a three-wave model. While theory suggests that the mental health-to-PD misuse relationship is directional (Becker *et al.*, 2007), our model cannot make this causal inference. PD misuse, for example, might exacerbate students' mental health symptoms (Soule & Connery, 2018). To address this limitation, our model should be replicated in a fully longitudinal design that uses shorter (e.g., 6 month) lags between waves. Future research could also use ecological momentary assessment to examine these relationships on a day-to-day basis.

Second, our PD misuse questions were worded inconsistently. The DEP-ADO was chosen because it is standardized and has reliably used to measure Canadian high school students' substance use (Landry *et al.*, 2004). It has been validated for use with both English- and French-speaking Québécois youth (Castonguay-Jolin *et al.*, 2013) – like those in our sample. Despite these strengths, the DEP-ADO is limited. It provides little information about students' diversion sources, administration routes, or motives for use. Further, while the sedative and tranquilizer items include key phrases for assessing misuse (e.g., "non-prescribed" and "without a prescription"), the phrasing of the opioid and stimulant items was more ambiguous. As such, these items may have captured use that was not necessarily misuse (e.g., that was medically-sanctioned). Finally, the stimulant item listed examples of non-prescribed (e.g., speed) and prescribed (e.g., methamphetamine, amphetamine, Benzedrine) drugs. Barrett *et al.*'s (2008) review suggests that PD misuse is often operationalized inconsistently. Smith *et al.* (2015b) further concluded that none of the instruments published to date can adequately assess PD misuse or context of use. Their review emphasizes that more valid PD misuse outcome measures must be developed. When these become available, our model should be replicated. This would reduce measurement error, allowing for a more accurate and refined test of personality's effects on PD use vs. misuse.

Finally, our models did not control for group participation (i.e., assignment to the intervention vs. control groups) as the trial was still taking place at the time the present study was conducted. Relatedly, our analyses did not control for the fact that students' data was nested within schools.

Conclusion

The present study sought to address the limitations of the extant literature on adolescent PD misuse, as outlined by Nargiso *et al.* (2015). We applied the four-factor vulnerability model (Pihl & Peterson, 1995) to adolescents' sedative/tranquilizer, opioid, and stimulant misuse. HOP, AS, and IMP conferred risk indirectly via the self-medication of negative affect and psychological dysregulation. HOP predicted opioid misuse via depression symptoms; AS predicted sedative/tranquilizer misuse via anxiety symptoms; and IMP predicted stimulant misuse via ADHD symptoms. Consistent with positive affect regulation, SS was marginally associated with stimulant misuse. Finally, IMP conferred risk for unconstrained PD misuse via deviance proneness. CD symptoms (and ADHD symptoms in the case of stimulant misuse) fully mediated the relationships between IMP and PD misuse.

Clinical Implications

Our model suggests that opioid and sedative/tranquilizer misuse may be best treated by targeting those high in internalizing traits. Cognitive-behavioural therapy could benefit teens high in HOP and AS. It would teach them to better cope with their respective symptoms of depression and anxiety (Colognori, Herzig, Reigada, Leiby, & Warner, 2014). Stimulant misuse may be best treated by targeting those high in externalizing traits. High-SS teens could be encouraged to pursue other stimulating, prosocial activities (Herie & Watkin-Merek, 2006). For example, "alternate rebellions" include dyeing your hair, getting a tattoo, or getting a piercing (Linehan, 2015). High-IMP teens could be taught behavioural ADHD-management techniques (Antshel & Olszewski, 2014). Finally, antisocial youth may be engaging in unconstrained PD misuse

among other conduct problems. Thus, these teens could benefit from increased future-oriented thinking. Motivational approaches could be used to teach them to weigh the short vs. long term consequences of their behaviour (Conrod, Stewart, Comeau, & Maclean, 2006).

Treating PD misuse is, of course, important. But, given the current North American opiate crisis (Fischer *et al.*, 2014), preventing it is *critical*. In the U.S. alone, 1.9 million individuals are estimated to initiate PD misuse annually (SAMHSA, 2011). The likelihood of reporting PD misuse in a given year, during adolescence, increases with age (Milner *et al.*, 2014). Rates rise consistently between Grade 8-12 and ages 12-17 (Johnston, O'Malley, Bachman, & Schulenberg, 2010). Thus, prevention efforts geared towards at-risk youth are especially vital. Our results suggests that identifying high personality-risk adolescents (i.e., those high in HOP, AS, SS, or IMP) would benefit both early intervention and targeted prevention strategies.

Personality-matched protocols have effectively reduced illicit drug use in youth (Conrod *et al.*, 2010) and PD misuse in adults (Conrod *et al.*, 2000b). The present study was embedded within a larger trial, which evaluated the longer-term efficacy of the Preventure Program (Newton *et al.*, 2016). This personality-matched prevention program targets teens with elevated four-factor trait scores (Pihl & Peterson, 1995). It is rooted in the cognitive-behavioural model and incorporates psycho-educational and motivational interviewing components. When applied to alcohol and illicit drug use, the Preventure Program has resulted in delayed onset and reduced escalation of misuse (O'Leary-Barrett *et al.*, 2017). Our results suggest that the Preventure Program should be applied to adolescent PD misuse next. Our model suggests that personality is similarly related to PD

misuse, via mental health symptoms. Thus, we believe that this personality-matched prevention and intervention could prevent PD uptake (if administered prior to onset) and reduce PD misuse.

Table 6.1. Frequencies and Descriptives.

	Grade 9			Grade 10		
	n	%	*M (SD)*	*n*	%	*M (SD)*
Age	3,024		14.79 (0.47)	2,869		15.83 (0.42)
Gender						
1. Male	1,463	50.7		1,374	50.1	
2. Female	1.425	49.3		1,371	49.9	
Ethnicity						
1. Canadian or American	2,535	87.8		2,413	87.9	
2. European	64	2.2		63	2.3	
3. African	57	2.0		46	1.7	
4. Caribbean	28	1.0		26	0.9	
5. East Asian	81	2.8		82	3.0	
6. South Asian	17	0.6		17	0.6	
7. Middle Eastern	21	0.7		21	0.8	
8. South or Central American	44	1.5		39	1.4	
9. Other	27	0.9		23	0.8	
10. Don't know	14	0.5		15	0.5	
Socioeconomic Status			5.36 (1.69)			5.37 (1.66)
Alcohol Misuse			.09 (.59)			.17 (.79)
Hopelessness			12.51 (3.92)			12.73 (3.83)
Anxiety Sensitivity			11.09 (2.95)			11.02 (2.97)
Sensation Seeking			16.14 (3.63)			16.37 (3.70)
Impulsivity			11.66 (2.91)			11.55 (2.87)

	Grade 9			Grade 10		
	n	%	*M (SD)*	*n*	%	*M (SD)*
Depression			5.32 (5.98)			5.45 (5.93)
Anxiety			2.81 (4.03)			2.82 (3.99)
ADHD			4.12 (2.40)			4.12 (2.38)
CD			2.18 (1.64)			2.09 (1.61)
Opioids	54	1.8		88	3.1	
Sedatives/Tranquilizers	95	3.2		100	3.5	
Stimulants	50	1.7		63	2.2	

Note. For Gender and Ethnicity, numbers represent codings. Socioeconomic Status was rated using a 10-point Likert scale (Currie *et al.*, 1997) with higher scores representing greater wealth. Alcohol Harms (range = 0-10) were assessed using the DEP-ADO (Landry *et al.*, 2004). Personality was assessed using the SURPS (Woicik *et al.*, 2009). Subscale ranges are as follows: 7-25 for Hopelessness, 5-25 for Anxiety Sensitivity, 6-30 for Sensation Seeking, and 5-25 for Impulsivity. Depression (range = 0-24) and Anxiety (range = 0-24) were assessed using the BSI-18 (Derogatis, 2001). ADHD is Attention-Deficit Hyperactivity Disorder (range = 0-10) and CD is Conduct Disorder (range = 0-10). Both were assessed using the SDQ (Goodman, 1997). PD misuse was assessed using the DEP-ADO (Landry *et al.*, 2004) and scored dichotomously.

Table 6.2. Correlation Matrix.

	2	3	4	5	6	7	8	9	10	11	12
Grade 9											
1. Hopelessness	**.27**	-.03	**.32**	**.11**	**.46**	**.35**	**.33**	**.22**	**.07**	**.11**	**.09**
2. Anxiety Sensitivity	1.00	**-.12**	**.19**	.02	**.23**	**.33**	**.14**	**.05**	-.03	-.01	-.01
3. Sensation Seeking		1.00	**.25**	**.14**	-.01	-.02	**.12**	**.16**	**.13**	**.11**	**.11**
4. Impulsivity			1.00	**.11**	**.22**	**.18**	**.45**	**.41**	**.11**	**.11**	**.12**
5. Alcohol Harms				1.00	**.08**	**.06**	**.08**	**.13**	**.12**	**.17**	**.20**
Grade 10											
6. Depression					1.00	**.73**	**.29**	**.24**	**.07**	**.16**	**.10**
7. Anxiety						1.00	**.29**	**.20**	**.06**	**.11**	**.07**
8. ADHD							1.00	**.40**	**.09**	**.09**	**.10**
9. CD								1.00	**.14**	**.14**	**.14**
10. Opioids									1.00	**.21**	**.30**
11. Sedatives/Tranquilizers										1.00	**.17**
12. Stimulants											1.00

Note. ADHD is Attention-Deficit Hyperactivity Disorder; CD is Conduct Disorder. Bold correlations are significant at $p < .05$.

Table 6.3. Tests of Hypothesized Indirect Effects.

Hypothesis	Predictor	Mediator	Outcome	Indirect Effect	95% Confidence Interval
H1	Hopelessness	Depression	Opioids	.003	[.00, .01]*
H2	Anxiety Sensitivity	Anxiety	Sedatives/Tranquilizers	.005	[.00, .01]**
H4	Impulsivity	ADHD	Stimulants	.005	[.00, .01]*
H5	Impulsivity	CD	Opioids	.005	[.00, .01]*
H6	Impulsivity	CD	Sedatives/Tranquilizers	.008	[.00, .01]**
H7	Impulsivity	CD	Stimulants	.005	[.00, .01]*

Note. ADHD is Attention-Deficit Hyperactivity Disorder; CD is Conduct Disorder. * $p < .05$, ** $p < .01$.

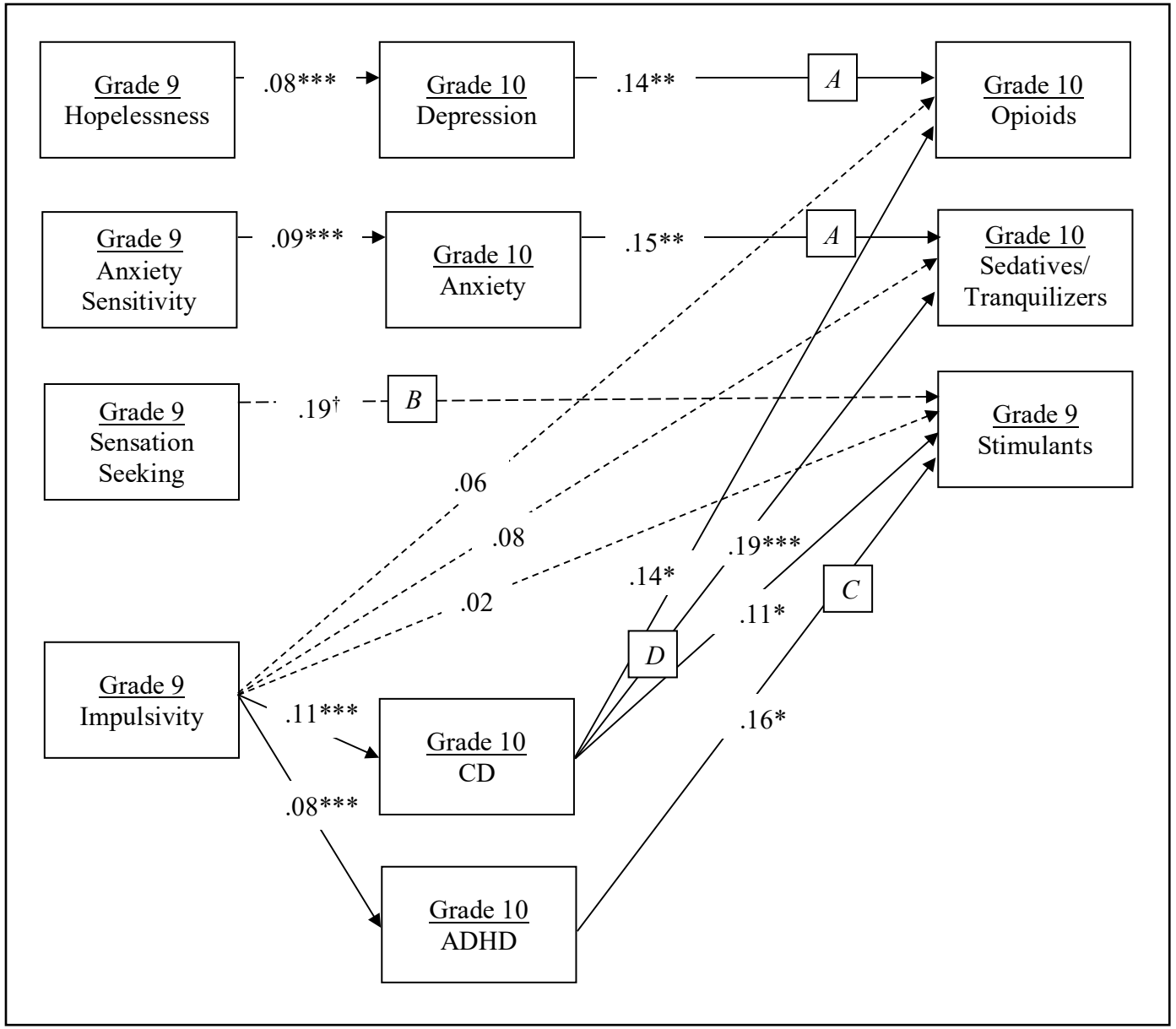
Grade 9 Hopelessness
.08***
Grade 10 Depression
.14**
A
Grade 10 Opioids
Grade 9 Anxiety Sensitivity
.09***
Grade 10 Anxiety
.15**
A
Grade 10 Sedatives/ Tranquilizers
Grade 9 Sensation Seeking
.19†
B
Grade 9 Stimulants
.06
.08
.02
.14*
.19***
.11*
C
D
Grade 9 Impulsivity
.11***
Grade 10 CD
.08***
Grade 10 ADHD
.16*

Figure 6.1. Model results. Solid arrows represent statistically significant hypothesized pathways; dotted arrows represent hypothesized but non-significant pathways. Numbers represent standardized coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$. † represents marginal significance at $p = .06$. *A* pathways are informed by the negative affect regulation model. The *B* pathway is informed by the positive affect regulation model. The *C* pathway is informed by the psychological dysregulation model. *D* pathways are informed by the deviance proneness model.

CHAPTER 7. DISCUSSION

My dissertation sought to better understand the effect of internalizing and externalizing personality on adolescents' and emerging adults' misuse of alcohol and PDs. This involved testing various tenets of established etiological addiction models (Castellanos-Ryan & Conrod, 2012) across three studies. The proceeding sections summarize and integrate my findings, as they pertain to previous literature and to this overarching goal. I then discuss my dissertation's theoretical implications, clinical implications, strengths, and limitations. Finally, I conclude by suggesting related directions for future research.

Summary and Integration of Findings

Summary: Study 1

Study 1, "*Neurotic personality traits and risk for adverse alcohol outcomes: Chained mediation through emotional disorder symptoms and drinking to cope*", examined the effects of internalizing personality on alcohol misuse. I predicted that AS and HOP would increase hazardous alcohol use and drinking harms via (1) emotional disorder symptoms and (2) specific coping drinking motives. Emerging adults in their first year of university (at three Canadian universities) were sampled. My model was cross-sectional and controlled for university and age. My hypotheses were supported: paths from AS to anxiety symptoms to DCWA to alcohol outcomes and from HOP to depressive symptoms to DCWD to alcohol outcomes were significant. However, results were not as specific as I had predicted. All sixteen of the indirect pathways that incorporated both mediators (i.e., emotional disorder symptoms and coping drinking motives) were significant. There was, however, some evidence of specificity. In the AS-

to-alcohol outcomes chain, DCWA motives were more strongly associated with anxiety than depression at a 95% CI. In the HOP-to-alcohol outcomes chain, depression was more strongly associated with HOP than AS at a 95% CI. Study 1 extended prior work (e.g., Allan *et al.*, 2014) by clarifying how AS and HOP confer vulnerability for alcohol misuse. I concluded that inhibited personality was first associated with increased emotional disorder symptoms, and then drinking to cope with them, resulting in hazardous and harmful alcohol use. For high-AS drinkers, this pathway mainly involved greater anxiety symptoms and DCWA motives. To a lesser extent, it also involved depressive symptoms and drinking to cope with them. For high-HOP drinkers, this pathway mainly involved greater depressive symptoms and DCWD motives. To a lesser extent, it also involved anxiety symptoms and drinking to cope with them.

Summary: Study 2

Study 2, "*Personality and prescription drug use and misuse among first year undergraduates*", examined personality's effects on overall PD use, medically-sanctioned PD use, and PD misuse. I predicted that: AS would be related to sedatives/tranquilizers, HOP to opioids, SS to stimulants, and IMP to all three drug classes (i.e., unconstrained PD use). Emerging adults in their first year of university (across two cohorts from one Canadian university) were sampled. My model was cross-sectional and controlled for cohort, age, gender, and alcohol dependence. My hypotheses were largely supported. In the "any use" model, all hypothesized pathways were significant – save for IMP to opioid use. In the "medically-sanctioned use" model, AS was associated with sedatives/tranquilizers, HOP with opioids, and IMP with stimulants. In the "misuse" model, AS was (marginally) associated with sedatives/tranquilizers, SS with stimulants,

and IMP with unconstrained PD misuse. Study 2 extended prior work (e.g., Woicik *et al.*, 2009) by applying Pihl & Peterson's (1995) four-factor vulnerability model to distinct patterns of PD-taking. Internalizing and externalizing traits were found to differentially predict the use and misuse of specific PD classes, in ways that were largely consistent with theory (Castellanos-Ryan & Conrod, 2012).

Summary: Study 3

Study 3, "*Personality, mental health, and prescription drug use among high school students*", examined if specific sets of mental health symptoms mediated the effect of personality on PD misuse. Adolescents (from 31 Canadian high schools) were sampled in Grade 9 and again in Grade 10. Retention was high (95%) across this one-year follow-up. At baseline, students' personality was assessed. At both time points, they completed measures of mental health symptoms and PD misuse. My model was semi-longitudinal and controlled for school, baseline mental health symptoms, baseline PD misuse, age, gender, ethnicity, socioeconomic status, and alcohol misuse. My hypotheses were largely supported. AS predicted sedative/tranquilizer misuse via anxiety symptoms, HOP predicted opioid misuse via depressive symptoms, and IMP predicted stimulant misuse via ADHD symptoms. IMP also predicted unconstrained PD misuse (i.e., misuse of all three drug classes) via CD symptoms. After controlling for alcohol misuse, SS was only marginally associated with stimulant misuse. Unexpectedly, IMP was not directly associated with the misuse of sedatives/tranquilizers, opioids, or stimulants. Study 3 extended Study 2's findings by helping specify *how* personality is linked to PD misuse. It suggests that personality's influence on PD misuse stems from its association with specific sets of mental health symptoms.

Integration

Study 1 (emerging adults) added to the extant literature by examining specific vulnerability pathways that explain the links between internalizing personality and alcohol problems. Study 2 (emerging adults) was novel in its operationalization and delineation of specific PD-taking patterns: overall use, medically-sanctioned use, and misuse. Study 3 (adolescents) was the first to examine theoretical, mediational paths from personality to mental health symptoms to PD misuse. All three support the continued applicability of Pihl & Peterson's (1995) four-factor personality vulnerability model to young people's use and misuse of alcohol and PDs, as summarized in Table 7.1.

AS is thought to confer risk for substance misuse via the self-medication of anxiety. High-AS users are more sensitive, pharmacologically, to the arousal-dampening properties of alcohol and benzodiazepines (Conrod *et al.*, 1998; MacDonald *et al.*, 2000). Thus, they misuse alcohol (Conrod *et al.*, 1998; Stewart *et al.*, 1995, 1997) and anxiolytics (Conrod *et al.*, 2000a) to cope with the arousal-related sensations they so fear (Norton, 2001; Stewart *et al.*, 1999, 2000; Zvolensky *et al.*, 2001). My dissertation's findings offer additional support for this AS risk-pathway. In Study 1, AS predicted hazardous alcohol use and drinking harms via (1) symptoms of anxiety/depression and (2) coping drinking motives. There was some evidence of specificity; DCWA motives were more strongly associated with anxiety than depression. In Study 2, AS predicted the use and medically-sanctioned use of sedatives/tranquilizers. AS was also marginally associated with sedative/tranquilizer misuse. In Study 3, AS's relationship with sedative/tranquilizer misuse was found to be mediated by anxiety symptoms.

HOP is thought to confer risk for substance misuse via the self-medication of depression. HOP is associated with alcohol and opioid misuse (Conrod *et al.*, 2000a; Krank *et al.*, 2011). Both have inhibitory, analgesic effects (Gray, 1982). HOP is further related to depression (Mackie *et al.*, 2011) and to substance use motives related to coping with depression (Grant *et al.*, 2007b; Woicik *et al.*, 2009). My dissertation's findings offer additional support for this HOP risk-pathway. In Study 1, HOP predicted hazardous alcohol use and drinking harms via (1) symptoms of anxiety/depression and (2) coping drinking motives. There was some evidence of specificity; depression was more strongly associated with HOP than AS. In Study 2, HOP predicted the use and medically-sanctioned use of opioids. Unexpectedly, HOP failed to predict opioid misuse. Opioids, however, are not prescribed to treat depression (Singh & Reece, 2014). It could therefore be argued that taking them to manage HOP would constitute non-prescribed use (i.e., misuse; Haydon *et al.*, 2006). Consistent with this suggestion, in Study 3, HOP's relationship with opioid misuse was found to be mediated by depressive symptoms. This suggests that opioids are being used by young people high in HOP to numb negative affect and to self-medicate depressive symptoms.

SS confers risk for substance misuse via enhancement motives (Cooper *et al.*, 1995). High-SS substance users are particularly sensitive to drugs' rewarding properties (Castellanos-Ryan & Conrod, 2012). They tend to misuse immediately-reinforcing stimulants (Low & Gendaszek, 2002) to increase their positive affect (Simons *et al.*, 2005). For the most part, my dissertation's findings offer additional support for this SS risk-pathway. In Study 2 SS predicted the overall use and misuse of stimulants and in Study 3 SS (marginally) predicted stimulant misuse.

IMP has been linked to a motivationally undefined pattern of substance use (Woicik *et al.*, 2009). Availability is the best predictor of drug misuse in high-IMP individuals (Hecimovic *et al.*, 2014). My dissertation supports several distinct IMP risk-pathways. In Study 2, IMP predicted medically-sanctioned stimulant use. This makes sense; IMP is implicated in ADHD (APA, 2013) and stimulants are prescribed to treat ADHD (Cortese *et al.*, 2013). IMP also predicted the use of sedatives/tranquilizers and stimulants and the misuse of sedatives/tranquilizers, opioids, and stimulants. These findings are in keeping with previous research, which suggests that high-IMP substance users are unconstrained in their drug-taking (Woicik *et al.*, 2009). In Study 3, IMP's relationship with unconstrained PD misuse was found to be fully mediated by CD symptoms. IMP's relationship with stimulant misuse was also partially mediated by ADHD symptoms. This suggests that in the pathways from IMP to PD misuse, sedative/tranquilizer, opioid, and stimulant misuse occurs in the context of other deviant behaviours (i.e., elevated CD symptoms). In the pathway from IMP to ADHD to stimulant misuse, these medications might be misused to self-medicate dysregulation.

Discrepancies between Studies

Overall, across studies, my dissertation's findings were very consistent. My theoretical predictions, which emerged from Pihl & Peterson's (1995) four-factor vulnerability model and Castellanos-Ryan & Conrod's (2012) adaptation, were reliably supported. However, some minor inconsistencies across studies bear mentioning.

The first discrepancy pertains to whether AS predicts depressive symptoms and HOP predicts anxiety symptoms – or whether the pathways from these inhibited traits to these emotional disorder symptoms are more specific (i.e., AS to anxiety and HOP to

depression). Study 1 suggested that both AS and HOP were related to both anxiety and depression. But modification indices in Study 3 indicated that the inclusion of paths from AS-to-depression and HOP-to-anxiety did not improve model fit. This discrepancy may have been due to inconsistent sampling and/or measurement across Studies 1 and 3. First, these internalizing paths could be distinct in adolescence (Study 3) before generalizing in emerging adulthood (Study 1). Second, anxiety and depression are highly correlated constructs and highly comorbid disorders (Long, Young, & Hankin, 2018). In Study 3 we used the BSI-18 (Derogatis, 2001), which has validated Anxiety vs. Depression subscales. In Study 1, we used the K10 (Kessler *et al.*, 2002). While my supplementary analyses validated a second-order anxiety vs. depression factor structure, the K10 was not originally designed to be used in this manner and other measures may better separate these constructs. Furthermore, Cummings, Caporino, & Kendall (2014) have highlighted the need for empirical work that does not treat anxiety as a single, isomorphic class of symptoms. Generalized anxiety, social anxiety, panic, and separation anxiety, for example, have different ages of onset, longitudinal trajectories, and symptom expressions (Copeland, Angold, Shanahan, & Costello, 2014; Weems & Costa, 2005). Moreover, their degrees of co-occurrence with depression and other psychopathologies vary (Hankin *et al.*, 2016). Thus, Study 1's failure to support more specific internalizing paths may have been secondary to measurement error in our assessment of anxiety and depression.

The second discrepancy pertains to AS's and HOP's effects on PD misuse. In Study 2, AS marginally predicted sedative/tranquilizer misuse and HOP failed to predict opioid misuse. In Study 3, AS was associated with sedative/tranquilizer misuse (via symptoms of anxiety) and HOP was associated with opioid misuse (via symptoms of

depression). These discrepancies could have been due to my use of different developmental stages (emerging adulthood vs. adolescence) and/or designs (cross-sectional vs. longitudinal) across Studies 2 and 3. More likely, as discussed throughout this dissertation, these discrepancies could have been due to Study 2 vs. 3's conceptualization and measurement of PD misuse. Study 2 included questions about past-term frequency and patterns of use. Study 3 used the DEP-ADO (Landry *et al.*, 2004), which assesses the lifetime use of many substances. In their review of PD misuse motives, Bennett and Holloway (2017) concluded that published studies used different methods, examined different drug classes, and phrased items inconsistently – resulting in discrepant results.

The final discrepancy pertains to SS's effects on stimulant misuse. Specifically, there was a slight inconsistency across Studies 2 and 3 as to whether SS remained linked to stimulant misuse once alcohol was controlled. Study 2 controlled for alcohol dependency and SS had a significant effect. Study 3 controlled for alcohol misuse and SS had a marginal effect. This discrepancy may have been due to inconsistent sampling and/or measurement across Studies 2 and 3. Study 2 sampled emerging adults; Study 3 sampled adolescents. Alcohol use would have been legal for Study 2's participants but illegal for Study 3's. Thus, co-varying alcohol represented control of a more deviant behaviour in Study 3 than it did in Study 2. This might explain why the significant effect was lost only in Study 3, once alcohol was controlled. Alternatively, the discrepancy may have resulted from measurement differences. The AUDIT (Saunders *et al.*, 1993) was used in Study 2; it assessed impaired control over drinking, increased salience of drinking, and morning drinking in the past term. The DEP-ADO (Landry *et al.*, 2004)

was used in Study 3; it assessed lifetime alcohol-related issues with physical health, psychological health, familial relationships, intimate relationships, academics, finances, delinquency, risky behaviour, alcohol tolerance, and treatment seeking. Thus, high-SS users might be engaging in simultaneous harmful drinking (as assessed in Study 3) that does not yet meet criteria for dependence (as assessed in Study 3).

The three discrepancies discussed above are relatively minor. For the first, Study 1's model supported partial specificity. In four cases, the theorized pathways (i.e., AS-anxiety-DCWA-alcohol outcomes and HOP-depression-DCWD-alcohol outcomes) were stronger than the non-theorized ones. The second and third pertain to differences between marginal vs. significant findings. This suggests that these findings are likely real, but that measurement error resulted in different *p* values. Overall, across Studies 1-3, my dissertation has found remarkably consistent support for predictions of the four-factor vulnerability model (Pihl & Peterson, 1995) across developmental stages (adolescence vs. emerging adulthood) and substances (alcohol vs. PDs).

Theoretical Implications

In their chapter, Castellanos-Ryan & Conrod (2012) reviewed and expanded the four-factor model's (Pihl & Peterson, 1995) underlying theory. They discussed personality's contribution to four etiological addiction models: affect regulation, pharmacological vulnerability, deviance proneness, and physiological dysregulation. My dissertation's contribution to each will be discussed, in turn, below. Because these models (while theoretically distinct) are overlapping, I have also spoken to their broader implications.

Affect Regulation Model

As outlined in Chapter 1, this model suggests that individuals use substances to regulate their affective states. Specifically, negative affect regulation (Greeley & Oei, 1999; Sher, 1987) involves misusing alcohol and PDs to relieve stress, depressed mood, and/or anxiety. This is consistent with negatively reinforcing drinking motives (i.e., conformity, DCWA, DCWD; Grant *et al.*, 2007b) and self-medication PD misuse motives (McCabe *et al.*, 2009b). Inhibited young people (i.e., those high in AS or HOP) are more likely to engage in substance use for negative affect regulation (Comeau *et al.*, 2001; Conrod *et al.*, 1998, 2000a; Kushner *et al.*, 2001; Mackinnon *et al.*, 2014; O'Connor *et al.*, 2008; Stewart & Kushner, 2001). My dissertation's findings offer additional support for this model, and its applicability to AS and HOP. In Study 1, AS and HOP predicted alcohol misuse via symptoms of anxiety/depression and DCWA/DCWD. In Study 3, AS predicted sedative/tranquilizer misuse via anxiety symptoms and HOP predicted opioid misuse via depressive symptoms. My dissertation has also advanced this theory. Study 1 was the first to test chained mediation using emotional disorder symptoms and, in turn, specific coping drinking motives. Studies 2 and 3 were the first to examine specific paths from AS to sedative/tranquilizer misuse and from HOP to opioid misuse in young people. Previously, AS and HOP's respective relationships with these PD classes had only been tested in adult treatment-seeking substance misusers (Conrod *et al.*, 2000a).

Positive affect regulation, on the other hand, involves misusing alcohol and PDs for positive reinforcement (Cooper *et al.*, 1992). This is consistent with positively reinforcing drinking motives (i.e., enhancement, social; Grant *et al.*, 2007b) and

recreational PD misuse motives (McCabe *et al.*, 2009b). High-SS young people are more likely to engage in substance use for positive affect regulation (Comeau *et al.*, 2001; Cooper *et al.*, 1995; Simons *et al.*, 2005; Woicik *et al.*, 2009). My dissertation's findings offer additional support for this model, and its applicability to SS. In Study 2, SS predicted stimulant use and misuse directly. In Study 3, once alcohol misuse was controlled, SS only marginally predicted stimulant misuse. This suggests that high-SS adolescents may be co-ingesting stimulants and alcohol (McCabe *et al.*, 2017). Staying awake, partying longer, and drinking more alcohol are major reasons for misusing stimulants (Arria & Wish, 2005; Barrett, Darredeau, Bordy, & Pihl, 2005; Low & Gendaszek, 2002; Prudhomme-White, Becker-Blease, & Grace-Bishop, 2006). Non-medical stimulant misuse and heavy alcohol use are highly correlated (Teter *et al.*, 2003). This suggests that the motivational processes that underlie SS-to-stimulant misuse are similar to those linking SS and heavy alcohol use (Conrod *et al.*, 2006, 2008; Mackie *et al.*, 2011). Prior work concluded that SS was directly and indirectly (through enhancement motives) related to problematic drinking (Adams, Kaiser, Lynam, Charnigo, & Milich, 2012). In sum, my dissertation adds to the growing body of literature that associates the need for intense, stimulating experiences (i.e., SS) with the use and misuse of stimulants (Low & Gendaszek, 2002).

Pharmacological Vulnerability Model

This model suggests that individuals respond differently to substances (Sher, 1991; Sher *et al.*, 2005). High-AS young people, for example, are more sensitive to the arousal-dampening effects of alcohol and anxiolytics (e.g., Conrod *et al.*, 1998; Stewart & Pihl, 1994). High-SS young people, on the other hand, are more sensitive to stimulant-

induced rewards (Brunelle *et al.*, 2004; Conrod *et al.*, 1997; Erblich & Earleywine, 2003; Sher *et al.*, 2000; Zuckerman & Kuhlman, 2000). This model has less explanatory value for users high in HOP or IMP. While we did not manipulate or measure pharmacological sensitivity, my dissertation's findings offer additional, indirect support for this model and its applicability to AS and SS. The AS and SS pathways described in the preceding model (i.e., affect regulation) suggest that these traits exert their influence via a pharmacological vulnerability that affects user's motives for misuse (i.e., via a psychopharmacological vulnerability). Because high-AS youth are sensitive to the anxiety-reducing properties of alcohol, sedatives, and tranquilizers, they are motivated to self-medicate their anxiety with these substances. Because high-SS youth are sensitive to drug-induced rewards, they are motivated to take stimulants recreationally for their positively reinforcing effects. The subjective experience of taking a stimulant (e.g., euphoria, stimulation, arousal; Lambert, McLeod, & Schenk, 2006) matches high-SS users' desires and motives (i.e., for high stimulation, enhancement, positive affect regulation; Cooper, 1994).

Deviance Proneness Model

This model conceptualizes substance use as a deviant behaviour (Sher, 1991; Sher & Slutske, 2003). IMP is associated with comorbid antisocial and addictive behaviour (Castellanos-Ryan & Conrod, 2011). My dissertation's findings offer additional support for this model, and its applicability to IMP. In Study 3, IMP's relationships with sedative/tranquilizer and opioid misuse were fully mediated by IMP's association with CD symptoms. In the case of stimulant misuse, CD symptoms were again implicated as a mediator – along with ADHD symptoms (see the proceeding model; i.e., psychological dysregulation). My dissertation has also advanced this theory. In Study 2, IMP predicted

sedative/tranquilizer, opioid, and stimulant misuse (i.e., unconstrained PD misuse). In Study 3 (where my model included mediated paths through CD), IMP was not directly associated with the misuse of any of these PD classes. This suggests that IMP predicts unconstrained PD misuse by way of a general deviance proneness. This deviance includes many forms of antisocial behaviour, as captured by the diagnostic criteria of CD.

Psychological Dysregulation Model

This model suggests that adversity leads to substance misuse in liable young people (Tarter *et al.*, 2003; Tarter *et al.*, 1999). Psychological dysregulation (including IMP) makes it more difficult to adapt (cognitively, behaviourally, or emotionally) to environmental challenges. My dissertation's findings offer additional support for this model, and its applicability to IMP. Specifically, I have put forth that the psychological dysregulation model is epitomized in youth with ADHD symptoms – as this disorder is characterized by IMP and persistent, distressing dysregulation (APA, 2013). In Study 3, IMP's relationship with stimulant misuse was partially mediated by ADHD symptoms. My dissertation has also advanced this theory. In Study 2, IMP predicted the use, medically-sanctioned use, and misuse of stimulants. This suggests that youth who are high in IMP, and who have ADHD-related symptoms, are using prescription stimulants to self-medicate their dysregulation. This pathway appears relevant to both medically-sanctioned stimulant use (i.e., the medical treatment of ADHD) and non-medical stimulant misuse (i.e., the self-medication of ADHD symptoms and cognitive difficulties). Stimulants are prescribed to treat ADHD (Cortese *et al.*, 2013) but are meant to be taken regularly (Svetlov *et al.*, 2007). Thus, taking stimulants "as a study aid" constitutes misuse. Moreover, ADHD's diagnostic criteria does not include the presence

of negative affect (e.g., anxiety or depressive symptoms; APA, 2013). The self-medication of ADHD symptoms is therefore theoretically distinct from the negative affect regulation model described above. It is better characterized by the misuse of prescription stimulants to cope with psychological dysregulation.

Summary

My dissertation supports Castellanos-Ryan & Conrod's (2012) contention that all four of the etiological addiction models described above can explain personality's effect on alcohol and PD misuse. I have confirmed this in both adolescence and emerging adulthood. The onset and development of substance misuse, however, is known to be multi-determined. Castellanos-Ryan & Conrod (2012) suggested that personality influences substance use motives, which then affect substance misuse and co-occurring mental health disorders. Their model is depicted in Figure 1.1. My dissertation supports a slightly modified model (depicted in Figure 7.1), with the following changes.

First, I have emphasized mental health symptoms as an important, early risk factor for substance misuse. Personality is a robust predictor of mental health symptoms (Adan *et al.*, 2016; Smith *et al.*, 2007). Mental health affects substance use motive endorsement. Anxiety and depression, for example, are differentially related to DCWA and DCWD (Grant *et al.*, 2007b). Then, motivational profiles predict the misuse of both alcohol (Kong & Bergman, 2010) and PDs (Bennett & Holloway, 2017). To better reflect mental health symptoms' central role, I have re-arranged the model's component variables. My dissertation supports paths from (1) personality to (2) co-occurring mental health disorders to (3) substance use motives to (4) alcohol and PD misuse. Because Study 1 and 2 were cross-sectional, further testing of the proposed directionality is

required. Study 3 began to do this by using a semi-longitudinal design to temporally separate personality from mental health symptoms and PD misuse. Second, my results allow for increased specificity with respect to the substances to which each personality is most readily drawn. Castellanos-Ryan & Conrod (2012) included a spectrum from sedatives to stimulants. In contrast, I have linked AS to alcohol and sedatives/tranquilizers, HOP to alcohol and opioids, SS to stimulants, and IMP to unconstrained PD misuse (i.e., misuse of all three drug classes). Finally, I have tentatively added paths from AS to mood disorders and from HOP to anxiety disorders. Castellanos-Ryan & Conrod (2012) included an arrow from AS down to negative affect regulation (a path that was shared with HOP). As described in the earlier section entitled "Discrepancies between studies", Study 1 suggests that the pathways from internalizing traits to substance misuse are partially specific while Study 3 suggests that they are fully specific. An additional arrow was therefore added from HOP to anxiety. Both arrows were dashed, to represent the ambiguity of my results on whether these more general paths should be included. More research is needed on this point.

In sum, my dissertation supports the following modified pathways. AS likely affects substance misuse via a psychopharmacological sensitivity to sedative and anxiolytic drug effects. AS is associated with anxiety disorders, self-medication motives, and the misuse of alcohol, sedatives, and tranquilizers by means of increased sensitivity to these drugs' stress-response-dampening effects. HOP affects substance misuse via a pathway characterized by negative affect regulation. HOP is associated with mood disorders, self-medication motives, and the misuse of alcohol and opioids. SS affects substance misuse via a psychopharmacological vulnerability to stimulant drug effects.

Finally, IMP affects substance misuse via both deviance proneness and psychological dysregulation. High-IMP youth with co-occurring CD engage in unconstrained PD use. Those with co-occurring ADHD engage in stimulant misuse to manage their dysregulation.

Clinical Implications

The relationship between mental health and substance use disorders can be explained using several theoretical models (Conrod & Stewart, 2005). In the first, chronic substance abuse is thought to contribute to psychopathology. In the second, psychiatric symptoms are thought to cause, exacerbate, or maintain substance abuse. In the third, their relationship is thought to be bidirectional. In the fourth, they are linked by an underlying variable. Personality, for example, could be conceptualized as a third variable contributing to both psychiatric and addictive psychopathology (Adan *et al.*, 2016; Castellanos-Ryan & Conrod, 2012; Pihl & Peterson, 1995; Smith *et al.*, 2007). My dissertation supports a combination of models 2 and 4. I am suggesting that a third variable (i.e., personality) predicts mental health symptoms, which then predict substance misuse. Model 2 would suggest treating mental health symptoms over addiction issues. But simultaneous attention to comorbid disorders is more effective than therapies that target either diagnosis alone (Stice, Shaw, Bohon, Marti, & Rohde, 2009). Model 4 advocates for early, personality-targeted intervention that can effectively reduce both sets of symptoms. Focusing on personality-related risk further avoids complications related to comorbidity at later stages of both disorders (Conrod & Stewart, 2005).

Personality traits are differentially associated with specific mental health symptoms (Davis, Cohen, Davids, & Rabindranath, 2015; Kotov, Gamez, Schmidt, &

Watson, 2010), substance use motives (Cooper *et al.*, 1995), patterns of coping (Connor-Smith & Flachsbart, 2007), sensitivity to drug-effects (Conrod *et al.*, 1998; Leyton, 2002), and drugs of choice (Conrod *et al.*, 2000a). By addressing these vulnerability factors, personality-matched interventions enhance individual relevance and impact. Moreover, "treatment-matching" suggests that therapeutic impact increases when clients are appropriately matched to tailored interventions (compared to those who are not so matched; Miller & Cooney, 1994). Brief AS-, HOP-, SS-, and IMP-specific protocols have been developed for Canadian youth (Conrod *et al.*, 2006) like those in my samples. Each intervention includes three main components.

The first component is psycho-educational. Youth learn about their personality profile and its associated problematic coping behaviours (Bien, Miller, & Tonigan, 1993). The second component is motivational. Youth weigh the short- and long-term consequences of behavioural strategies. The third component is cognitive-behavioural. Youth learn how to identify and challenge their personality-specific cognitive distortions. In the AS intervention, students learn to better manage their anxiety by challenging thoughts related to catastrophic thinking, avoidance, and interpersonal dependence (Barlow & Craske, 1988; Conrod *et al.*, 2000b). In the HOP intervention, students learn to better manage their depression by challenging thoughts related to internalization, generalization, pessimism, and withdrawal (Barlow, 1985; Conrod *et al.*, 2000b). Based on my substantiation of a HOP to medically-sanctioned opioid use pathway, this intervention might be supplemented with pain-related cognitive restructuring (Ehde & Jensen, 2004). In the SS intervention, students learn to challenge thoughts related to boredom-susceptibility and reward-seeking (Conrod *et al.*, 2006; Conrod *et al.*, 2000b).

In the IMP intervention, students learn to better manage their externalizing symptoms by challenging impulsive, aggressive, and antisocial thoughts (Kendall & Braswell, 1985). Based on my substantiation of an IMP to ADHD symptoms to stimulant misuse pathway, this intervention might be supplemented with skills related to improving attentional focus. Alcohol and drug misuse are targeted in all four personality-matched interventions. Students explore how these behaviours, while personally relevant, constitute problematic ways of coping (Castellanos & Conrod, 2006).

An extensive body of literature supports the efficacy of personality-matched intervention. Targeting AS, HOP, SS, and IMP has decreased psychopathology and co-occurring substance misuse (O'Leary, Castellanos-Ryan, Pihl, & Conrod, 2016). Targeting AS reduces anxiety (McNally & Lorenz, 1987), targeting HOP reduces depression (Lewinsohm, Steinmetz, Larson, & Franklin, 1981), and targeting IMP reduces externalizing disorders (Kendall & Braswell, 1985; Pepler, King, & Byrd, 1991). Personality-matched intervention has also effectively reduced: alcohol misuse in adolescents (Castellanos & Conrod, 2006; Conrod & Castellanos, 2005; Conrod *et al.*, 2000a, 2006; O'Leary *et al.*, 2016); alcohol misuse in emerging adults (Watt *et al.*, 2006); illicit drug misuse in adolescents (Conrod *et al.*, 2010); and PD misuse in adults (Conrod *et al.*, 2000b).

Study 1 only assessed AS and HOP. It supported chained mediation from these traits to alcohol misuse via mental health symptoms and coping drinking motives. Thus, my results suggest that personality-targeted approaches might effectively reduce both psychopathology and alcohol misuse – a prediction that has been borne out in RCT data (O'Leary *et al.*, 2016). However, my model was only partially specific. Thus, it is unclear

whether the use of AS- and HOP-specific intervention protocols vs. general internalizing intervention protocols (e.g., the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders; Barlow *et al.*, 2011) are best indicated for use with emerging adults. Further, my results cannot speak to the effectiveness of matched (i.e., high-AS with an AS protocol) vs. mismatched (i.e., high-AS with a HOP protocol) interventions within the internalizing spectrum. Future RCTs might therefore compare internalizing personality-matched, internalizing personality-mismatched, lumped AS and HOP (i.e., the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders; Barlow *et al.*, 2011), and control groups.

Studies 2 and 3 offer preliminary support for the use of personality-matched interventions in reducing or even preventing uptake of PD misuse in adolescence and emerging adulthood. Specifically, my results suggest the possible utility of: an AS intervention for anxiety symptoms and sedative/tranquilizer misuse; a HOP intervention for depressive symptoms and opioid misuse; an SS intervention for stimulant misuse; and an IMP intervention for externalizing symptoms and general PD misuse. Castellanos & Conrod (2006) caution that even if one problematic behaviour is prevented – this effect will not necessarily generalize to other, similar behaviours. In their study, behaviours that were discussed in the intervention decreased, while behaviours that were not discussed did not. Personality-matched interventions have effectively reduced the misuse of various substances, but have yet to be adapted for PD misuse. Thus, follow-up RCTs should test the applicability of these interventions to adolescent and emerging adult PD misuse. The Canadian Institutes of Health Research-funded Canadian Underage Substance Abuse Prevention Trial (CUSP Trial; Conrod *et al.*, 2018-2023) intends to test this with high

school students and the Social Sciences and Humanities Research Council of Canada-funded Univenture Collaborative (Stewart *et al.*, 2018-2019) intends to test this with university students.

Finally, we know that the development of substance use disorders in adulthood is more likely if users were younger upon onset of regular drug use (Grant & Dawson, 1998; Lynskey *et al.*, 2003). For each year that alcohol or drug use is delayed in adolescence, rates of adult substance misuse and dependence are reduced by up to 10% (Becher *et al.*, 2001). Study 1 suggests that emerging adult university students are engaging in hazardous, harmful alcohol use. Studies 3 and 2 suggest that both adolescent high school students and emerging adult university students (respectively) are misusing sedatives/tranquilizers, opioids, and stimulants. For the interventions described above to be optimally effective, it is recommended that they be implemented early, before problematic coping mechanisms become entrenched and before at-risk youth progress from use to misuse. School-based approaches offer large, controlled, culturally-unbiased samples (Conrod *et al.*, 2010). Thus, I further recommend the use of school-based alcohol and PD uptake prevention strategies. This would also facilitate early screening and systematic follow-up.

Strengths and Limitations

My dissertation has several strengths. Its greatest strength is the stability of my findings. Personality was shown to reliably affect mental health symptoms, then substance misuse motives, then substance misuse – across drugs (alcohol vs. PDs) and developmental stages (adolescents vs. emerging adults). Studies 1-3 are progressive; the novelty and complexity of the models increases successively. Additionally, my

dissertation was theory-based. I also used large samples that allowed for powerful tests of my hypothesized models. Finally, my findings were largely consistent, despite different conceptualizations and measures of mediating variables and misuse across studies. This attests to the robustness of my findings.

Despite these strengths, my dissertation (as a whole) was limited in several ways. First, Studies 1 and 2 were cross-sectional. Study 3, while prospective, was only semi-longitudinal. True tests of mediation require at least three to four waves of data. Theory (Becker *et al.*, 2007; Cox & Klinger, 1988) and previous research (Cooper *et al.*, 2016; Stein *et al.*, 1987) suggest that personality is a determinant of drug use. But it is possible that my variables (i.e., personality, mental health symptoms, substance use motives, and substance misuse outcomes) are related via a different causal chain than that proposed in Figure 7.1. Personality, for example, has been shown to mediate the relationships between internalizing/externalizing symptoms and substance misuse (Davis *et al.*, 2015; Kotov *et al.*, 2010). It is also possible that mediation occurs in the reverse direction (i.e., from substance misuse to substance use motives to mental health symptoms to personality). To better support the etiological models of addiction reviewed herein, my models should be re-tested using fully-longitudinal designs.

Second, Study 1 only assessed AS and HOP. The extant alcohol misuse literature (and the results of Studies 2 and 3) suggest that the following pathways might have also been supported: (1) SS to enhancement motives to alcohol misuse and (2) IMP to CD symptoms to alcohol misuse. SS is robustly associated with enhancement motives (Cooper *et al.*, 1995; Comeau *et al.*, 2001; Simons *et al.*, 2005; Woicik *et al.*, 2009). SS predicts substance misuse (Mackie *et al.*, 2011) driven by the need for stimulation and

positive affect (Comeau *et al.*, 2001). Finn *et al.* (2000) found that SS was indirectly related to alcohol use problems, via alcohol use and positive alcohol expectancies. Castellanos-Ryan *et al.* (2011) concluded that the SS-binge drinking relationship was mediated by a reward response bias. IMP, on the other hand, is characterized by an inability to inhibit behaviour (Finn *et al.*, 2002). Self-reported IMP is associated with deficits in response execution and inhibition (Castellanos-Ryan *et al.*, 2011; Gay, Rochat, Billieux, d'Acremont, & Van der Linden, 2008). Poor response inhibition is a risk factor for both CD (Herba *et al.*, 2006) and substance misuse (Li *et al.*, 2006). Mackie *et al.* (2011) found that IMP predicted alcohol use via CD. Thus, Study 1's model of alcohol misuse should be replicated with the addition of these externalizing traits and these hypothesized mediational pathways.

Third, Study 3 focused on the mediating effects of mental health symptoms. It did not assess or include motives for PD misuse, however. In their review of PD misuse motives, Bennett & Holloway (2017) concluded that published studies had used different methods, examined different drug classes, and phrased items inconsistently. After synthesizing these results, Bennett & Holloway (2017) concluded that sedatives, tranquilizers, analgesics, and stimulants were predominantly misused in two ways. Youth misused PDs for personal enhancement of performance (e.g., sports, academics); mental health (e.g., more sleep, less anxiety); or physical health (e.g., to manage a pre-existing illness). They also misused PDs for pleasure (e.g., to party, get high, or experiment). Both negative reinforcement (i.e., self-medication) and positive reinforcement (i.e., recreation) motives have been associated with increased PD misuse frequency (Kelly *et al.*, 2015). Boyd *et al.* (2006) and McCabe *et al.* (2009a) have published measures of non-medical

PD use motives. It would be interesting to use these measures to test whether personality affects motives for PD misuse, like it does motives for alcohol (Cooper *et al.*, 1995) and cannabis (Hecimovic *et al.*, 2014) misuse. Like I did in Study 1 for alcohol misuse, future models of PD misuse could incorporate all four of my variables of interest (i.e., personality, mental health symptoms, PD misuse motives, and PD misuse outcomes).

Fourth, all measures in all studies were self-report. We relied on adolescents' and emerging adults' perceptions of their own personality, mental health symptoms, substance use motives, and substance misuse. The SURPS (Woicik *et al.*, 2009), K10 (Kessler *et al.*, 2002), BS1-18 (Derogatis, 2001), SDQ (Goodman, 1997), DMQ Revised Short Form (Kuntsche & Kuntsche, 2009), AUDIT (Saunders *et al.*, 1993), and DEP-ADO (Landry *et al.*, 2004) have good psychometric properties. But relying on retrospective self-report to assess personality and motives assumes that respondents are aware of their traits and the motives underlying their behaviour (e.g., substance misuse). Previous studies have reported mismatches between self-reported and observed acts (Gosling, John, Craik, & Robins, 1998) and between self-estimated and actual predictor-mood correlations (Wilson, Laser, & Stone, 1982). Personality can be more objectively measured by obtaining corroborative reports from informants. Motives can be more objectively measured by using ecological momentary assessment (Shiffman, 2009) or by observing the situations in which young people misuse substances. Relying on retrospective self-report to assess mental health symptoms further assumes that respondents are not engaging in positive or negative impression management. More objective strategies for assessing mental health include obtaining corroborative reports from informants, cross-referencing medical records, or using structured clinical

interviews (e.g., the Structured Clinical Interview for *DSM-5*; First, Williams, Karg, & Spitzer, 2015). Finally, Brener, Billy, & Grady (2003) reviewed the factors that affect the validity of adolescents' retrospective self-reported health-risk behaviours. They suggested that cognitive (e.g., retrieval issues, comprehension problems) and situational (e.g., biases related to social desirability and fear of reprisal) factors can affect the validity of youth's self-reported alcohol and drug use. They recommended asking questions more than once, using biochemical validation measures, and/or including a question on the use of a fictitious drug to weed out those "faking bad". Thus, my studies should be replicated using some of these more objective assessment strategies.

Finally, the literature suggests that measuring PD misuse accurately is particularly challenging. The outcomes of interest in Studies 2 and 3 involved PD use and misuse. Barrett *et al.*'s (2008) review suggests that PD misuse is often operationalized inconsistently. Smith *et al.*'s (2015b) review further concluded that no instruments available to date could adequately measure inappropriate medication use events or context of use. Like all studies in the field of PD misuse, these criticisms apply to my second and third studies. Once more reliable and valid PD outcome measures become available, my models should be replicated. This would reduce measurement error, thereby allowing for a stronger test of my theoretical predictions.

Directions for Future Research

In addition to the ideas discussed in each manuscript and throughout this chapter, future research could expand on my dissertation findings in the following ways. First, instead of using the SUPRS (Woicik *et al.*, 2009) to assess personality, models might incorporate specific facets of the four-factor traits (Pihl & Peterson, 1995). The Anxiety

Sensitivity Index-3 (Taylor *et al.*, 2007), for example, measures physical, cognitive, and social AS concerns. Barratt's Impulsiveness Scale-11 (Patton *et al.*, 1995), on the other hand, taps into attentional, motor, and non-planning IMP.

Second, instead of using dichotomous outcomes, substance misuse might be measured continuously. In Studies 2 and 3, I was unable to model PD misuse continuously because of the skew, kurtosis, and zero-inflation of these variables. To get around this, future studies might use selected samples (i.e., selecting for users). Models that include frequency and quantity of use, for example, could better address how personality affects severity of substance misuse (as opposed to just non-misuse vs. misuse). Future studies could also take advantage of daily diary formats to explore how personality affects substance misuse on a more micro-level. Daily diaries have been reliably used in alcohol studies (e.g., O'Hara, Armeli, & Tennen, 2015) but this design has not yet been applied to PD misuse. Electronic diaries or monitoring caps could be used to assess what, why, and how PDs were taken each day.

Third, related variables could be added to my models. In a follow-up to Study 1, for example, the remaining drinking motives (enhancement, coping, social, and conformity; Cooper, 1994) might be co-varied. In follow-ups to Studies 2 and 3, PD diversion sources and routes of administration might be modelled. In follow-ups to all studies, the moderating effects of certain demographic or environmental factors might be tested. For example, internalizing vs. externalizing pathways could be stronger for girls vs. boys (Daughters *et al.*, 2009). Environmental control, parenting, peer characteristics, academic competency, and socio-cultural context (Dick, Pagan, Viken, & Purcell, 2007; McGue, Elkins, & Iacono, 2002; Rehm *et al.*, 2004; Wills, Sandy, & Yaeger, 2002) are

examples environmental factors that could minimize or maximize the impact of personality risk on substance use behaviours or outcomes.

Fourth, my models could be replicated in non-student and clinical samples. By virtue of being in school (i.e., in high school or university), my participants may have differed from other adolescents and emerging adults who were school drop-outs, in community college, unemployed, or employed. Such participants could be recruited using targeted advertising. Clinical samples, on the other hand, would likely endorse higher substance misuse base rates (Battista *et al.*, 2013). In these samples, marginal paths may become significant. In the case of PD misuse, young people in treatment might also have active psychotherapeutic prescriptions, which they could be using appropriately and/or inappropriately. In non-student and clinical samples, attrition might be more indicative of poorer functioning.

Conclusion

In conclusion, my dissertation sought to better understand how personality affects adolescents' and emerging adults' substance misuse. In so doing, I tested tenets of established etiological addiction models (Castellanos-Ryan & Conrod, 2012). Overall, I found that (1) personality leads to (2) mental health symptoms to (3) substance use motives to (4) the use and misuse of alcohol and PDs. Specially, AS predicted anxiety symptoms, anxiety self-medication motives, and the misuse of alcohol, sedatives, and tranquilizers. HOP predicted depressive symptoms, depression self-medication motives, and the misuse of alcohol and opioids. SS predicted stimulant misuse. IMP predicted ADHD symptoms and stimulant misuse, and CD symptoms and unconstrained PD misuse. Taken together, these results offer ongoing support for the four-factor personality

vulnerability model (Pihl & Peterson, 1995) and for the affect regulation, pharmacological vulnerability, deviance proneness, and psychological dysregulation etiological addiction models (Castellanos-Ryan & Conrod, 2012). They also suggest the potential utility of the personality-targeted approach (Conrod *et al.*, 2006) for prevention of and early intervention for PD misuse in young people.

Table 7.1. Integration of Study Findings.

	Study 1 Demographic: Emerging adults Substance: Alcohol	**Study 2** Demographic: Emerging adults Substance: Prescription drugs Control: Alcohol dependence	**Study 3** Demographic: Adolescents Substance: Prescription drugs Control: Alcohol misuse
Anxiety Sensitivity	AS → symptoms of anxiety/depression → drinking to cope with symptoms of anxiety/depression → hazardous alcohol use and drinking harms. Partial evidence of specificity.	• AS → overall sedative/tranquilizer use • AS → medically-sanctioned sedative/tranquilizer use • AS → sedative/tranquilizer misuse*	• AS → anxiety symptoms → sedative/tranquilizer misuse
Hopelessness	HOP → symptoms anxiety/depression → drinking to cope with symptoms of anxiety/depression → hazardous alcohol use and drinking harms. Partial evidence of specificity.	• HOP → overall opioid use • HOP → medically-sanctioned opioid use	• HOP → depressive symptoms → opioid misuse
Sensation Seeking		• SS → overall stimulant use • SS → stimulant misuse	• SS → stimulant misuse*
Impulsivity		• IMP → overall sedative/tranquilizer and stimulant use • IMP → medically-sanctioned stimulant use • IMP → unconstrained PD misuse (i.e., sedative/tranquilizer, opiate, and stimulant misuse)	• IMP → ADHD symptoms → stimulant misuse • IMP → CD symptoms → unconstrained PD misuse (i.e., sedative/tranquilizer, opiate, and stimulant misuse)

Note. * represents marginal significance at $p \leq .06$.

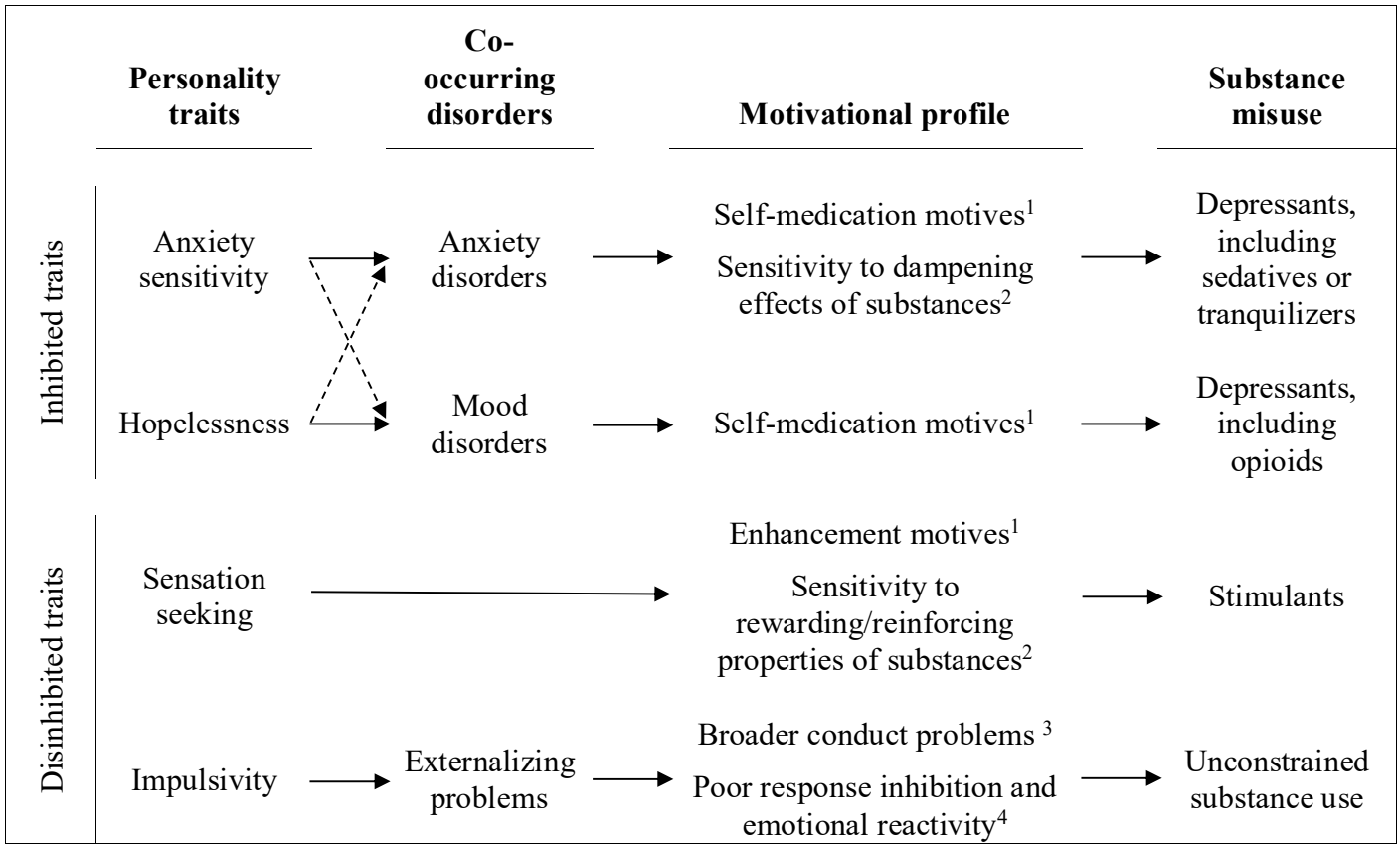

Figure 7.1. An adapted version of Castellanos-Ryan & Conrod's (2012) model of four distinct personality pathways to substance misuse and comorbid psychopathology. [1]offers support for the affect regulation model, [2]offers support for the psychopharmacological vulnerability model, [3]offers support for the deviance proneness model, and [4]offers support for the psychological dysregulation model.

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APPENDIX A

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APPENDIX B

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Title: Neurotic Personality Traits and Risk for Adverse Alcohol Outcomes: Chained Mediation through Emotional Disorder Symptoms and Drinking to Cope

Author: A. Chinneck, K. Thompson, K. S. Dobson, et al

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BACK CLOSE WINDOW

APPENDIX C

Substance Use Risk Profile Scale (SURPS)

Please indicate the extent to which you agree with the following statements by circling the appropriate response on the scale:

#	Item	Strongly Disagree	Disagree	Agree	Strongly Agree
1	I am content.	1	2	3	4
2	I often don't think things through before I speak.	1	2	3	4
3	I would like to skydive.	1	2	3	4
4	I am happy.	1	2	3	4
5	I often involve myself in situations that I later regret being involved in.	1	2	3	4
6	I enjoy new and exciting experienced even if they are unconventional.	1	2	3	4
7	I have faith that my future holds great promise.	1	2	3	4
8	It's frightening to feel dizzy or faint.	1	2	3	4
9	I like doing things that frighten me a little.	1	2	3	4
10	It frightens me when I feel my heart beat change.	1	2	3	4
11	I usually act without stopping to think.	1	2	3	4
12	I would like to learn how to drive a motorcycle.	1	2	3	4
13	I feel proud of my accomplishments.	1	2	3	4
14	I get scared when I'm too nervous.	1	2	3	4
15	Generally, I am an impulsive person.	1	2	3	4
16	I am interested in experience for its own sake even if it's illegal.	1	2	3	4
17	I feel that I'm a failure.	1	2	3	4
18	I get scared when I experience unusual body sensations.	1	2	3	4
19	I would enjoy hiking long distances in wild and uninhabited territory.	1	2	3	4
20	I feel pleasant.	1	2	3	4
21	It scares me when I'm unable to focus on a task.	1	2	3	4
22	I feel I have to be manipulative to get what I want.	1	2	3	4
23	I am very enthusiastic about my future.	1	2	3	4

APPENDIX D

Kessler Psychological Distress Scale (K10)

During the past 30 days, about how often did you feel…

#	Item	All of the Time	Most of the Time	Some of the Time	A Little of the Time	None of the Time
1	Tired out for no good reason?	1	2	3	4	5
2	Nervous?	1	2	3	4	5
3	So nervous that nothing could calm you down?	1	2	3	4	5
4	Hopeless?	1	2	3	4	5
5	Restless or fidgety?	1	2	3	4	5
6	So restless that you could not sit still?	1	2	3	4	5
7	Depressed?	1	2	3	4	5
8	So depressed that nothing could cheer you up?	1	2	3	4	5
9	That everything was an effort?	1	2	3	4	5
10	Worthless?	1	2	3	4	5

APPENDIX E

Adapted version of the Drinking Motive Questionnaire (DMQ) Revised Short Form

Below is a list of reasons people sometimes give for drinking alcohol. Thinking of all the times you drank this term, how often would you say that you drank for each of the following reasons?

#	Item	Almost Never/ Never	Some of the Time	Half of the Time	Most of the Time	Almost Always/ Always
1	Because I like the feeling	1	2	3	4	5
2	Because it helps you enjoy a party	1	2	3	4	5
3	To forget my worries	1	2	3	4	5
4	Because I feel more self-confident or sure of myself	1	2	3	4	5
5	To get a high	1	2	3	4	5
6	Because it helps me when I am feeling nervous	1	2	3	4	5
7	Because it's fun	1	2	3	4	5
8	Because it makes a social gathering more enjoyable	1	2	3	4	5
9	To cheer me up when I'm in a bad mood	1	2	3	4	5
10	To be liked	1	2	3	4	5
11	Because it helps me when I am feeling depressed	1	2	3	4	5
12	To reduce my anxiety	1	2	3	4	5
13	To fit in with a group I like	1	2	3	4	5
14	Because it improves parties and celebrations	1	2	3	4	5
15	So I won't feel left out	1	2	3	4	5

APPENDIX F

Alcohol Use Disorders Identification Test (AUDIT)

For the next set of questions, a standard drink is defined as a regular bottle of beer (355 ml or 12 oz.); a glass of wine (150mL or 5 oz.); or a shot of liquor such as vodka, gin, scotch, bourbon, brandy or rum (44 mL or 1.5 oz.). A cooler counts as 1.5 standard drinks and a King Can of beer counts as 2 standard drinks.

#	Item	0	1	2	3	4
1	How often do you have a drink containing alcohol?	Never	Monthly or less	2-4 times per month	2-3 times per week	4+ times per week
2	How many drinks containing alcohol do you have on a typical day when you are drinking?	1 or 2	3 or 4	5 or 6	7-9	10+
3	How often do you have six or more drinks on one occasion?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
4	How often during the last year have you found that you were not able to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
5	How often during the last year have you failed to do what was normally expected from you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
6	How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
7	How often during the last year have you had a feeling of guilt or remorse after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
8	How often during the last year have you been unable to remember what happened the night before because you had been drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
9	Have you or someone else been injured as a result of your drinking?	No		Yes, but not in the last year		Yes, during the last year
10	Has a relative or friend, or a doctor of other health worker been concerned about your drinking or suggested you cut down?	No		Yes, but not in the last year		Yes, during the last year

APPENDIX G

Measure of Alcohol Harms

How often have you experienced the following as a result of using alcohol this term?

#	Item	Never	Once or Twice	3-5 Times	More than 5 Times
1	Got nauseated or vomited	0	1	2	3
2	Passed out	0	1	2	3
3	Had a hangover	0	1	2	3
4	Had memory loss	0	1	2	3
5	Was criticized for drinking	0	1	2	3
6	Poor academic performance	0	1	2	3
7	Missed a class	0	1	2	3
8	Let someone down	0	1	2	3
9	Felt remorse or guilt	0	1	2	3
10	Felt down or depressed	0	1	2	3
11	Considered attempting suicide	0	1	2	3
12	Damaged property	0	1	2	3
13	Thought I had a substance problem	0	1	2	3
14	Been in trouble with authorities	0	1	2	3
15	Gotten into an argument	0	1	2	3
16	Gotten into a physical fight	0	1	2	3
17	Engaged in sexual activity that I otherwise would not have	0	1	2	3
18	Been hurt or injured	0	1	2	3
19	Had a trip to the hospital	0	1	2	3
20	Was taken advantage of sexually	0	1	2	3
21	Took advantage of someone else sexually	0	1	2	3
22	Did something I regretted	0	1	2	3
23	Tried unsuccessfully to stop using	0	1	2	3
24	Drove a car while under the influence of alcohol	0	1	2	3
25	Rode with someone who was under the influence of alcohol	0	1	2	3
26	Had financial troubles	0	1	2	3
27	Lost belongings	0	1	2	3

APPENDIX H

Supplementary tables

Table H.1. Study 1 Supplementary Correlation Matrix

	2	3	4	5	6	7	8	9	10
1. AS	**.24**	**.39**	**.38**	**.20**	**.16**	**-.11**	**.12**	.03	**.01**
2. HOP	1.00	**.47**	**.66**	**.29**	**.29**	**-.13**	**.14**	-.02	.04
3. Anxiety		1.00	**.70**	**.36**	**.32**	-.05	**.24**	-.03	**.01**
4. Depression			1.00	**.32**	**.36**	**-.08**	**.22**	-.02	.04
5. DCWA				1.00	**.67**	**.22**	**.35**	-.03	.004
6. DCWD					1.00	**.19**	**.39**	.002	-.01
7. Hazardous Alcohol Use						1.00	**.48**	**-.10**	-.05
8. Drinking Harms							1.00	-.04	.02
9. Site								1.00	**.09**
10. Age									1.00

Note. AS is Anxiety Sensitivity (range = 5-15) and HOP is Hopelessness (range = 7-35). Both were measured using the SURPS (Woicik *et al.*, 2009). Anxiety (range = 4-20) and Depression (range = 6-30) were measured using the K10 (Kessler *et al.*, 2002). DCWA is Drinking to Cope with Anxiety (range = 3-15) and DCWD is Drinking to Cope with Depression (range = 3-15). Both were measured using the DMQ Revised Short Form (Kuntsche & Kuntsche, 2009). Hazardous Alcohol Use (range = 0-12) was measured using the AUDIT-3 (Saunders *et al.*, 1993). Drinking Harms included 27 potential harms (range = 0-27). Site and Age were the covariates. Bold correlations are significant at $p < .05$.

Table H.2. Study 2 Supplementary Correlation Matrix

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. AS	**.22**	**-.21**	**.15**	**.11**	**.08**	.05	**.10**	**.07**	**.06**	**.06**	.03	.03	.02	-.01	.01	**-.20**
2. HOP	1.00	**-.10**	**.21**	**.12**	**.09**	**.09**	**.08**	**.07**	.03	**.10**	.05	**.08**	**.08**	-.003	**.05**	**-.06**
3. SS		1.00	**.29**	.01	-.03	**.14**	-.03	-.05	.003	.04	.02	**.15**	**.15**	-.03	.04	**.18**
4. IMP			1.00	**.10**	**.06**	**.17**	.05	-.003	**.05**	**.10**	**.10**	**.17**	**.25**	.04	.04	**.09**
5. Any Sed/Tranq Use				1.00	**.16**	**.25**	**.74**	**.11**	**.13**	**.65**	**.12**	**.21**	**.12**	**.05**	**.08**	**-.08**
6. Any Opioid Use					1.00	**.11**	**.07**	**.84**	-.01	**.16**	**.51**	**.13**	**.07**	**-.08**	.03	**-.09**
7. Any Stimulant Use						1.00	**.15**	-.002	**.44**	**.20**	**.20**	**.89**	**.24**	-.007	**.06**	.02
8. Medically-Sanctioned Sed/Tranq Use							1.00	**.10**	**.12**	-.02	-.03	**.11**	.06	.03	**.07**	**-.06**
9. Medically-Sanctioned Opioid Use								1.00	-.03	.04	-.05	.01	.04	**-.06**	-.001	**-.08**
10. Medically-Sanctioned Stimulant Use									1.00	**.06**	.03	-.03	.05	.02	**.07**	.02
11. Sed/Tranq Misuse										1.00	**.22**	**.19**	**.11**	.05	.03	-.04
12. Opioid Misuse											1.00	**.21**	**.07**	-.05	**.06**	-.04
13. Stimulant Misuse												1.00	**.24**	-.02	.03	.01
14. Alcohol Dependence													1.00	.03	-.002	.01

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
15. Cohort													1.00	-.03	-.03
16. Age														1.00	.03
17. Gender															1.00

Note. AS is Anxiety Sensitivity (range = 5-15), HOP is Hopelessness (range = 7-35), SS is Sensation Seeking (range = 6-30), and IMP is Impulsivity (range = 5-25). All were measured using the SURPS (Woicik *et al.*, 2009). Sed is Sedative and Tranq is Tranquilizer. Alcohol Dependence was assessed using the AUDIT (Saunders *et al.*, 1993) and scored dichotomously. Alcohol Dependence, Cohort, Age, and Gender were the covariates. Bold correlations are significant at $p < .05$.

Table H.3. Study 3 Supplementary Correlation Matrix

	2	3	4	5	6	7	8	9	10	11	12	13
1. G9 HOP	**.27**	**.32**	0.03	**.63**	**.47**	**.38**	**.30**	**.46**	**.35**	**.33**	**.22**	**.05**
2. G9 AS	1.00	**.19**	**-.12**	**.28**	**.38**	**.16**	**.08**	**.23**	**.33**	**.14**	**.05**	-.01
3. G9 IMP		1.00	**.25**	**.28**	**.22**	**.57**	**.50**	**.22**	**.18**	**.45**	**.41**	**.07**
4. G9 SS			1.00	.02	-.001	**.16**	**.18**	-.01	-.02	**.12**	**.16**	**.10**
5. G9 Depression				1.00	**.75**	**.32**	**.30**	**.63**	**.48**	**.25**	**.20**	.03
6. G9 Anxiety					1.00	**.30**	**.27**	**.50**	**.57**	**.25**	**.16**	.03
7. G9 ADHD						1.00	**.44**	**.25**	**.24**	**.68**	**.35**	**.07**
8. G9 CD							1.00	**.24**	**.22**	**.35**	**.59**	**.13**
9. G10 Depression								1.00	**.73**	**.29**	**.24**	**.07**
10. G10 Anxiety									1.00	**.29**	**.20**	**.06**
11. G10 ADHD										1.00	**.40**	**.06**
12. G10 CD											1.00	**.10**

	14	15	16	17	18	19	20	21	22	23	24	25
1. G9 HOP	**.09**	**.08**	**.07**	**.11**	**.09**	**.11**	**.07**	**-.07**	.03	**.23**	-.02	**-.05**
2. G9 AS	.02	-.02	-.03	-.01	-.01	.02	.01	**-.10**	**.06**	**.26**	.000	.01
3. G9 IMP	**.08**	**.10**	**.11**	**.11**	**.12**	**.11**	**.14**	-.003	.03	-.02	.01	.02
4. G9 SS	**.10**	**.12**	**.13**	**.11**	**.11**	**.14**	**.13**	.03	**.04**	**-.10**	-.02	**.04**
5. G9 Depression	**.13**	**.08**	**.09**	**.11**	**.08**	**.11**	**.08**	**-.07**	**.05**	**.32**	-.01	-.04

	14	15	16	17	18	19	20	21	22	23	24	25
6. G9 Anxiety	**.11**	**.05**	**.06**	**.06**	.04	**.08**	**.06**	-.04	**.04**	**.27**	.02	-.01
7. G9 ADHD	**.07**	**.07**	**.10**	**.09**	**.10**	**.11**	**.13**	-.01	.01	**.06**	**-.07**	.004
8. G9 CD	**.12**	**.14**	**.13**	**.13**	**.14**	**.17**	**.17**	**-.04**	**.06**	-.03	**.05**	.01
9. G10 Depression	**.09**	**.06**	**.07**	**.16**	**.10**	**.08**	**.10**	**-.06**	**.05**	**.30**	.01	-.03
10. G10 Anxiety	**.10**	**.05**	**.06**	**.11**	**.07**	**.06**	**.10**	**-.06**	**.05**	**.28**	-.01	.02
11. G10 ADHD	**.05**	**.05**	**.09**	**.09**	**.10**	**.08**	**.12**	.01	.003	.02	**-.07**	.02
12. G10 CD	**.11**	**.09**	**.14**	**.14**	**.14**	**.13**	**.17**	**-.05**	.04	**-.07**	.02	.04
13. G9 Opioid Misuse	**.22**	**.30**	**.29**	**.14**	**.24**	**.35**	**.15**	-.004	**.08**	**-.04**	-.01	.03
14. G9 Sed/Tranq Misuse	1.00	**.14**	**.17**	**.26**	**.15**	**.14**	**.09**	**.06**	**.04**	**.05**	.02	.03
15. G9 Stimulant Misuse		1.00	**.20**	**.10**	**.46**	**.20**	**.16**	.002	**.07**	.02	**.04**	.02
16. G10 Opioid Misuse			1.00	**.21**	**.30**	**.12**	**.22**	.03	.03	**-.05**	-.02	.01
17. G10 Sed/Tranq Misuse				1.00	**.17**	**.17**	**.17**	-.02	.01	.02	-.02	.03
18. G10 Stimulant Misuse					1.00	**.20**	**.27**	-.001	**.06**	-.003	.03	**.08**
19. G9 Alcohol Misuse						1.00	**.25**	.01	**.06**	.01	-.03	**.06**
20. G10 Alcohol Misuse							1.00	.03	.04	-.01	-.03	.03
21. School								1.00	**-.09**	-.03	**-.10**	**.10**
22. Age									1.00	-.02	**.06**	.03
23. Gender										1.00	-.02	.02
24. Ethnicity											1.00	**-.08**
25. Socioeconomic Status												1.00

Note. G9 is Grade 10 and G10 is Grade 10. HOP is Hopelessness (range = 7-35), AS is Anxiety Sensitivity (range = 5-15), SS is Sensation Seeking (range = 6-30), and IMP is Impulsivity (range = 5-25). All were measured using the SURPS (Woicik *et al.*, 2009). Depression (range = 0-24) and Anxiety (range = 0-24) were assessed using the BSI-18 (Derogatis, 2001). ADHD is Attention-Deficit Hyperactivity Disorder (range = 0-10) and CD is Conduct Disorder (range = 0-10). Both were assessed using the SDQ (Goodman, 1997). Sed is Sedative and Tranq is Tranquilizer. PD misuse was assessed using the DEP-ADO (Landry *et al.*, 2004) and scored dichotomously. Alcohol Harms (range = 0-10) were assessed using the DEP-ADO (Landry *et al.*, 2004). Socioeconomic Status was rated using a 10-point Likert scale (Currie *et al.*, 1997) with higher scores representing greater wealth. Alcohol Misuse, School, Age, Gender, Ethnicity, and Socioeconomic Status were the covariates. Bold correlations are significant at $p < .05$.

APPENDIX I

Copyright permission to include Study 2

Title: Personality and prescription drug use/misuse among first year undergraduates

Author: A. Chinneck,K. Thompson,I.T. Mahu,P. Davis-MacNevin,K. Dobson,S.H. Stewart

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BACK CLOSE WINDOW

APPENDIX J

Measure of Prescription Drug Involvement

1. Have you used any prescription drugs at all this term? (Including those prescribed by a doctor)?

   ☐ Yes
   ☐ No

If "no", skip to next section.

---

2. How often have you used painkillers (i.e., opiates such as Codeine, OxyContin, or Percocet) this term?

   ☐ Never
   ☐ Monthly or less
   ☐ 2-4 times per month
   ☐ 2-3 times per week
   ☐ 4 or more times per week

If "never", skip to question 4.

3. How have you used painkillers (i.e., opiates such as Codeine, OxyContin, or Percocet) this term? Check all that apply.

   ☐ Used as prescribed by my doctor to treat a medical condition
   ☐ I have a prescription but sometimes do not use it as prescribed (e.g., I take more, snort pills, etc.)
   ☐ I use them without a prescription to treat a medical condition (e.g., obtain or purchased from a family member, friend, or other)
   ☐ I sometimes take prescription drugs and then drink alcohol
   ☐ I sometimes use them to get high
   ☐ I sometimes use them as a study aid (e.g., they help me stay awake, focused, or concentrate)

4. How often have you used sedatives or tranquilizers (i.e., downers, Ativan, or Xanax) this term?

   ☐ Never
   ☐ Monthly or less
   ☐ 2-4 times per month
   ☐ 2-3 times per week
   ☐ 4 or more times per week

If "never", skip to question 6.

5. How have you used sedatives or tranquilizers (i.e., downers, Ativan, or Xanax) this term? Check all that apply.

- [ ] Used as prescribed by my doctor to treat a medical condition
- [ ] I have a prescription but sometimes do not use it as prescribed (e.g., I take more, snort pills, etc.)
- [ ] I use them without a prescription to treat a medical condition (e.g., obtain or purchased from a family member, friend, or other)
- [ ] I sometimes take prescription drugs and then drink alcohol
- [ ] I sometimes use them to get high
- [ ] I sometimes use them as a study aid (e.g., they help me stay awake, focused, or concentrate)

6. How often have you used stimulants (i.e., uppers, Adderall, or Ritalin) this term?

- [ ] Never
- [ ] Monthly or less
- [ ] 2-4 times per month
- [ ] 2-3 times per week
- [ ] 4 or more times per week

If "never", skip to next section.

7. How have you used stimulants (i.e., uppers, Adderall, or Ritalin) this term? Check all that apply.

- [ ] Used as prescribed by my doctor to treat a medical condition
- [ ] I have a prescription but sometimes do not use it as prescribed (e.g., I take more, snort pills, etc.)
- [ ] I use them without a prescription to treat a medical condition (e.g., obtain or purchased from a family member, friend, or other)
- [ ] I sometimes take prescription drugs and then drink alcohol
- [ ] I sometimes use them to get high
- [ ] I sometimes use them as a study aid (e.g., they help me stay awake, focused, or concentrate)

APPENDIX K

Copyright permission to include Study 3

The following letter was sent to all co-authors: Dr. Kara Thompson, Dr. Patricia Conrod, Dr. Mohammad Afzali, Dr. Raquel Nogueira-Arjona, Mr. Ioan Mahu, and Dr. Sherry Stewart.

June 25, 2018

Dear co-authors,

I am preparing my Ph.D. thesis for submission to the Faculty of Graduate Studies at Dalhousie University, Halifax, Nova Scotia, Canada. I am seeking your permission to include a manuscript version of the following paper in the thesis:

Chinneck, A., Thompson, K., Conrod, P. J., Afzali, M. H., Nogueira-Arjona, R., Mahu, I. T., & Stewart, S. H. (Paper submitted July 2018). Personality to prescription drug misuse in adolescents: Testing affect regulation, psychological dysregulation, and deviance proneness pathways. *Journal of Consulting and Clinical Psychology*.

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Full publication details and a copy of this permission letter will be included in the thesis.

Thank you,

Annie Chinneck

Their responses (i.e., permissions) are included below.

SS

Sherry Stewart <sherry.h.stewart@gmail.com>

Yesterday, 12:32 PM

Kara Thompson <kdthomps@stfx.ca>; +6 more

Reply all

I am happy to provide this permission Annie.

Best wishes

Sherry

KT

Kara Thompson <kdthomps@stfx.ca>

Mon 6/25, 12:48 PM

Annie Chinneck; +5 more

Reply all

Hi Annie,

I confirm that I agree to the below statement regarding this manuscript.

Best,

Kara

Kara Thompson, PhD
Assistant Professor, Department of Psychology
St. Francis Xavier University
Annex 125
902-867-5338

Research Affiliate, Centre for Addictions Research of BC

KA

Kamran Afzali <k.afzali@gmail.com>

Mon 6/25, 12:26 PM

Annie Chinneck; kdthomps@stfx.ca; +4 more

Reply all

Hi Annie,
I approve the above.
My complete name Mohammad Hassan Afzali
...

RN

Raquel Nogueira <rnarjona@gmail.com>

Mon 6/25, 12:29 PM

Annie Chinneck; +5 more

Reply all

Dear Annie,
I agree to the above.
Raquel Nogueira Arjona
...

TM

Tiberiu Mahu

Today, 9:06 AM

Hi Annie,

I agree with the statement you provided.

Cheers,
Tib

Tiberiu Mahu
Ph.D. Student, Clinical Psychology
Department of Psychology and Neuroscience
Dalhousie University
Halifax, Nova Scotia
(438) 823-0341
...

APPENDIX L

Brief Symptom Inventory-18 (BSI-18)

Rate your level of distress over the last 7 days:

#	Item	Not at All	Rarely/ Occasionally	Sometimes	Often	Extremely Often
1	Faintness or dizziness	0	1	2	3	4
2	Feeling no interest in things	0	1	2	3	4
3	Nervousness or shakiness inside	0	1	2	3	4
4	Pains in heart or chest	0	1	2	3	4
5	Feeling lonely	0	1	2	3	4
6	Feeling tense or keyed up	0	1	2	3	4
7	Nausea or upset stomach	0	1	2	3	4
8	Feeling blue	0	1	2	3	4
9	Suddenly scared for no reason	0	1	2	3	4
10	Trouble getting your breath	0	1	2	3	4
11	Feeling of worthlessness	0	1	2	3	4
12	Spells of terror or panic	0	1	2	3	4
13	Numbness or tingling in parts of your body	0	1	2	3	4
14	Feeling hopeless about the future	0	1	2	3	4
15	Feeling so restless you couldn't sit still	0	1	2	3	4
16	Feeling weak in parts of your body	0	1	2	3	4
17	Thoughts of ending your life	0	1	2	3	4
18	Feeling fearful	0	1	2	3	4

APPENDIX M

Youth Self-Report Strengths and Difficulties Questionnaire (SDQ)

For each item, please mark the box for Not True, Somewhat True, or Certainly True. It would help us if you answered all items as best you can even if you are not absolutely sure. Please give your answers on the basis of how things have been for you over the last six months.

#	Item	Not True	Somewhat True	Certainly True
1	I try to be nice to other people. I care about their feelings	0	1	2
2	I am restless. I cannot stay still for long	0	1	2
3	I get a lot of headaches, stomach-aches, or sickness	0	1	2
4	I usually share with others (for example CD's, games, food)	0	1	2
5	I get very angry and often lose my temper	0	1	2
6	I would rather be alone than with people of my age	0	1	2
7	I usually do as I am told	0	1	2
8	I worry a lot	0	1	2
9	I am helpful if someone is hurt, upset, or feeling ill	0	1	2
10	I am constantly fidgeting or squirming	0	1	2
11	I have one good friend or more	0	1	2
12	I fight a lot. I can make other people do what I want	0	1	2
13	I am often unhappy, depressed, or tearful	0	1	2
14	Other people my age generally like me	0	1	2
15	I am easily distracted. I find it difficult to concentrate	0	1	2
16	I am nervous in new situations. I easily lose confidence	0	1	2
17	I am kind to younger children	0	1	2
18	I am often accused of lying or cheating	0	1	2
19	Other children or young people pick on me or bully me	0	1	2
20	I often offer to help others (parents, teachers, children)	0	1	2
21	I think before I do things	0	1	2
22	I take thinks that are not mine from home, school, or elsewhere	0	1	2
23	I get along better with adults than with people my own age	0	1	2
24	I have many fears. I am easily scared	0	1	2
25	I finish the work I'm doing. My attention is good	0	1	2

APPENDIX N

Detection of Alcohol and Drug Problems in Adolescents (DEP-ADO)

Have you used one or more of these substances in your lifetime and if so, how often?

#	Item	Never	On Occasion	About Once a Month	On Weekends or 1-2 Times a Week	3+ Times a Week, but Not Everyday	Everyday
1	Tobacco	0	1	2	3	4	5
2	Alcohol	0	1	2	3	4	5
3	Marijuana, hashish, pot, weed	0	1	2	3	4	5
4	Cocaine, crack, free base	0	1	2	3	4	5
5	Stimulants, speed, amphetamine, Benzedrine, methamphetamine	0	1	2	3	4	5
6	Heroin	0	1	2	3	4	5
7	Opiates like Codeine, Demerol, Morphine, Percodan, Methadone, Darvon, Opium, Dilaudil, Talwin	0	1	2	3	4	5
8	Psychedelics or hallucinogens like LSD, acid, mescaline, Peyote, psilocybin, DMT	0	1	2	3	4	5
9	PCP or Angel Dust	0	1	2	3	4	5
10	Ecstasy or E	0	1	2	3	4	5
11	Semorone drugs like “Nacky”	0	1	2	3	4	5
12	Non-prescribed medications like barbiturates, sedatives, “downers”, or sleeping pills like “Seconal” or “Quaaludes”	0	1	2	3	4	5
13	Tranquilizers or anti-anxiety pills like “Valium”, “Librium”, Ativan (without a prescription)						
14	Steroids (anabolic), weight loss pills, protein supplements	0	1	2	3	4	5
15	Glue, inhalants (essence), cleaning products, paint	0	1	2	3	4	5
16	Other drugs, excluding alcohol (specify which)	0	1	2	3	4	5

Have you experienced the following alcohol-related harms in your lifetime?

#	Item	No	Yes
1	Harm to your physical health caused by your alcohol use (e.g., digestive problems, overdose, infections, nasal irritation, you were injured, etc.)	0	1
2	Psychological difficulties caused by your alcohol use (e.g., anxiety, depression, difficulty concentration, suicidal thoughts, etc.)	0	1
3	Harm to your relationships with family members caused by your alcohol use	0	1
4	Harm to a friendship or a romantic relationship caused by your alcohol use	0	1
5	Problems at school because of your alcohol use (e.g., caught with alcohol or using, absences, suspension, lower grades, decreased motivation, etc.)	0	1
6	Spending too much money or losing a large amount of money due to alcohol use	0	1
7	Committing a delinquent act while under the influences of alcohol (e.g., theft, injuring someone, vandalism, dealing drugs, driving under the influence, etc.)	0	1
8	Taking risks while under the influence of alcohol (e.g., unprotected sex, sex that likely would not have happened if you had been sober, riding a bike or doing sports while intoxicated, etc.)	0	1
9	Feeling as though the same quantities of alcohol were having less effect on you than they once did	0	1
10	Discussing your alcohol use with a counsellor	0	1