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A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Epidemiology and Biostatistics

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SCREENING FOR ADULT ADHD IN ONTARIO: A CROSS-SECTIONAL STUDY
EXAMINING SEX DIFFERENCES, MENTAL HEALTH CORRELATES AND
SUBSTANCE USE

(Spine title: Screening for Adult ADHD in Ontario)

(Thesis format: Monograph)

by

Deanne Daigle

Graduate Program in Epidemiology

A thesis submitted in partial fulfillment of the requirements for the degree of Master of
Science

The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

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THE UNIVERSITY OF WESTERN ONTARIO
SCHOOL OF GRADUATE AND POSTDOCTORAL STUDIES

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Screening for Adult ADHD in Ontario: A cross-sectional study examining sex differences, mental health correlates and substance use

is accepted in partial fulfilment of the
requirements for the degree of

Master of Science

Date _____

Chair of the Thesis Examination Board

ABSTRACT

The vast majority of studies on attention-deficit/hyperactivity disorder (ADHD) are based on samples with inherent age, sex, and referral biases. Therefore, the current study used population-based data to 1) estimate the prevalence of adult ADHD (ADHD screening status as well as previous diagnosis and medication use using an ADHD screener) and co-occurring psychiatric distress and substance use in Ontario 2) examine the sex differences in ADHD screening status and co-occurring psychiatric distress and substance use and 3) model ADHD screening status as a risk factor for psychiatric distress using the 2011 cycle of the Centre for Addiction and Mental Health Monitor. A positive ADHD screen was significantly associated with psychiatric distress and substance use; however the majority of those with a positive ADHD screen did not exhibit these issues. Symptom overlap and lack of diagnosis and treatment may have contributed to the findings in this sample. Importantly, the effect of age must also be accounted for in future studies where sample size permits.

Keywords: ADHD, Adult, Ontario, CAMH Monitor, Prevalence, Sex, Psychiatric distress, Antidepressants, Anti-anxiety medications, and Substance use

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to my supervisor Dr. Evelyn Vingilis for granting me this research opportunity and for her expertise and guidance throughout. I would also like to thank Jane Seeley for her continuous support and advice, as well as my thesis committee: Dr. Robert Mann and Dr. Marnin Heisel for their helpful comments on the final draft.

I would like to thank the Centre for Addictions and Mental Health (CAMH) for providing me with the data for this project. Specifically, I would like to thank Gina Stoduto and Anca Ialomiteanu for their assistance with the data analysis. In addition, I would like to thank all study participants for their time.

I would especially like to thank the faculty in the Department of Epidemiology and Biostatistics, Western University, for providing me with the knowledge that made the completion of this work possible. Last but not least, I would like to thank my family and friends for their support and encouragement.

This research was supported by grants from the Canadian Institute for Health Research and the Government of Ontario.

For Keegan H.

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Chapter 1: ADHD Prevalence, Aetiology and Concurrent Disorders

1.1 What is ADHD?

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurobiological disease characterized by difficulties with attention, motor activity, and impulse control (American Psychiatric Association, 1994). The diagnostic criteria for ADHD in North America are outlined in the *Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV)*. This diagnosis of ADHD requires that six or more of the symptoms of inattention, hyperactivity, or impulsivity cause considerable impairment in two or more settings and have persisted for the last six months to an extent that is maladaptive and inconsistent with developmental level (APA, 1994). Furthermore, these symptoms must have occurred before the age of seven to warrant a diagnosis (APA, 1994). The clinical heterogeneity of ADHD is recognized in the *DSM-IV* by three subtype classifications based on primary symptom category endorsement. These subtypes include the predominantly inattentive subtype (ADHD-I), the predominantly hyperactive-impulsive subtype (ADHD-H) and a combined subtype (ADHD-C) (APA, 1994).

ADHD is a controversial disorder in part due to its aetiological complexity, but also because conceptualizations of the disorder have varied drastically since its inception (Toplak, Connors, Shuster, Knezevic, & Parks, 2008). Gomez and colleagues (1999) posit that no other disorder has been subject to as much renaming and reconceptualization within different versions of the *DSM*. Since its clinical emergence in 1902 (Still, 1902), ADHD has been designated by a myriad of terms including: minimal brain damage, hyperkinetic syndrome, hyperactivity and attention-deficit disorder (ADD) among others. The most notable shift in the conceptualization of ADHD occurred in the 1970s, when

attention dysfunction was presented as the defining feature of the disorder (Douglas, 1972). Although based on a progressively larger empirical foundation, the evolution of the nomenclature for the disorder from *DSM-III* to *DSM-IV* has broadened the case definition so that more individuals are diagnosed with ADHD. Furthermore, these changes reflect an adaptation from earlier definitions that stressed a more narrow focus on motor activity, to the current conceptualization which emphasizes difficulty with sustained attention and deficits in the regulation of cognitive functioning (Faraone *et al.*, 2000).

1.2 Aetiology

The aetiology of ADHD is unknown; recent findings suggest that a strong genetic link and environmental factors interact in the genesis of the disorder.

Compelling evidence of the heritability of ADHD is derived from family, adoption, and twin studies, as well as from neurophysiological and molecular genetics research.

The rate of ADHD in the biological relatives of individuals with ADHD is significantly higher in comparison to the rates of ADHD in families of children without ADHD (e.g., Faraone, Biederman, & Friedman, 2000). Specifically, 30-35% of the siblings of ADHD-diagnosed individuals also met criteria for ADHD, with a relative risk for ADHD that was approximately five times that of the estimated population prevalence of the disorder. Furthermore, when analyses were restricted to those with ADHD persisting into adolescence or young adulthood, the risk increased several fold (reviewed in Faraone *et al.*, 2000). Although the heritability estimates for ADHD are not as high as those proposed for autism, they are however substantially higher than those estimated for

other highly heritable disorders such as bipolar affective disorder and schizophrenia (NIMH Genetics Workgroup, 1997).

Results from adoption studies showed that the biological siblings and parents of non-adopted children with ADHD exhibited significantly higher rates of ADHD and associated attention problems, whereas adoptive parents of individuals with ADHD were not significantly different from parents of comparison children without ADHD (reviewed in Willcutt, 2005).

Further evidence of the biological underpinnings of the disorder come from studies of monozygotic and dizygotic twins. ADHD twin studies have found that the concordance rate was significantly higher among monozygotic pairs (58% - 82%) than same-sex dizygotic pairs (31% - 38%) (Levy, Hay, McStephen, Wood, & Waldman, 1997; Levy, McStephen, & Hay, 2001; Sherman, McGue, & Iacono, 1997; Willcutt, Pennington, & DeFries, 2000). In addition, Willcutt (2005) found that the mean heritability across a number of large-scale population-based twin studies was 73%, demonstrating that individual differences in ADHD symptoms are largely attributable to genetic influences.

Neurophysiological research indicates that the prefrontal cortex (specifically the dorsolateral prefrontal cortex and the orbital frontal cortex), the basal ganglia (striatum), the caudate nucleus, and the cerebellum play a significant role in ADHD because of their involvement in complex processes that regulate behaviour (Castellanos, 1997; Seidman, Valera & Makris, 2005).

The primary deficits in ADHD typically involve executive function. Executive function refers to a variety of cognitive processes that are implicated in managing other

cognitive functions (Elliot, 2003). Executive function includes planning, working memory, attention, problem-solving, inhibition, and task switching (Monsell, 2003). Therefore, areas primarily responsible for these functions have been implicated in the disorder (Seidman *et al.*, 2005). Specifically, under-activation in the dorsolateral prefrontal cortex and the orbital frontal cortex has been found to be responsible for inattention, disinhibition, and hyperactivity in ADHD (Giedd, Blumenthal, Molloy & Castellanos, 2006). Functional neuroimaging studies of ADHD support this theory, as these studies suggest hypoactivation in prefrontal neural processing in individuals with ADHD (Silberstein, Farrow, Levy, Pipingas, Hay & Jarman, 1998).

Another hypothesis regarding the aetiology of ADHD suggests that prenatal and perinatal damage to the striatum, an area within the basal ganglia, is responsible for deficits in executive function as the circuitry within the basal ganglia is essential for executive processes (Lou, 1996; Seidman *et al.*, 2005). The striatum has been found to be particularly vulnerable to complications during pregnancy and delivery, premature birth, and low birth weight which have been found to occur at higher than normal rates in ADHD (Sprich-Buckminster, Biederman, Milberger, Faraone & Krifcher Lehman, 1993). Furthermore, animal studies of experimentally-induced striatal lesions have demonstrated that insult to this area produces hyperactivity and poor performance on working memory and response inhibition tasks akin to that seen in ADHD (Alexander, DeLong & Strick, 1986). Lastly, stimulant medications used to treat ADHD have been found to affect the striatum, possibly via the rich source of dopaminergic synapses in this area (Dougherty, Bonab, Spencer, Rauch, Madras & Fischman, 1999; Volkow, Fowler, Wang, Ding &

Gatley, 2002). Taken together, these results implicate striatal anomalies in the pathophysiology of ADHD.

The cerebellum was originally thought to be primarily involved with motor control; however findings over the past 20 years indicate that the cerebellum is also involved in cognitive and affective processes. Interestingly, Middleton and Strick (2001) have shown cerebellar-cortical connections that provide an anatomic substrate for a cerebellar-prefrontal circuit in the pathophysiology of ADHD. In addition, reduced cerebellar volume has also been found in studies of ADHD children and cerebellar volume has been shown to be significantly and negatively correlated with attention problems (Seidman *et al.*, 2005). Therefore, anomalies in the cerebellum are also thought to be characteristic of the pathophysiology of ADHD.

Dysregulation in catecholamine neurotransmission is implicated in the pathophysiology of ADHD (Faraone & Biederman 2002). Converging evidence from animal studies of behaviour and biochemistry (Gainetdinov, Wetsel, Jones, Levin, Jaber & Caron, 1999; Giros, Jaber, Jones, Wightman & Caron, 1996; Jaber *et al.*, 1999), neuropharmacological studies of the effectiveness of methylphenidate in reducing symptoms of hyperactivity and inattention, and neuroimaging studies demonstrating the association of ADHD with executive functions and the fronto-striatal pathways dependent upon dopamine transmission (Dougherty *et al.* 1999; Krause, Dresel, Krause, Kung & Tatsch, 2000), have made the dopaminergic pathways and their candidate genes areas of intense study in ADHD (Banaschewski, Becker, Scherag, Franke & Coghill, 2010).

Molecular genetics research has found limited but suggestive evidence of a causal relationship between DNA variants in serotonin and dopamine transporter genes SLC6A3, DRD4, DRD5, HTR1B, SLC6A4 and SNAP25 and ADHD (Banaschewski *et al.*, 2010). As such, although family, adoption, and twin studies implicate a genetic component in the aetiology of ADHD, no specific DNA variants have been identified as sufficient risk factors for the development of the disorder.

Recent studies have examined the existence of rare Copy Number Variants (CNVs) in individuals with ADHD. CNVs are rare genetic duplications or deletions that are likely to directly affect gene function. A study by Williams and colleagues (2010) demonstrated that individuals with ADHD had a significantly higher burden of rare CNVs than controls. Although several rare variants have been found, a confirmed common variant for ADHD has yet to be discovered. Therefore at present, it can be concluded that a substantial number of DNA variants may be implicated in the disorder. The genetic findings to date underscore the biological basis of the disorder, however future research using large sample sizes akin to those used in studies of schizophrenia and bipolar disorder are necessary to establish genome-wide significance as hard evidence of genetic causation in ADHD.

Genetics alone are not sufficient to produce ADHD and several environmental factors have also been proposed as possibly contributing to the aetiology of the disorder. Risk factors such as diet (Feingold, 1976), ineffective parenting (Willis & Lovaas, 1977), and television exposure (Christakis *et al.*, 2004) were proposed; however, these hypotheses have since been disproven by later studies (Kavale & Forness, 1983; Obel

Henriksen, Dalsgaard, Linnet, Skajaa, & Thomsen, 2004; Stevens & Mulsow, 2006; Wolraich, Wilson & White, 1995).

Environmental factors that have demonstrated sufficient evidence of a temporal association and/or suggestive evidence of a causal relationship with ADHD include cerebral hemorrhage and traumatic brain injury (Max *et al.*, 2002; Herskovits, Megalooikonomou, Davatzikos, Chen, Bryan & Gerring, 1999), low birth weight (Breslau *et al.*, 1996), maternal smoking and alcohol use during pregnancy (Mick, Biederman, Faraone, Sayer, & Kleinman, 2002; Milberger, Biederman, Faraone, Guite & Tsuang, 1997), exposure to toxins (reviewed in Banerjee, Middleton & Faraone, 2007), maltreatment and emotional trauma (Famularo, Kinscherff & Fenton, 1992), and family psychosocial adversity (Milberger *et al.*, 1997). However, these factors may interact with parental genotype as parents who have ADHD may be more likely to smoke, give birth prematurely, use other substances, neglect their children, and face psychosocial adversity.

Because ADHD is a complex disorder, its aetiology most likely involves the heritability of specific genes and DNA variants, environmental exposures and the critical timing of such exposures. Importantly, aetiological mechanisms include not only biology and the environment, but synergistic interactions between these factors which are much more challenging to identify. Biederman and Faraone (2005) suggested that the developmental pathophysiology of ADHD can best be conceptualized as consisting of a genetic predisposition to the disorder and early environmental insults which lead to fronto-subcortical catecholamine dysfunction and ADHD in childhood. ADHD in turn may lead to later environmental exposures such as substance use and psychosocial

adversity which lead to the secondary effects of the disorder such as low-self-esteem, school failure, social disability and ultimately in unremitting cases, adult ADHD.

1.3 ADHD Prevalence

ADHD often emerges in childhood and is usually apparent during the first few years of grade school (Goldman, Genel, Bezman & Slanetz, 1998) with the majority of ADHD diagnoses being made between 4 to 12 years of age (Biederman & Faraone, 2005). ADHD had been deemed, “the most common neurobehavioral disorder of childhood” (American Academy of Pediatrics, 2000) as children with ADHD comprise up to 50% of some child psychiatric populations (Cantwell, 1996). The *DSM-IV* states that the prevalence of ADHD is about 3 to 5% among school-age children (APA, 1994). This estimate, although frequently cited, is poorly documented. For example, three reviews of ADHD in pediatric clinical settings reported prevalence estimates ranging from 1.7% to 17.8%, 3% to 6% and 4% to 12%, respectively (Brown *et al.*, 2001; Elia, Ambrosini & Rapoport, 1999; Goldman *et al.*, 1998). Furthermore, epidemiological studies that applied the *DSM-IV* criteria to school populations have yielded prevalence estimates as high as 11% to 16% (Cantwell, 1996). In their meta-analysis of the epidemiology of ADHD, Polanczyk and Rhode (2007) found that the worldwide prevalence of ADHD is around 5.29% for children and adolescents and 4.4% in adults.

Prevalence estimates of adult ADHD worldwide range from 1.0% to 7.3% (Almeida Montes, Hernandez Garcia & Ricardo-Garcell, 2007; Barbaresi *et al.*, 2004; Bitter, Simon Balint, Meszaros & Czobor, 2012; DuPaul *et al.*, 2001; Faraone & Biederman, 2005; Fayaad *et al.*, 2007; Gadow, Sprafkin, Schneider, Nolan, Schwartz & Weiss, 2007; Heiligenstein, Conyers, Berns & Miller, 1998; Kooij, Buitelaar, van den

Oord, Furer, Rijnders & Hodiament, 2005; Medina-Mora *et al.*, 2005; Murphy & Barkley, 1996; Weyandt, Linterman & Rice, 1995) and from approximately 4 to 5% in the United States (Faraone & Biederman, 2005). Yet due to the use of convenience samples with low mean ages and gender biases, results from many of these studies cannot accurately be extrapolated to the general population. Higher prevalence rates have also been documented among urban compared to rural communities (Offord, Boyle & Szatmari, 1987), although it is uncertain whether these findings indicate greater access to mental health and medical care in urban populations. Nonetheless, the substantial variation in ADHD prevalence rates appears to be largely affected by the methodological characteristics of these studies. Furthermore, Anderson (1996) states that standardized diagnostic criteria and methodology can reduce the variability in reported prevalence, even in studies of highly diverse populations such as the United States, China, and Kenya.

1.4 ADHD in Adults

Because ADHD was originally conceptualized as a disorder of childhood, debate exists around the legitimacy of the disorder in adults. However growing evidence supports the persistence of symptoms into adulthood. Longitudinal studies of individuals diagnosed with ADHD in childhood demonstrate that ADHD persists into adulthood in a substantial proportion of cases (Barkley, Fischer, Smallish, & Fletcher, 2004; Mannuzza, Klein, & Moulton, 2003; Weiss & Hechtman, 1993). Follow-up studies of children with ADHD estimate that 10 to 66% of individuals experience symptoms of the disorder throughout adolescence and into adulthood (Gittelman *et al.* 1985; Manuzza *et al.* 1993; Weiss, 1985; Weiss *et al.* 1985; Weiss & Hechtman, 1992). Furthermore, approximately

30-60% of children with ADHD will continue to meet full criteria for the disorder as adults (Biederman, 1998; Biederman, Mick & Faraone, 2000; Manuzza *et al.* 1993; Weiss, 1985; Weiss & Hechtman, 1992). Moreover, a meta-analysis of these studies revealed that as many as 65% of children with ADHD will show symptoms of sufficient severity to impair functioning in adulthood (Faraone, Biederman, & Mick, 2006).

Corroborating evidence of the legitimacy of the disorder in adulthood comes from findings from neurobiology and genetics that mirror results seen in children (Faraone, 2004). Specifically, ADHD adults show evidence of structural and functional brain anomalies akin to those found in ADHD children (Paloyelis, 2007; Valera, Faraone, Murray & Seidman, 2007). ADHD adults share similar neuropsychologic deficits, executive dysfunction and familial transmission as ADHD youth (Barkley, Murphy & Fischer, 2008; Manuzza *et al.*, 1993; Faraone, Doyle, Lasky-Su, Sklar, D'Angelo, Gonzalez-Heydrich, Kratochvil, *et al.*, 2008). ADHD adults also present similar clinical features as ADHD children and have been found to respond to the same pharmacological interventions used with younger populations (Faraone & Glatt, 2010). Finally, ADHD adults also exhibit psychiatric difficulties analogous to those found among their younger counterparts (Barkley, Murphy & Fischer, 2008).

Diagnosis of adult ADHD is particularly challenging. A developmental shift in the disorder in adulthood has been suggested, whereby overt hyperactivity is reduced but inattention and disorganization persist (Bierderman, Mick & Faraone, 2000; Faraone, Bierderman, Spender, Wilens, Seidman, Mick & Doyle, 2000). As such, the *DSM-IV* criteria for ADHD may not be applicable to adults because the type and number of

symptoms listed in the current edition (*DSM-IV*) may not accurately reflect adult behaviours (Ingram, Hetchman & Morgenstern, 1999).

Furthermore, a diagnosis of ADHD requires that symptoms be present before the age of seven (APA, 1994). Therefore, in order to be diagnosed as an adult, individuals must provide corroborating evidence from childhood either via parents, siblings, pediatricians or childhood teachers or they must retrospectively report experiencing symptoms during childhood. However, numerous studies have revealed discrepancies in self-reports of ADHD symptoms relative to other informants (Barkley, Fischer, Smallish, & Fletcher, 2002). These discrepancies may result from deficient self-awareness or a positive illusory bias- the tendency toward positive self-perception (Knouse, Bagwell, Barkley, & Murphy, 2005) often seen among ADHD children (Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007). Nonetheless, self-report measures are a commonly used method for diagnosing ADHD in adults.

As previously mentioned, prevalence rates for adult ADHD vary considerably between studies due to the characteristics of the sample and study methodology. Despite the fact that interest in adult ADHD has increased in recent years, it remains relatively under-investigated in Canada. Consequently, the prevalence of adult ADHD in Canada is currently unknown.

1.5 Sex and ADHD

Most of what we know about ADHD at present is based on studies of boys (Gershon, 2002). Much less is known about adult manifestations of this disorder, and even less is known about how symptoms and outcomes may be differentially expressed by sex in adulthood. Clinical diagnostic studies in children indicate that a considerable

discrepancy exists in the diagnosis of ADHD in childhood. The gender ratio comparing males to females has ranged anywhere from 2:1 to 9:1 (Biederman, Faraone, Keenan, Knee & Tsuang, 1990; Gittelman, Mannuzza, Shenker & Bonagura, 1985; Weiss, Hechtman, Milroy & Perlman, 1985). More recent prevalence estimates come from a nation-wide telephone survey conducted in 2003-2004 that indicated the prevalence of ADHD was 14% in 10-year-old boys and 6% in 10-year-old girls, with stimulant medication use rates of 9% and 4%, respectively (Swanson *et al.*, 2007).

However, other recent epidemiological evidence suggests that prevalence may be similar in both sexes. Diagnosis of ADHD in a representative national US survey of adults found an odds ratio of 1.6 between men and women, reflecting 5.4% of men and 3.2% of women (Kessler *et al.*, 2006), indicating an equalization of the sex distribution of the disorder in adulthood (Kessler *et al.*, 2005; McGough *et al.*, 2005). A representative student survey conducted in Atlantic Canada included an ADHD screening and found a non-significant difference between girls and boys, with a prevalence of 6.2% and 5.9%, respectively (Poulin, 2007).

The vast majority of the research on sex and ADHD was conducted with clinically-referred samples. Therefore, the preponderance of males versus females possibly reflects the higher number of referrals for males with ADHD, due to their greater propensity towards disobedience in educational settings (Gershon, 2002). Females with ADHD however tend to display inattentive and therefore less disruptive behaviour than males with ADHD. Since disruptive behaviour is likely to be a motivating force behind clinic referrals, girls are more liable to be overlooked, resulting in an underrepresentation of females in clinical samples (Gaub & Carlson, 1997). Thus,

clinically-referred girls likely display particularly disruptive behaviour, but may not accurately represent the majority of women with ADHD (Gershon, 2002). As Miller and Leger (2003) state, “recent scholarship regards the association of males with ADHD as largely mythic, proposing that the clinical imbalance derives from under-diagnosis among girls and a similar failure to identify ADHD in older women”.

Thus, the research suggests contradictory findings: some studies found higher prevalence of ADHD males to females, while other studies found no sex differences in prevalence of ADHD. Only one Canadian non-clinical study, a high school survey, was found which indicated a non-significant difference favouring girls over boys.

1.6 ADHD and Concurrent Disorders

Whether ADHD stands alone as a distinct disorder is unclear, as ADHD symptoms have been found to converge with other forms of psychopathology (Furman, 2005). The difficulty distinguishing ADHD from other pathology indicates that ADHD may not be a distinct neurological or psychological disease entity and may represent a common behavioural pathway for a plethora of emotional, psychological and/or learning difficulties (Furman, 2005). In young people, ADHD has been found to co-occur with anxiety disorders in 25-35% of cases, mood disorders in 20% of cases, and oppositional defiant disorder (ODD) and conduct disorder (CD) in 25-50% of cases (Steele, Jensen & Quinn, 2006). ADHD therefore, often co-occurs with other psychiatric disorders in child populations.

Interestingly, high levels of psychiatric difficulties in ADHD adults parallel findings from ADHD children. Studies consistently show that adult ADHD frequently co-occurs with mood, anxiety, substance use and antisocial personality disorders (Biederman

et al., 1991a; Downey, Stelson, Pomerleau, & Giordani, 1997). Shekim and colleagues (1990) studied a group of 56 adults who met the *DSM-III-R* criteria for ADHD. Of this sample, only 14% met criteria solely for ADHD; 20% had one comorbid diagnosis, 29% met criteria for two other diagnoses, 11% met criteria for three additional diagnoses, and as many as 33% suffered from four other diagnoses. As such, converging results indicate that ADHD commonly co-occurs with other mental disorders among both children and adults.

1.7 Internalizing Disorders: Mood and Anxiety Disorders

1.7.1 ADHD and Depression in Children. Depression is a mental state characterized by feelings of sadness, low mood and anhedonia, accompanied by reduced energy (APA, 1994). The association between ADHD and depression in children has been demonstrated across a number of studies (reviewed in Angold, Costello, & Erkanli, 1999) with up to 47% of clinical ADHD children cases having this comorbidity (Wilens *et al.*, 2002). Moreover, a substantial overlap between ADHD and major depressive disorder (MDD) has been reported from both population (Anderson, Williams, McGee & Silva, 1987; Bird *et al.*, 1988) and clinical studies (Biederman, Faraone, Keenan, Knee & Tsuang, 1990; Jensen *et al.*, 1988; Woolston, Rosenthal, Riddle, Sparrow, Cicchetti & Zimmerman, 1989). Reviews of the literature have concluded that the two disorders co-occur more frequently than expected by chance alone and that their relationship is bi-directional as relatively high rates of ADHD and depression have been reported in studies of both mood disorders and ADHD (Angold & Costello, 1993; Biederman *et al.*, 1992; Angold, Costello & Erkanli, 1999; Bird, Gould, & Staghezza, 1993; Butler, Arrendondo, & McCloskey, 1995; MTA Cooperative Group, 1999).

The association between ADHD and depression has been demonstrated in studies of clinically referred children and adolescents as well as in community samples. For example, results from a study by LeBlanc and Morin (2004) indicated that children 7-12 years of age with ADHD reported significantly higher scores on a self-report measure of depressive symptoms ($n = 68$). A study by Nolan and colleagues (1999) used parent and teacher checklists based on *DSM-IV* diagnostic criteria to obtain information about depressive symptoms in 222 clinic-referred children between the ages of three and 18 years of age. Results from this study showed that ADHD was significantly associated with MDD according to both parent and teacher reports (Nolan, Volpe, Gadow & Sprafkin, 1999). Souza, Pinheiro, Denardin, Mattos and Rohde (2004) examined ADHD and comorbid depression in two clinical samples consisting of three to 17 year olds and found that prevalence rates were 10.3% and 11.4% respectively. As such, studies examining clinical samples demonstrate high rates of ADHD and comorbid depression.

The association between ADHD and depression has also been demonstrated in community samples. Romano and colleagues (2005) studied a community sample of adolescents ($n = 1,201$) and found that 2.9% met diagnostic criteria for both ADHD and MDD. Also, based on parent reports in a community sample of 7,231 children attending grades one to four, Blackman, Ostrander and Herman (2005) found that 10% of participants with ADHD also had depression; hence having a diagnosis of ADHD increased the risk of having depression. Furthermore, in their study comparing children and youth with ADHD from both clinic ($n = 763$) and community ($n = 1,896$) samples, Bauermeister and colleagues (2007) found that the rates of depression were 9.27% and 22.73% in the community and clinic samples, respectively. Therefore, the concurrence of

ADHD and depression, although higher among clinical samples, is also apparent in community samples.

1.7.2 ADHD and Depression in Adults. ADHD has also been found to be associated with clinical depression in adults. Similar to studies of ADHD children, studies of ADHD adults have reported high levels of depression with a correspondingly wide range of prevalence rates in both clinic and epidemiological samples. A prospective follow-up study comparing individuals referred for clinical assessment in childhood ($n = 147$) to community controls ($n = 71$), found that adults with ADHD were at a greater risk for developing MDD, with a prevalence rate of 26% versus 12% in community controls (Fischer, Barkley, Smallish & Fletcher, 2002). A study conducted by Sobanski and colleagues (2007) compared clinic-referred adults with ADHD ($n = 70$) to age and sex matched controls ($n = 70$) to ascertain the difference in prevalence rates for Axis-I disorders. Results showed that the prevalence of depression was 55% in adults with ADHD versus 25.3% in controls (Sobanski *et al.*, 2007). Cumyn, French and Hechtman (2009) also compared ADHD to non-ADHD adults ($n = 477$) and found that the ADHD group had significantly higher rates of MDD with a prevalence of 19.2%.

Furthermore, using a nationally representative survey of adults ($n = 9282$), findings from the National Comorbidity Survey Replication demonstrated that 38.3% of ADHD adults had a concurrent mood disorder (Kessler *et al.* 2006). Concurrent ADHD and depression is related to poor long-term prognosis and psychiatric impairment (Biederman, Faraone, Keenan, & Tsuang, 1991b; Biederman, Faraone, Milberger, Guite, Mick & Chen, 1996; Ollendick & King, 1994) including higher rates of suicide (Biederman *et al.*, 1991; Lewinsohn, Rohde, & Seeley, 1994).

As indicated by the above studies, prevalence rates for comorbid ADHD and depression range extensively with higher rates found in clinical samples (Biederman *et al.*, 1992; Biederman, Faraone, *et al.*, 1991b; Biederman *et al.*, 1987; Butler, Arrendondo & McCloskey, 1995). Yet it is likely that clinical cases are more symptomatic and more impaired than the general population, resulting in a referral bias for more troubled individuals. Another reason for the considerable discrepancy in prevalence rates is the use of various measures and cut off scores for determining the presence of ADHD and depression. Some studies use third party reports in the form of checklists and questionnaires, whereas others use standardized interviews with the individual, parent, or teacher, and still others use self-report questionnaires or combinations of these measures. Therefore, prevalence rates show substantial variability across studies not only due to differing study populations, but also due to the lack of consistent measures and varying disease thresholds.

1.7.3 ADHD and Anxiety Disorders in Children.

In addition to mood disorders, anxiety disorders are also particularly prevalent among individuals with ADHD. Anxiety disorders refer to those disorders for which severe anxiety is a salient and ongoing symptom (APA, 1994). Pediatric studies of clinical and community samples from the United States have documented a concurrence between anxiety disorders and ADHD. Results from a review of the literature prior to 1998 showed that 15% to 35% of children with ADHD also exhibit considerable anxiety (Pliszka, Carlson & Swanson, 1999).

Several prevalence rates have been documented in referred pediatric samples.

Based on parental reports of children clinically referred for ADHD, the Multimodal Treatment Study of Children with ADHD Cooperative Group (1999b) found that, 33.5% of children with ADHD had comorbid anxiety disorders. A study by Karustis and colleagues (2000) examined the prevalence of comorbid anxiety using parent, teacher and child self-reports in a clinical sample of children aged 7-12 years ($n = 125$). Results from this study showed that the prevalence of comorbid anxiety was approximately 17% (Karustis, Power, Rescorla, Eiraldi & Gallagher 2000).

Similar rates of comorbid ADHD and anxiety have also been found among adolescent samples. Biederman and colleagues (1991c) assessed a sample of clinically-referred patients aged 6-17 years using structural diagnostic interviews with parents. Thirty percent of this sample met diagnostic criteria for ADHD and one or more comorbid anxiety disorders (Biederman *et al.*, 1991c). Another study compared the prevalence of ADHD and comorbid psychopathology in preschool children and school-aged youth and found similar degrees of ADHD and comorbid anxiety in both cohorts, with prevalence rates around 30% (Wilens *et al.*, 2002).

1.7.4 ADHD and Anxiety Disorders in Adults. An elevated prevalence of anxiety disorders is also present in adult ADHD. In the National Comorbidity Survey replication, Kessler and colleagues (2006) found a 47% prevalence rate of anxiety disorders among adults with ADHD. Adults with ADHD also had significantly higher odds of anxiety compared to the general population (OR = 3.7). Also, Biederman and colleagues (1993) reported on a large sample of clinic-referred adults with ADHD identified during a family study of children with ADHD, and a control group of adults without ADHD obtained through the same study. The results indicated a high incidence

of lifetime diagnoses of anxiety disorders (43% to 52%). Shekim and colleagues (1990) examined 56 clinically-referred adults who met *DSM-III-R* criteria for ADHD ($n = 56$), 53% of whom also met criteria for generalized anxiety disorder (Shekim, 1990). A clinical study conducted by Murphy and Barkley (1996) ($n = 202$) found the prevalence of anxiety in adult ADHD to be approximately 32% (Murphy & Barkley, 1996). Furthermore, the co-occurrence of ADHD and generalized anxiety appears to be robust, also existing in international populations (Souza, Pinheiro, Denardin, Mattos, & Rohde, 2004).

The vast majority of prevalence estimates for concurrent ADHD and anxiety disorders in children, adolescents, and adults are derived from clinically-referred samples and studies examining the co-occurrence of these disorders using representative samples of the general population are sparse. Therefore research on adult ADHD and concurrent disorders in the context of the general population in Canada is necessary not only to address the scarcity of population studies in the ADHD-anxiety literature, but also to investigate adult ADHD and internalizing disorders in a Canadian context.

1.8 ADHD and Externalizing Disorders

1.8.1 ADHD, Oppositional Defiant Disorder and Conduct Disorder. Along with ADHD, oppositional defiant disorder (ODD) and conduct disorder (CD) are the most frequently studied psychiatric conditions of childhood (Althoff *et al.*, 2003). The defining feature of ODD is a persistent pattern of hostile and defiant behaviour towards authority figures causing considerable impairment, whereas CD is described as a continuous pattern of aggressive behaviour that consistently violates both the rights of others and age-appropriate social norms (APA, 1994). The prevalence of ODD ranges

from 2-15% and the prevalence of CD ranges from 1.5-3.4%, with some estimates as high as 16% contingent upon the type of sample under study (Althoff *et al.*, 2003). Also, there is a higher prevalence of ODD and CD among boys than girls.

ADHD frequently co-occurs with ODD (35%) and CD (50%) (Althoff *et al.*, 2003). Comorbid disruptive disorders are so pervasive that may not be separable, with some authors suggesting that ADHD subtypes with ODD and CD should be considered (Jensen, Martin & Cantwell, 1997). Conversely, evidence also exists suggesting that ODD and CD are distinct disorders from each other and from ADHD (Loeber, Burke, Lahey, Winters & Zera, 2000).

Although the disruptive disorders (ADHD, ODD and CD) are among those most commonly seen by pediatricians, family practitioners, psychologists, and psychiatrists, they often remain underdiagnosed, misdiagnosed or untreated (Althoff *et al.*, 2003). The paucity of treatment of these disorders is concerning, since ODD and CD have been found to be associated with negative psychosocial outcomes such as substance abuse and criminality (Robins & Price, 1991; Walters & Knight, 2010). CD has been found to be a strong predictor of antisocial personality disorder (ASPD) in adulthood (Robins & Price, 1991) as several studies of clinic and population-based samples have shown that childhood conduct problems predict serious adult antisocial behaviour (Hill, 2003; Kratzer & Hodgins, 1997; Robins, 1978).

1.8.2 ADHD and Antisocial Personality Disorder. Antisocial personality disorder (ASPD) is a term used to designate a pattern of behaviours which include the failure to conform to social norms and the law, a reckless disregard for the safety of others and consistent irresponsibility among those 18 years or older (APA, 1994). A

diagnosis of antisocial personality also warrants the diagnosis of its childhood precursor conduct disorder (APA, 1994). ADHD has been found to be significantly associated with ASPD in adulthood, as longitudinal studies of ADHD children have revealed high rates of ASPD in later life (Barkley, Fischer, Smallish, & Fletcher, 2004; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Satterfield & Schell, 1997; Weiss, Hechtman, Milroy & Perlman, 1985). In their 15-year prospective follow-up study of 61 hyperactive boys and 41 controls, Weiss and colleagues (1985) found that the only *DSM-III* diagnosis that was significantly more prevalent in the probands than the comparison subjects was ASPD (23% versus 2%) ($\chi^2 = 8.22$, $df = 1$, $p < .01$). Another prospective follow-up study of clinically-referred boys ($n = 104$) showed that approximately 20% of children with ADHD enter adulthood with ASPD (Manuzza *et al.*, 1998). In an earlier study by the same authors, 27% of the probands and only 8% of the comparison subjects had ongoing ASPD ($\chi^2 = 12.50$, $df = 1$, $p < .0001$) (Manuzza *et al.*, 1993). These findings were also replicated in yet another study conducted by the same authors with an independent cohort of 104 boys. Here, 32% of the probands and 8% of the comparison subjects had ongoing ASPD ($\chi^2 = 15.11$, $df = 1$, $p < .0001$) (Manuzza *et al.*, 1991). These studies provide evidence for an association between ADHD in childhood and ASPD in adulthood.

Magnetic resonance imaging (MRI) studies of the brains of persons with ASPD suggest that these individuals also exhibit frontal cortical deficits (Raine, Lencz, Bihrlé, LaCasse & Colletti, 2000). Studies using both positron emission tomography (PET) (Goyer *et al.*, 1994; Volkow *et al.*, 1995; Raine, Meloy, Bihrlé, Stoddard, Lacasse & Buchsbaum, 1998) and single-photon emission computed tomography (SPECT) (Amen, Stubblefield, Carmicheal & Thisted, 1996; Kuruoglu, Arikan, Vural, Karatas,

Arac & Isik, 1996) have shown that poor prefrontal processing is exhibited in violent, antisocial persons, a deficit that is also a cardinal feature of ADHD. Therefore, similar abnormalities in areas that are critical in modulating emotion, arousal, and attention are characteristic of both ADHD and ASPD.

In addition to common neural deficits, both ADHD and ASPD individuals are low on the personality trait known as effortful control (Frick & Morris, 2004; Nigg, 2006). Effortful control refers to the ability to focus attention resources and to inhibit behavioural responses and is critical to emotion regulation (Frick & Morris, 2004). There is robust evidence that a temperamental vulnerability termed 'low effortful control' is a risk factor for the development of oppositional/noncompliant antisocial behaviour (Frick & Morris, 2004; Nigg, 2006).

ADHD has been shown to increase the risk for ODD and CD, thus consequently increasing the risk for the development of antisocial behaviour, as explained by developmental pathway theories that argue that antisocial behaviour evolves in a largely predictable manner (Frick & Marsee, 2006; Loeber, Green, & Lahey, 2003; Waschbusch, 2002). Therefore, it is not surprising that ASPD has been found to be significantly associated with ADHD due to corresponding features present in both disorders.

1.9 ADHD and Substance Use Disorders

Substance use refers to the ingestion of drugs of abuse, inhalants, or medications for the purpose of intoxication (APA, 1994). As outlined in the *DSM-IV*, the substance use disorders (SUDs) are divided into substance abuse disorder and substance dependence disorder. The necessary feature of substance abuse disorder is a recurrent pattern of substance use despite negative consequences related to the use of the substance

(APA, 1994). An example of substance abuse would be bingeing on alcohol or drugs. Substance dependence disorder on the other hand, is a collection of cognitive, behavioural and physiological symptoms demonstrating that the individual continues substance use despite substance-related problems including the development of tolerance and withdrawal symptoms, hence being 'dependent' on a particular substance or substances (APA, 1994). Of the SUDs, substance dependence disorder is more severe than substance abuse disorder.

Concurrent SUDs are of particular concern in the treatment of ADHD because habit-forming stimulant medication is currently the gold standard in treatment of the disorder. Given the need to identify antecedent risk factors to initiate early intervention in SUD, in addition to the clinical implications of concurrent ADHD and SUD, several authors have investigated the relationship between ADHD and SUD and have even proposed ADHD as a possible causal mechanism for the development of subsequent SUD.

In their meta-analysis of longitudinal studies that prospectively followed children with and without ADHD, Lee, Humphreys, Flory, Lui and Glass (2011) found that children with ADHD were significantly more likely to have ever used drugs, but not alcohol. In addition, children with ADHD are also more likely to develop disorders of abuse/dependence for alcohol, marijuana, cocaine, and other substances (i.e., unspecified) (Lee *et al.*, 2011). The authors' concluded that individuals with ADHD are significantly more likely to develop SUDs than those without ADHD and that this increased risk was evident despite demographic and methodological differences that varied across studies.

The relation between ADHD and SUDs is bidirectional in nature in that the prevalence of ADHD is approximately 1-5% in the general adult population, yet it affects between 11 and 35% of adults with SUD (Kalbag & Levin, 2005) and between 25 and 50% of adolescents with SUD (Wilens & Biederman, 2006). Furthermore, several studies involving adults with alcohol and drug use disorders show that from 15 to 25% referred for SUD also have ADHD (Carroll & Rounsaville, 1993; Levin, Evans & Kleber, 1998; Schubiner *et al.*, 2000; Wilens, 2004a).

A sex difference in ADHD and the risk for SUDs has also been documented. Research using both community and clinical samples suggests that compared to boys, ADHD girls have a higher risk for substance use by early adolescence (Biederman *et al.*, 1999; Disney, Elkins, McGue & Iacono, 1999). For example, as part of the Minnesota Twin project, Disney and colleagues (1999) reported trends towards higher rates of SUD within the past month in 17 year old ADHD girls compared to ADHD boys of the same age (any substance use: 73% versus 44%; SUD: 29% versus 14%, respectively). Similarly, studies by Biederman and colleagues (1991, 1999) demonstrate a greater age-corrected risk for SUD in ADHD girls relative to boys. As such, ADHD girls appear to be at greater risk for SUD than ADHD boys, yet ADHD alone nonetheless acts as a risk factor for SUD regardless of gender.

Studies suggest that compared to their non-ADHD peers, ADHD individuals appear to preferentially use drugs instead of alcohol (Biederman *et al.*, 1995; Biederman *et al.*, 1997; Molina & Pelham, 2003). However, the choice of a specific drug of abuse (e.g. cocaine over marijuana) or drug type (stimulant or depressant) has not been shown for persons with ADHD and concurrent SUD (Biederman *et al.*, 1995; Biederman *et al.*,

1997). Pharmacological treatment of ADHD has been found to attenuate the risk of subsequent SUD in some studies. For instance, a meta-analysis of the literature revealed that ADHD youth who were treated with stimulant medication had twice the reduction in risk for SUD compared to those not receiving pharmacotherapy for ADHD (Wilens *et al.*, 2003). However, other studies have found no evidence that stimulant medication increases or decreases risk for subsequent SUD (Biederman, Monuteaux, Spence, Wilens, MacPherson & Faraone, 2008). Regardless of the various reasons for use, adult ADHD has an especially negative impact on the development and course of SUD. Individuals with ADHD have been found to show an earlier onset of SUD, a more rapid progression of SUD, a greater severity of SUD, and a prolonged course of SUD (Wilens & Morrison, 2011).

As shown in the above literature review, studies of the prevalence of ADHD and concurrent disorders show a substantial variability in estimates. This, in part, may be due to an inconsistency of measures used to ascertain these estimates. In addition, the selection of cut points to determine the presence or absence of a given disorder may differ, resulting in variable figures. Moreover, some studies employed measures whose psychometric properties were wanting.

The observed variability in the prevalence of ADHD and concurrent disorders can be attributed to the clinical nature of the samples. Specifically, ADHD studies are largely based on clinical pediatric samples exhibiting significant gender discrepancies resulting in biased results that cannot be generalized to populations that do not conform to the specified characteristics of these samples. Furthermore, most ADHD studies to date have been conducted in the United States and therefore, may not be comparable to Canadian

findings. There is a marked deficit of Canadian epidemiological evidence regarding ADHD mental health correlates. As such, studies using population-based data from Canadian adults are necessary not only to address age differences, but also referral bias concerns and gender bias in order to better describe ADHD prevalence, mental health correlates and substance use in Canada.

1.10 Study Objectives

The current study used population-based data from Ontario adults to 1) estimate the prevalence of adult ADHD (using an ADHD screener), previous diagnosis and stimulant medication use correlated mental health issues and substance use in Ontario; 2) examine the sex differences in the prevalence of ADHD screener status, psychiatric distress and substance use in ADHD and 3) model ADHD screener status as a risk factor for psychiatric distress. The above aims were achieved using data gathered from the 2011 cycle of the Centre for Addiction and Mental Health (CAMH) Monitor, a cross sectional telephone survey of Ontarians 18 years of age and older.

Chapter 2: Methods

2.1 Study Design

This study was a secondary data analysis conducted using cross-sectional data from the Centre for Addiction and Mental Health (CAMH) Monitor, a large ongoing population-based survey of Ontario adults aged 18 and over collected in 2010-2011 (Ialomiteanu & Adlaf, 2012).

2.2 CAMH Monitor

2.2.1 Survey Design. Data were collected using the CAMH Monitor, an ongoing cross-sectional telephone survey of Ontario adults aged 18 and over. The survey was designed to monitor addictions and mental health issues in Ontario, including alcohol and drug consumption, public opinion on these topics, and mental health status. The 2011 CAMH Monitor was an aggregation of 12 independent monthly surveys (January to December).

2.2.2 Sampling Strategy. The target population of the current study was non-institutionalized adults 18 years of age and older residing in Ontario households during the year 2011. The sampling frame was based on adult telephone subscribers residing in Ontario who were capable of completing the interview in English. Participants were contacted via random-digit dialing and were selected from a sampling frame of all active area codes and exchanges in Ontario provided each month by the American Telephone and Telegraph (ATT) Long Lines Tape.

Since 2000, the CAMH Monitor sampling frame has consisted of listed 10-digit telephone numbers in Ontario. The numbers that are listed and selected, along with telephone numbers between or on either side of that number, are included in the sampling

frame. For example, if the selected number 416-651-8513 is published in a directory, then all numbers from 416-651-8510 to 416-651-8519 are included in the sampling frame even if they are cell phone numbers or unlisted numbers. Numbers are only excluded if they are identified “not-in-service” numbers. A computer is then used to generate a random sample of telephone numbers from this frame from which each quarterly sample is drawn. Because unlisted numbers, cell phone numbers, and newly published numbers are interspersed among published numbers in the sampling frame, this strategy provides a much more robust sample than one restricted to listed landline numbers alone (Ialomiteanu & Adlaf, 2012). Moreover, studies using exclusively landlines have been shown to underestimate several health behaviours such as binge drinking and smoking (Blumberg, Luke, & Cynamon, 2006).

The sampling design employed a stratified two-stage probability selection procedure occurring each quarter. For the purpose of this survey, the province is divided into six geographical regions: Toronto, Central West, Central East, West, East, and North. In the first stage of selection, a random sample of telephone numbers was selected from within each of the six regional strata. In stage two, one respondent aged 18 or older who was able to complete the interview in English was selected without replacement based on the most recent birthday of all household members (Ialomiteanu & Adlaf, 2012). According to O’Rourke and Blair (1983), the most recent birthday technique is a relatively non-intrusive method for producing an unbiased sample. Unanswered numbers were called back a minimum of 12 times and households that initially refused to participate were re-contacted to ensure maximum participation (Ialomiteanu & Adlaf, 2012).

Two-stage probability sampling differs in subtle ways from simple random sampling (SRS). The selection of respondents from household by most recent birthday creates a design effect (DEFF). Design effects are the ways departures of the sampling frame from simple random sampling impact statistical estimates from the sample. The DEFF is basically the ratio of the actual variance under the sampling method used, to the variance computed under the assumption of simple random sampling (Henry, 1990). The implication of using SRS formulas on estimates from complex sampling designs is the underestimation of the error and the construction of narrower confidence intervals than truly exist, resulting in false positive findings of statistical significance (Henry, 1990). As such, a greater number of type I errors (false positive statistically significant differences) are more likely.

A DEFF of 1.0 indicates that the variance of a given sample design is equivalent to the variance of a SRS and most complex survey designs tend to have DEFFs larger than one (Ialomiteanu & Adlaf, 2012). However, random-digit dialing telephone surveys typically have smaller DEFFs relative to multistage, clustered area samples (Ialomiteanu & Adlaf, 2012). DEFFs in the current study ranged from 1 to 2 (Ialomiteanu & Adlaf, 2012). Therefore, most of the statistically significant differences generated from standard statistical software would be correct, yet some may not be, especially those involving small sample sizes.

2.3 Study Population, Inclusion Criteria and Exclusion Criteria

The current study included respondents from the 2011 cycle of the CAMH Monitor ($n = 3039$). This cycle was the first to include questions regarding adult ADHD, previous ADHD diagnosis, and previous ADHD medication use. Furthermore, the 2011

cycle was also the first cycle to include questions on antisocial personality disorder. Excluded by design are Ontario households that are phoneless, which represent 1% of Ontario residents (Statistics Canada, 2011). Also excluded are those too ill or aged to be interviewed and those unable to communicate on the telephone or in English. Therefore, the CAMH Monitor is representative of non-institutionalized English-speaking Ontarians age 18 and older who are sufficiently healthy to answer a telephone ($n = 9,118,084$ from 2001 Ontario Census) (Ialomiteanu & Adlaf, 2012).

2.4 Data Collection

Data were collected from January 3rd through December 20th 2011. All interviews were conducted by trained staff from the Institute for Social Research (ISR) at York University. On average, the 2011 interviews lasted 23 minutes (range 6-71 minutes; median 22 minutes) with 90% of interviews completed within 30 minutes (Ialomiteanu & Adlaf, 2012). The interviews used computer-assisted telephone interviewing (CATI) which consists of telephone, video monitor and computer keyboard to ask questions and record participants' responses (Bondy, 1994). The computer program associated with the questionnaire controlled the presentation of items on the video monitor during the interview. The CATI system followed a programmed skip pattern and customized the wording of some items to make the interview flow smoothly and ensure consistency between interviewers. Interviews were conducted by 60 ISR interviewers, many of whom had considerable CATI experience and had completed interviews on prior CAMH surveys (Ialomiteanu & Adlaf, 2012).

CATI technology allowed for the blind supervision of interviewers while simultaneously storing participant responses in computer data files. CATI systems are

preferred over the more traditional paper and pencil questionnaires as they produce fewer errors and missing data (Cattlin & Ingram, 1988). One advantage of the CATI system over paper and pencil questionnaires is that the computer program notifies the interviewer of out of range values and interviews cannot proceed until the value error is rectified.

A matrix interview design was used in order to reduce respondent burden and maximize questionnaire content and flexibility. Here, random subsets of respondents within each panel are asked one set of questions, whereas other subsets of respondents are asked a different set of questions. The majority of the interview consists of core items which were asked of all respondents. Two interview schedules were employed for the remaining questions on the survey. Panel A represents interviews with 1,040 respondents, and Panel B represents interviews with 1,999 respondents. Only panel B was analyzed in this study as it comprised all questions pertaining to ADHD.

2.5 Response Rates

Of the 8,277 telephone numbers selected over the four quarters of 2011, 5,677 were estimated to be eligible and 3,039 respondents participated, representing an effective response rate of 51% (quarterly response rates varied from 50% to 52%). This response rate was lower than previous years (57% in 2009, 55% in 2008, 53% in 2007, and 60% in 2006) (Ialomiteanu & Adlaf, 2010). Unit response rates for the 20 surveys conducted between 1991 and 2010 were found to vary from 51% to 69% with an average of 62% (Ialomiteanu & Adlaf, 2010).

These response rates are similar to those achieved by other high quality surveys in the past decade. For example, the Canadian Alcohol and Drug Use Monitoring Survey, conducted in 2010, obtained an overall response rate of 44% (Health Canada, 2010).

Also, the Behavioural Risk Factor Surveillance System, the largest health risk survey conducted in the United States by the Centers for Disease Control and Prevention, obtained an overall response rate of 41% in 2004 (Centers for Disease Control and Prevention, 2004). Further, the University of Michigan's Survey of Consumer Attitudes found a decline in response rates from 60% in 1996 to 48% in 2003 (Curtin, Presser, & Singer, 2005).

2.6 Survey Instrument

The CAMH Monitor was written for use with the CATI interviewing system. The corresponding telephone interview averages 25 minutes in duration (Ialomiteanu & Adlaf, 2010). There are over 300 items, but no respondents were administered all items due to panel divisions and logical skip patterns designed into the survey. For example, if a respondent was asked if they consumed any alcoholic beverages in the past year and responded no, any questions regarding alcohol consumption in the last seven days would be skipped.

2.7 Measures

2.7.1 Adult ADHD Self-Report Scale (ASRS-v1.1). Developed by the World Health Organization, the Adult ADHD Self-Report Scale (ASRS- v1.1) Screener is a six-item checklist used to assess ADHD symptoms based on *DSM-IV* criteria for ADHD. The ASRS-v1.1 Screener was designed to effectively capture the three primary symptom domains of ADHD: hyperactivity, impulsivity and inattention. The ASRS-v1.1 Screener questions were selected using stepwise logistic regression analysis. This method selects the least redundant set of symptoms in an effort to maximize prediction of an external

criterion, in this case, the *DSM-IV* diagnosis of ADHD (Kessler, Adler, Gruber, Sarawate, Spencer & Van Brunt, 2007).

The wording of questions in the ASRS-v1.1 Screener differs slightly from the wording in other ADHD rating scales. Unlike the items in previous ADHD scales, the questions in the ASRS-v1.1 Screener are designed to suit adult, rather than child respondents. The language in the ASRS also provides a context for symptoms to which adults can relate, to better capture adult ADHD symptom manifestations. To this end, references to such things as play and schoolwork were deleted. Therefore by using language and context specific to adults, the ASRS-v1.1 Screener addresses the issue of variability in ADHD symptom expression between children and adults, which was a concern raised by Biederman *et al.* (2000) and Faraone *et al.* (2000) in regards to other ADHD scales.

The ASRS-v1.1 Screener measures the frequency of ADHD symptoms. The developers of the instrument employed frequency-based ratings in order to allow respondents to focus on symptom occurrence, rather than on symptom severity. The ASRS-v1.1 Screener has an expanded rating scale of zero to four, which allows more accurate discrimination of symptom frequency. For example, the previously combined ‘never or rarely’ response option from other ADHD rating scales, was separated into: 0 ‘never’, 1 ‘rarely’, 2 ‘sometimes’, 3 ‘often’, and 4 ‘very often’ on the ASRS Screener. The optimal scoring approach for the ASRS-v1.1 Screener involves summing the items’ numeric response options (0-4). This method yields a summary score with a theoretical range of 0-24 (as opposed to the 0-6 scoring approach which was found to be less discriminative) (Kessler *et al.*, 2007).

Using the 0-24 scoring approach with a cutoff score of 14, the ASRS-v1.1 Screener demonstrated high concurrent validity, as the Screener was found to have a strong concordance with clinician diagnosis with an area under the receiver operating characteristic curve (AUC) of 0.90 (Kessler *et al.*, 2007). The ASRS-v1.1 Screener has a sensitivity of 0.65 ($SE = 0.23$), specificity of 0.94 ($SE = 0.3$), a positive predictive value (PPV) of .50, and a negative predictive value (NPV) of .97 (Kessler *et al.*, 2007). Overall, the ASRS-v1.1 Screener had a total classification accuracy of 0.92 ($SE = 0.38$) (Kessler *et al.*, 2007). These findings show that the ASRS-v1.1 is better at ruling in adult ADHD than ruling it out. However, a more recent study by Hines and colleagues (2012) compared the ASRS-v1.1 Screener and the Conner's Adult ADHD Self Report Scale Self Report-Short Version (CAARS-S: S) in a primary care setting. These authors found that the ASRS showed a sensitivity of 1.0, a specificity of 0.71, a PPV of 0.52, and a NPV of 1.0 (Hines, King & Curry, 2012). The high sensitivity suggests that cases of adult ADHD are rarely missed by the ASRS-v1.1. Furthermore, the moderately high specificity and NPV of 0.99 suggest that the ASRS-v1.1 Screener does not identify someone as having adult ADHD in they in fact do not. Therefore, these values show that the ASRS-v1.1 Screener would rarely miss someone with adult ADHD and would also be successful at discounting non-cases.

In order to assess the test-retest reliability of the ASRS-v1.1 Screener, Kessler and colleagues (2007) administered the Screener at baseline, again six months to one year after initial screening, and once more one to three months after the second screening. This allowed the authors to calculate the correlations between times one, two and three. The ASRS-v1.1 Screener was found to have test-retest reliabilities ranging from .58- .77

(Kessler *et al.*, 2007). The ASRS-v1.1 Screener was found to have internal consistency reliabilities (Cronbach's alphas) ranging from .63- .72, demonstrating acceptable intercorrelations between items on the Screener (Kessler *et al.*, 2007). The authors note however that very high Cronbach's alphas would not be expected because the ASRS Screener questions were selected by stepwise logistic regression analysis, a method which optimizes inconsistency among items in a way that would be reflected in low estimates of internal consistency (Kessler *et al.*, 2007). In addition to its applicability in assessing adult ADHD, the sound psychometric properties of the six-question ASRS-v1.1 Screener make it a useful screening tool for epidemiological research.

Variables **adh1-adh6** represented the six ASRS Screener items. The ASRS Screener items are presented in Appendix A.

Response options to variables **adh1** to **adh6** were 1 'never', 2 'rarely', 3 'sometimes', 4 'often' and 5 'very often' in the Monitor. These variables were recoded to correspond to the Kessler and colleagues (2007) response options, 0 'never', 1 'rarely', 2 'sometimes', 3 'often', and 4 'very often' mentioned above. Responses to each question were then summed, yielding summary scores with a theoretical range of 0-24. A derived variable delineating positive from negative ADHD screens was created using a summary score threshold of 13/14, as it corresponds to the optimal cutoff score for case-finding using the 0-24 scoring approach recommended by Kessler and colleagues (2007).

Kessler and colleagues (2007) also recommended that the 0-24 summary scores be classified according to four strata denoting high negative to high positive Screener scores. A derived variable was created with four values: 1 'high negative, 2 'low negative, 3 'low positive and 4 'high positive' (**strata**). The high negative stratum

represented participants with ASRS Screener scores 0-9; the low negative, scores 10-13; the low positive, scores 14-17; and the high positive, scores greater than or equal to 18.

2.7.2 Previous ADHD diagnosis and medication use. Previous ADHD diagnosis was assessed by the item ‘have you ever been diagnosed with Attention Deficit Disorder (ADD) or Attention Deficit Hyperactivity Disorder (ADHD) by a doctor or health care professional?’ (**adh11**).

Drawn from the Ontario Student Drug Use and Health Survey (Paglia-Boak *et al.*, 2012), the item ‘have you ever been treated with MEDICATION for ADHD or ADD by a doctor or health care professional?’ (**adh12**) assessed previous ADHD medication use.

2.7.3. General Health Questionnaire (GHQ-12). The GHQ-12 is a self-report screening instrument that measures current mental health status while focusing on two areas: the inability to execute normal ‘healthy’ functions and the appearance of novel and distressing experiences. The questionnaire asks respondents about their recent experience of particular psychological symptoms and probes whether these symptoms are worse than usual. The GHQ-12 is intended for adults 16 and over and consists of 12 questions balanced in agreement sets. That is, half of the items are worded positively and the other half is worded negatively. Responses are categorized along a four point Likert scale with response options 0 ‘not at all’, 1 ‘no more than usual’, 2 ‘rather more than usual’, and 3 ‘much more than usual’. Items **gq1** through **gq12** in the CAMH Monitor correspond to the GHQ-12 items.

Lewis and Wessely (1990) found that the convergent validity between the GHQ-12 and the Hospital Anxiety and Depression Scale was 0.74. Furthermore, Goldberg and colleagues (1997) studied the validity of the GHQ-12 in 15 countries and found no

significant differences in the validity of results by age, sex and education or between developed and developing countries.

In a comprehensive review of the GHQ-12, Vieweg and Hedlund (1983) found that Cronbach's alpha coefficients ranged from .82 to .90 (Vieweg & Hedlund, 1983; Goldberg *et al.*, 1997). Clinical assessments of psychiatric illness have been found to be directly proportional to the number of symptoms reported on the GHQ-12, demonstrating good external consistency (Goldberg & Huxley, 1980). The median sensitivity and specificity drawn from 17 studies was 83.7% and 79.0% respectively (Goldberg *et al.*, 1997).

There are three scoring approaches for the GHQ-12, a binary method (ex. 0-0-1-1), a Likert method (ex. 0-1-2-3) and the C-GHQ method. The standard method of scoring the GHQ is the binary method where symptomatic responses to each item are scored '1' and summed over the items, resulting in a score ranging from 0-12 (Goldberg *et al.*, 1997). The Likert scoring method involves assigning response scores of 0-3 to each item. These scores are then summed across items, giving an overall score ranging from 0 to 36 (Goldberg *et al.*, 1997). Another scoring method, the C-GHQ method, scores positive items in the binary method and negative items are scored 0-1-1-1, thus assuming that the 'no more than usual' response to negative questions indicates the presence of a chronic problem rather than good health (Goldberg *et al.*, 1997). In all three scoring methods, higher scores indicate an increased likelihood of psychiatric distress.

Goldberg and colleagues (1997) found that the Likert and C-GHQ scoring methods offer no advantage over the simpler binary scoring method for the GHQ-12. The same study found 1/2 to be the optimal threshold with an ROC of 0.88, 83.5% sensitivity

and 75.1% specificity (Goldberg *et al.*, 1997). However, in their study using the CAMH Monitor to determine optimal GHQ-12 threshold values for the Ontario population, Mann and colleagues (2011) identified 4 as the value at which both sensitivity and specificity are maximized. This cut-off provided estimates of prevalence of probable anxiety and mood disorder (psychiatric distress) in the Ontario population nearly identical to those found in the Canadian adult population with the CIDI (Rush *et al.*, 2008).

Therefore, after summing GHQ-12 responses using the binary scoring method, a cut-off score of 4 was used to delineate cases of psychiatric distress from non-cases.

2.7.4 Psychotropic medication use. Two items addressed psychotropic medication use. One item corresponded to antidepressant medication use, ‘in the past 12 months have you taken any prescription medication to treat depression?’ (**ps11**) and the other to anxiolytic medication use, ‘in the past 12 months have you taken any prescription medication to reduce anxiety or panic attacks?’ (**ps16**). Both items had binary response options ‘yes’ and ‘no’.

2.7.5 The Antisocial Personality Disorder Scale from the Mini-International Neuropsychiatric Interview (MINI-APD). The Mini-International Neuropsychiatric Interview (MINI) is a screening tool used to identify individuals in need of further mental health assessment (Sheehan *et al.*, 1998). The MINI consists of a structured diagnostic interview comprised of 120 questions, and assesses the presence of the 19 most common *DSM-IV* and *International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10)* disorders, 17 Axis I disorders, one Axis II disorder (antisocial personality disorder) and suicidal ideation/attempts. Items are answered dichotomously as either ‘yes’ or ‘no’.

The current study was concerned solely with the antisocial personality disorder module from the MINI. The MINI-APD module consists of 12 behavioural questions largely drawn directly from the diagnostic criteria for ASPD and from the diagnostic criteria for conduct disorder prior to age 15 (a necessary requirement for diagnosis of ASPD). The ASPD section involves two areas of interest. In the first section, subjects are asked about six specific problematic childhood misbehaviours; if two or more questions are endorsed, subjects are subsequently asked about six antisocial behaviours since age 15. Three or more of these behaviours are required for a diagnosis of ASPD.

The MINI was found to have acceptably high validity, inter-rater and test-retest reliability scores for most disorders when compared with both the Structured Clinical Interview for *DSM-III-R* (SCID) and the CIDI (Pinninti, Madison, Musser & Rissmiller, 2003; Lecrubier, Sheehan, Hergueta, & Weiller, 1997; Sheehan *et al.*, 1997). Sheehan and colleagues (1998) found kappas over 0.70 for most of the psychiatric diagnoses and only a single kappa value (current drug dependence) being under 0.50. Sensitivity was 0.70 for all disorders except dysthymia, obsessive-compulsive disorder, and current drug dependence. PPVs were above .75 for major depression, lifetime mania, current and/or lifetime panic disorder, lifetime agoraphobia, lifetime psychotic disorder, anorexia, and posttraumatic stress disorder. However, none of these studies assessed the antisocial personality disorder module of the MINI. As such, information regarding the validity and reliability of the ASPD module is lacking.

For the present study, the MINI-APD module was shortened by one item to 11 items in total. The first section consists of only 5 items instead of 6 (items **apd1** through **apd5**). For ethical reasons, item six from the MINI ASPD module, 'before you were 15

years old did you force someone to have sex with you?’ was excluded from the CAMH Monitor and therefore from the current study. Scoring the first five items was programmed directly into the CATI interviewing system and differed from the aforementioned scoring. In order to be asked the next set of ASPD questions, participants must have endorsed greater than three questions out of the initial five (instead of two out of six). Similar to the original scoring developed by Sheehan and colleagues (1998), antisocial personality disorder was identified as present if a participant scored three or higher on the latter six questions (**apd6** through **apd11**). A dichotomous variable delineating those who screened positive from those who screened negative for ASPD was created using a scoring threshold of 5/6.

2.7.6 Alcohol Use Disorders Identification Test (AUDIT).

The Alcohol Use Disorders Identifications Test (AUDIT) was developed by the World Health Organization, Department of Mental Health and Substance Dependence to identify the presence or absence of an alcohol use disorder. The AUDIT contains 10 items and 4 subscales and takes about two minutes to administer (Babor, Biddle-Higgins, Saunders & Monteiro, 2001). Response options range from zero to four, however the phrases associated with the numeric response options vary depending on the item. A score of eight or more denotes hazardous alcohol use (Babor, Biddle-Higgins, Saunders & Monteiro, 2001).

The AUDIT has demonstrated a high degree of internal consistency across a broad range of studies. In their review of studies prior to the year 2001, Shields and Caruso (2003) calculated a median reliability of .81, with a range of .59 to .91. In a review of 18 studies published since 2002, Reinert and Allan (2007) found a comparable

median reliability coefficient of .83, with a range of .75 to .97. Using the standard cut-point of eight, three studies were conducted with general population samples and reported test-retest reliability kappas of .70, .86, and .89 respectively (Dybek, Bischof, Grothues, Reinhardt, Meyer, Hapke, *et al.*, 2006; Rubin, Migneault, Marks, Goldstein, Ludena & Friedman, 2006; Selin, 2003).

Interclass correlations have also confirmed the stability of the test-retest reliability of the AUDIT in general population samples. Rubin and colleagues (2006) derived an interclass correlation coefficient of .87 among 102 participants from the general U.S. population who were screened by telephone with a seven day interval between screenings. In their study of 61 participants from the general Swedish population, Bergman and Kallmen (2002) reported an interclass correlation of .93 between initial screening and three to four week follow-up. Moreover, Dybek and colleagues (2006) screened 99 German general practice patients and found an interclass correlation of .95 between the initial in-person screening and a one month follow-up by telephone. The AUDIT has therefore repeatedly been shown to have good test-retest reliability.

Regarding the construct validity of the AUDIT, the stability of a two factor structure has been demonstrated across numerous studies. These studies support a 'consumption' factor (items one to three) and an 'adverse consequences of drinking' factor (items four to ten). Bergman and Kallmen (2002) found a Cronbach's alpha reliability coefficient of .69 and a test-retest reliability of .98 for the consumption factor items. Shields, Guttmanova and Caruso (2004) found Cronbach's alphas of .74 and .81 for the scores on the same consumption factor in a clinical and a college student sample.

Measures of the criterion validity of the AUDIT have varied across studies. Studies examining the concurrent and the predictive validity of the AUDIT using the *DSM-IV* criteria for alcohol abuse or dependence reported that the sensitivity of the AUDIT ranged from .67 to 1.00 and the specificity from .65 to .97 (Cherpitel, 1997; Cherpitel, 1998; Cherpitel, 2001; Clements, 1998; Cook, Chung, Kelly & Clark, 2005; Dawe, Seinen & Kavanagh, 2000; Hearne, Connolly & Sheehan, 2002; Kelly, Donovan, Chung, Cook & Delbridge, 2004; Maisto, Carey, Carey, Bordon & Gleason, 2000; McCann, Simpson, Ries & Roy-Byrne, 2000). These same studies reported positive predictive values (PPVs) and negative predictive values (NPVs) ranging from .32 to .87 and .88 to .98 respectively.

The AUDIT is scored as follows. The first response for each question (e.g. ‘never’) is scored zero. The second response option (e.g. ‘less than monthly’) is scored one, the third (e.g. ‘monthly’) is scored two, the fourth (e.g. ‘weekly’) is scored three and the last response (e.g. ‘daily or almost daily’) is scored four. Questions nine (**aud9t**) and 10 (**aud10t**) have three response options and these are scored zero, two, and four respectively. Scores are then summed to obtain a total score ranging from 0-40 (**audit**). A total score of eight or more denotes hazardous alcohol use. The variable **audit8** delineates those who screened positive for hazardous alcohol use from those who screened negative for hazardous alcohol use.

2.7.7 Alcohol, Smoking and Substance Involvement Screening Test (ASSIST).

The Alcohol and Substance Involvement Screening Test (ASSIST) was developed for the World Health Organization by an international collaborative of substance abuse researchers to screen primary care patients for psychoactive substance use and related

problems (WHO ASSIST Working Group, 2002). The ASSIST is a self-report measure consisting of eight questions, the first of which asks the respondent about any lifetime use of tobacco, alcohol, cannabis, cocaine, stimulants, inhalants, sedatives/hypnotics, hallucinogens, opioids and 'other drugs'. If the respondent answers no, the screen is complete. If the respondent answers yes however, the next seven items are administered. These items probe areas such as health, social, financial or legal problems, inability to manage routine responsibilities, expression of concern by friends or family, prior attempts to control use associated with each drug that was positively endorsed in the first question. The final question asks about intravenous drug use. Questions are rated on a five-point Likert scale. The ASSIST takes less than five minutes to administer and is designed to ascertain both lifetime and current substance use (WHO ASSIST Working Group, 2002).

In their study examining the feasibility and reliability of the ASSIST with a group of 236 participants, the WHO ASSIST Working Group (2002) found test-retest reliability coefficients ranging from .58 to .90 with retest interviews occurring one to three days after the initial screening. In general, reliabilities showed substantial agreement. The same study evaluated the internal consistency reliability of the ASSIST and found that the correlations were high for most items across all substance classes with Cronbach's alphas ranging from .73 to .92 (WHO ASSIST Working Group, 2002).

The concurrent, construct and discriminative validity of the ASSIST was examined in a multi-site international study consisting of 1047 participants (Humeniuk *et al.*, 2008). This study utilized a stratified sampling procedure to ensure balanced recruitment in regards to sex and age groups (18-25) (26-35) and (36-45) years

(Humeniuk *et al.*, 2008). Concurrent validity of the ASSIST was assessed by comparison of scores with the Addiction Severity Index-Lite (ASI-Lite), the MINI-plus, the Severity of Dependence Scale (SDS), the Revised Fagerstrom Tolerance Questionnaire (RTQ) and the AUDIT. The authors found significant positive correlations between the ASSIST and the ASI-Lite ($r = .76- .88; p < .001$), the MINI-Plus ($r = .76, p < .001$), the SDS ($r = .59, p = .001$), the RTQ ($r = .78, p < .001$) and the AUDIT ($r = .82, p < .001$) (Humeniuk *et al.*, 2008) demonstrating the concurrent validity of this instrument.

The ASSIST also showed good internal consistency with Cronbach's alpha calculations displaying good inter-item correlations for the total substance involvement score (.89) as well as for the specific substance involvement scores (.86 for cannabis) (Humeniuk *et al.*, 2008). Cannabis abuse and dependence thresholds were taken from the Technical Report of Phase II Findings of the WHO ASSIST Project (Humeniuk & Ali, 2006). The authors investigated the discriminative validity of the ASSIST by comparing ASSIST scores grouped by known standards of dependence, abuse and non-problematic use. The dependent group consisted of individuals who were recruited from drug and alcohol treatment centres and met independent clinical evaluation criteria for current dependence of specific substances. The subjects recruited from primary health care settings were classified as abusers or non-problematic users according to the presence of a diagnosis for current abuse on the MINI Plus. ASSIST Scores for Global continuum of substance use risk, and Specific Substance ASSIST scores for alcohol, cannabis, cocaine, amphetamines, sedatives and opioids were compared for all three groups using ANOVA and ROC curves. Results showed that the optimal ASSIST cut-off score for cannabis (the specific substance of interest in the current study) use and abuse was 0.96 with 91%

sensitivity, 90% specificity and an ANOVA Scheffe's differences between groups of 8.1, $p < .001$ (Humeniuk & Ali, 2006). An ASSIST cut-off score of 10.5 differentiated cannabis abuse from cannabis dependence with an AUC of .62, 57% sensitivity, 61% specificity and an ANOVA Scheffe's differences between groups of 2.2, $p < .001$ (Humeniuk & Ali, 2006). Hence the ASSIST can better discriminate use from abuse than abuse from dependence (Humeniuk & Ali, 2006).

The ASSIST was added to the CAMH Monitor in 2004 and was only employed for the assessment of cannabis use. The first item asks about lifetime use of cannabis (**cn1**). If the participant answers yes to lifetime cannabis use, subsequent items probe areas such as compulsion to use, problems and failure to function as expected and others expressing concern about use and attempts to control use are administered. The last question pertaining to intravenous drug use was excluded from the CAMH Monitor because of its irrelevance to cannabis use. Therefore, the version of the ASSIST used in this study consisted of seven rather than eight questions.

Scoring the ASSIST for specific substance involvement consists of summing participants' responses to questions two through six. Each question is rated along a five-point Likert scale. The scores associated with each response option vary depending on the question. Question two has response options: 0 'never', 2 'once or twice', 3 'monthly', 4 'weekly', and 6 'daily or almost daily'. Question three has the same response options but the numbers associated with each option range instead from 0 to 3, 4, 5 and 6. Similarly for question 4, yet the numbers are 0, 4, 5, 6 and 7 and likewise for question 5 however numeric scores are 0, 5, 6, 7 and 8. Question 5 and 6 differ in that the response options are 0 'no, never', 6 'yes, in the past 3 months' and 3 'yes, but not in the past 3 months'.

Questions two through seven of the ASSIST originally corresponded to Monitor items **ascan1**, **ascan2**, **ascan3**, **ascan4**, **ascan5** and **ascan6** with response options 0-4 (0 ‘never’, 1 ‘once or twice’, 2 ‘monthly’, 3 ‘weekly’, 4 ‘daily’). These items were recoded according to the ASSIST scoring guidelines mentioned above and made into new variables **ascan1** to **ascan6**. Items **ascan1** through **ascan6** were then summed to obtain a cannabis involvement summary score ranging from 0 to 39. A cut-off score of 1.5 for abuse was used to create the cannabis abuse variable.

2.7.8 Standard questions for alcohol, cannabis and cocaine use. Standard quantity and frequency questions pertaining to alcohol, cannabis, and cocaine consumption identical to those used in previous studies by the Addiction Research Foundation and the Centre for Addiction and Mental Health were included in the Monitor.

2.7.9 Socio-demographic information. Socio-demographic information relating to age, sex, level of education, employment status, marital status, annual household income, and household location were also collected. Age was originally recorded as a continuous variable and was later divided into four categories according to the 2006 census: 18-24, 25-44, 45-64, and 65+ (**agecen4**). Male or female sex was recorded (**sex**). Level of education was coded as less than high school, completed high school, some post-secondary education, and university degree (**educat4**). Employment status was divided into employed, unemployed, and other (**empcat8**). Marital status was coded into three categories: married/living with a partner; previously married (divorced, widowed or separated); and never married (**marstat3**) because this was a common method used in the

literature. Annual household income was divided into 5 categories (**hinccat5**). Household location was divided into two categories: rural or urban (**rur_urb**).

2.8 Secondary Data source

A secondary data source was used for this study. Secondary data are defined as data that have not been collected for the purpose of this study (Sorenson, Sebroe & Olsen, 1996). Secondary data sources have numerous advantages as they cost less, save time, and are usually more representative of the population as they often consist of large samples. In addition, they have a reduced chance of bias due to the effect of the diagnostic process or attention caused by the research question (Sorenson *et al.*, 1996). One of the major disadvantages of secondary data sources is that the researchers do not control data collection, choice of questions and data quality.

2.9 Data Analysis

All analyses were conducted using Statistical Package for Social Sciences (SPSS) 20. Variables of interest were selected from the information collected during the interviews for the 2011 cycle of the CAMH Monitor. All analyses were conducted using appropriate sampling weights to ensure estimates are representative of the Ontario general population. All significance (alpha) levels were set at .05.

Use of the Bonferroni correction is still under debate (e.g. Bender & Lange, 1999; García, 2004; Morgan, 2007; Perneger, 1998); therefore, multiple comparisons were not accounted for in the interpretation of results. However, exact p-values are reported so that readers may adjust for multiple comparisons using the Bonferroni correction of $p = .001$ for 69 comparisons conducted in this study.

2.9.1 Data cleaning and transformation. Prior to all analyses, data were subjected to data cleaning to locate out-of-range values, univariate outliers and missing data. The distributions of each variable of interest were examined for normality, skewness and kurtosis. Data were also inspected for multivariate outliers. Lastly, variables were also evaluated for multicollinearity and singularity. Diagnostic information for all variables of interest is presented in Appendix F.

2.9.2 Weighting. Since equal numbers of participants were selected from each of the six regions in Ontario, weights are required to restore population representation. The final annualized weight (**FWGHT**) used in the current study is a function of the selection weight and a post stratification adjustment.

The quarterly aggregated sampling weight variable (**RHHWGTC1-4**) consisted of four components: the relative household weight (**HHWGTC1-4**), which is equal to the proportion of household residents age 18 and older; the relative region weight (**RWGTC1-4**), which is directly proportional to the percentage of all Ontario households located in the region; survey quarter (the quarterly sampling interval) and post-strata adjustments. Cycles are weighted so that each quarterly wave makes an equal contribution to the weighted N. Quarterly relative household and relative region variables were summed into cumulative relative household (**HHWGTALL**) and relative region (**RWGTALL**) variables. Finally these variables were aggregated into the cumulative region-household variable (**RHHWGTALL**).

In order to reduce bias and adjust for non-response and non-coverage of households without telephones, probability surveys usually apply post-strata population adjustments to the base weight according to census information (Casady & Lepkowski,

1999). The post stratification adjustment was based on eight post-strata representing four age groups (18-24; 25-44; 45-64; 65+) by sex (male; female) configuration from the 2006 Census (Ialomiteanu & Adlaf, 2011). These adjustments were applied in calculating the final annualized weight (**FWGHT**) (Ialomiteanu & Adlaf, 2011). Therefore, the cumulative regional and household weight (**RHHWGTALL**) and the post-strata population adjustment weight (**postwtsa**) comprise the final annualized weight (**FWGHT**) variable used in this study. The final annualized weight (**FWGHT**) was applied to all analyses in the current study.

2.10 Descriptive and Bivariate Analyses

2.10.1 Demographic information. Preliminary analyses consisted of calculating the frequencies and 95% confidence intervals (C.I.s) of all demographic variables for the entire panel B sample. Demographic variables included in these analyses were: age, sex, marital status, education, employment, average annual household income and household location.

2.10.2 Internal consistency of measures. Next, the internal consistency of all measures used in the study was assessed. The Cronbach's alpha coefficients and inter-item correlations for the ASRS-v1.1 Screener, the GHQ-12, the AUDIT, the cannabis-specific version of the ASSIST and the MINI-APD were calculated.

2.10.3 Mental health in Ontario. The prevalence of psychiatric distress was estimated by calculating the frequency, proportion and 95% C.I.s of those who scored 4 or more on the GHQ-12. Also, the prevalence of past 12 month anti-anxiety and antidepressant medication use in Ontario was determined by calculating the frequencies, proportions and 95% C.I.s for these two variables. The prevalence of ASPD screening

status in Ontario was established by calculating the frequency, proportion and 95% C.I. of the ASPD screening status based on a cut-off score of 3 on the MINI-APD.

2.10.4 Substance use in Ontario. Hazardous drinking was defined by a score of 8 or more on the AUDIT. The frequency, proportion and 95% C.I. was calculated. The frequencies and 95% C.I.s for lifetime, past 12 month and past 3 month cannabis, marijuana or hash use were calculated along with the frequency, proportion and 95% of cannabis abuse.. The frequency, proportion and 95% C.I. of lifetime cocaine use were also calculated.

2.10.5 Adult ADHD screening status prevalence. The prevalence of a positive screen for adult ADHD in Ontario was estimated by calculating the frequency, proportion and 95% C.I. of the sample that scored 14 and above on the ASRS-v1.1 Screener (recommended by Kessler *et. al.*, 2007).

2.10.6 ADHD screening status and demographic variables. Frequencies, proportions, odds ratios (ORs) for ADHD screening status and all demographic variables (age, sex, marital status, education, employment, average annual household income and household location) were calculated along with their 95% C.I.s. Chi-square tests of independence were also performed when possible to determine whether significant differences in sex, age, marital status, education, employment, annual household income or household location existed between those who screened positive and those who screened negative for ADHD.

2.10.7 Previous ADHD diagnosis and ADHD medication use. Frequencies, proportions, chi-square tests, ORs and 95% C.I.s were calculated for ADHD screening

status and previous ADHD diagnosis, as well as previous ADHD medication use, and ADHD medication use prior to 18 years of age.

2.10.8 Adult ADHD screening status and mental health. Frequencies, proportions and ORs along with 95% C.I.s were calculated for past 12 month anti-anxiety medication use, and past 12 month antidepressant use. Chi-square tests of independence were also performed to examine if any significant differences in psychotropic medication use existed between those who screened positive and those who screened negative for ADHD.

ORs and 95% C.I.s were calculated for psychiatric distress and a chi-square test of independence was executed to determine if any significant differences in psychiatric distress existed between those who screened positive for ADHD and those who screened negative for ADHD.

The ORs and 95% C.I.s of the proportion of those who scored three or more on the ASPD module of the MINI were calculated and a chi-square test of independence was performed to investigate whether a significant difference existed in ASPD screening status rates between the ADHD and non-ADHD screened groups. The OR was also calculated along with its 95% C.I.

2. 10.9 ADHD screening status and substance use. The frequency, proportion , OR and 95% C.I. for hazardous alcohol use in both the ADHD positive and the ADHD negative screening groups were calculated and a chi-square test of independence was carried out to test for any differences in hazardous alcohol use between the two groups.

Frequencies and proportions of those who screened positive for ADHD and those who screened negative for ADHD along with the ORs and 95% C.I.s for lifetime, past 12

month, and past 3 month cannabis use as well as cannabis abuse were calculated. Chi-square tests were performed to test for significant differences between the ADHD positive and ADHD negative screening groups. The frequencies, proportions, ORs and 95% C.I.s for lifetime cocaine use were also calculated and chi-square tests were performed to test for significant differences between those in the ADHD positive and ADHD negative screener groups.

Only crude ORs could be calculated because our sample did not meet the guidelines for multinomial logistic regression that indicate a minimum of 10 cases per independent variable (Schwab, 2002). Thus the sample size was too small for the conduct of adjusted ORs. All the aforementioned analyses were also conducted stratified by sex. Moreover, differences between males and females who screened positive for ADHD, between males who screened positive and males who screened negative for ADHD, between females who screened positive and females who screened negative for ADHD as well as males and females who screened negative for ADHD were also examined.

2.11 Multivariate Analyses

Research evidence demonstrates that ADHD is a possible risk factor for psychiatric distress. As indicated in the literature review, a notably large percentage of individuals with ADHD also have at least one additional mental disorder, most commonly, depression or anxiety and this association has been observed across a numerous studies involving different populations.

In the current study ADHD was assumed to precede psychiatric distress (anxiety and depression). The theory that guided the multivariate model is that proposed by Biederman & Faraone (2005). These authors state that ADHD may lay the foundation in

childhood for subsequent adversity- both developmental and environmental, such as deficits in attention and cognition, learning disabilities, academic failure, deficits in emotion, fear and aggression regulation, as well as difficulties in parental attachment, interpersonal relationships, peer rejection and increases in impulsivity and risk-taking behaviours, all factors which may lead to the development of further psychiatric impairments in adolescence and adulthood (Biederman & Faraone, 2005). According to the *DSM-IV-TR* ADHD is a disorder of early developmental origin, while depression and anxiety have a later onset (APA, 2000). The onset of depression typically occurs during adolescence (Hankin, 2005) and increases in prevalence with age (Kessler, 2002). Ostrander and Herman (2006) found that parent management and locus of control mediated the relationship between ADHD and subsequent depression in children and adolescents, thus also modelling ADHD as a precursor to later depression. Also, depression and anxiety in ADHD may result from sequential mental health issues as for example; anxiety in ADHD may reflect concerns about competency and performance (Hankin 2006; Ostrander & Herman, 2006; Schatz & Rostain, 2006).

In the present study, we used hierarchical binary logistic regression to examine ADHD screening status as a predictor variable for psychiatric distress while controlling for age, sex, antisocial behaviour screening status and substance use. Table 1.1 displays all variables of interest in the present study.

Table 1.1

Study Variables

Variable and Type	Measurement
Exposure Variable	
ADHD Screening Status <i>Dichotomous</i>	Scores of 13 or more on the ASRS-v1.1 1 = Yes 0 = No
Outcome Variable	
Psychiatric Distress <i>Dichotomous</i>	Scores of 4 or more on the GHQ-12 using the binary scoring method 1 = Yes 0 = No
Demographic Variables	
Age <i>Categorical</i>	1 = 18-24 years old 2 = 25-44 years old 3 = 45-64 years old 4 = 65 + years old
Sex <i>Nominal</i>	1 = Male 0 = Female
Marital Status <i>Nominal</i>	1 = Married/Living with a partner 2 = Widowed, divorced, separated 3 = Never married
Education <i>Ordinal</i>	1 = Less than high school 2 = Completed high school 3 = Some post-secondary education 4 = University degree
Employment Status <i>Nominal</i>	1 = Employed 2 = Unemployed 3 = Other
Annual Household Income	1 = Less than 30,000

<i>Ordinal</i>	2 = 30, 000 - 49,000 3 = 50, 000 -79, 000 4 = 80,000 +
Household Location <i>Dichotomous</i>	1 = Urban 0 = Rural
Past 12 Month Anti-anxiety Medication Use <i>Dichotomous</i>	1 = Yes 0 = No
Past 12 Month Antidepressant Use <i>Continuous</i>	1 = Yes 0 = No
Antisocial Personality Disorder screening status <i>Dichotomous</i>	Scores of 6 or more on the MINI-APD 1 = Yes 0 = No
Substance Use Variables	
Hazardous Alcohol Use <i>Dichotomous</i>	Scores of 8 or more on the AUDIT 1 = Yes 0 = No
Lifetime Cannabis, Marijuana or Hash Use <i>Dichotomous</i>	1 = Yes 0 = No
Past 12 Month Cannabis, Marijuana or Hash Use <i>Dichotomous</i>	1 = Yes 0 = No
Past 3 Month Cannabis, Marijuana or Hash Use <i>Dichotomous</i>	1 = Yes 0 = No
Cannabis Abuse <i>Dichotomous</i>	Scores of 1.5 or more on the ASSIST 1 = Yes 0 = No
Lifetime Cocaine Use <i>Dichotomous</i>	1 = Yes 0 = No

Chapter 3: Results

3.1 Description of the Study Sample

A sample of 3039 Ontarians met eligibility criteria for this study (Panel A = 1040 and Panel B = 1999). However, only Panel B of the CAMH Monitor was selected for analyses as it comprised the items of interest in the current study. Therefore, all analyses were conducted on a weighted sample of 1999 individuals from the general population of Ontario. Only weighted results are reported herein.

The mean age of participants was 47 years old ($SD = 17$). When age was divided into four categories, the largest group based on numeric size was those between the ages of 25 to 44 years old while those 18 to 24 years old comprised the smallest age group. Slightly more than half (53%) of the sample was female. The majority of the sample (68%) were married or living with a partner. Over two thirds of the sample reported having some post-secondary education (35%) or a university degree (35%). The large majority of respondents (85%) lived in an urban area. Descriptive statistics for the full sample (Panel B) are presented below in Table 3.1.

Table 3.1

Descriptive Statistics for Demographic Variables (Panel B)

Variable	Freq.	Proportion (%)	[95% C.I.]
Age (4 categories)			
18-24	210	10.56	[9.27, 12.03]
25-44	747	37.58	[35.48, 39.79]
45-64	646	32.53	[30.48, 34.65]
65 +	324	16.29	[14.73, 18.03]
Sex			
Male	942	47.55	[45.22, 49.65]
Female	1044	52.56	[50.35, 54.78]
Marital Status			
Married/ Living with a partner	1353	68.12	[66.02, 70.17]
Previously married	210	10.57	[9.27, 12.03]
Never married	404	20.36	[18.60, 22.19]
Education			
< High school	193	9.71	[8.47, 11.13]
Completed high school	398	20.02	[18.31, 21.88]
Some post-secondary	688	34.61	[32.55, 36.79]
University degree	693	34.86	[32.80, 37.04]
Employment			
Employed	1281	64.80	[62.64, 66.90]
Unemployed	85	4.30	[3.47, 5.31]

Other	611	30.91	[28.89, 33.01]
Annual Household Income			
< 30,000	169	8.51	[7.34, 9.85]
30,000-49,000	223	11.23	[9.89, 12.72]
50,000-79,000	368	18.53	[16.86, 20.33]
80,000 +	764	38.47	[36.33, 40.66]
Don't know/Refused	462	23.26	[21.43, 25.19]
Household Location			
Rural	301	15.16	[13.63, 16.83]
Urban	1686	84.89	[83.22, 86.42]

3.2 Internal Consistency Reliability of Measures: Cronbach's Alphas

The alpha coefficients for the ASRS-v1.1 Screener, the GHQ-12, the MINI-APD, the AUDIT and the ASSIST are presented in Table 3.2.1 below.

Table 3.2.1

Cronbach's Alphas and Inter-Item Correlations of the ASRS-v1.1 Screener, the GHQ-12, the MINI APD, the AUDIT and the ASSIST

Measure	Alpha	Mean	Min.	Max.	Range	Variance	N of Items
ASRS-v1.1 Screener	.75	0.35	0.23	0.59	0.36	0.01	6
GHQ-12	.82	0.27	0.04	0.57	0.53	0.02	12
MINI-APD	.73	0.03	0.00	0.12	0.12	0.00	11
AUDIT	.78	0.35	0.14	0.67	0.53	0.02	10
ASSIST	.72	0.27	0.09	0.54	0.45	0.02	7

3.3 Descriptive Statistics and Population Proportions for Mental Health and Substance Use Variables (Full Panel B Sample)

3.3.1 Psychiatric Distress. Using the GHQ-12 with a cut-off value of 4, the prevalence of psychiatric distress in the Ontario population was 9.93% [8.66, 11.34]. The overall mean GHQ-12 score was 1.05 ($SD = 2.05$).

3.3.2 Past 12 month anti-anxiety medication use and past 12 month antidepressant use. The prevalence of past 12 month anti-anxiety medication use in the Ontario population was 7.06% [5.99, 8.30] and similarly, the prevalence of past 12 month antidepressant use in the Ontario population was 7.07% [6.00, 8.31].

3.3.3 Antisocial personality disorder screening status in Ontario. Using a cut-off score of five or more, approximately 0.71% [0.40, 1.21] of the Ontario population screened positive for antisocial personality disorder on the MINI-APD.

3.3.4 Hazardous alcohol use in Ontario. The prevalence of hazardous alcohol use in Ontario, as defined by a score of eight or more on the AUDIT, was approximately 13.06% [11.61, 14.66].

3.3.5 Cannabis, marijuana and hash use and abuse in Ontario. As shown in Table 3.3.6 below, approximately 41% of the population had used cannabis, marijuana or hash at least once in their lifetime. In addition, roughly 14% had used cannabis, marijuana or hash over the past 12 months, and nearly 10% reported using cannabis, marijuana or hash over the past three months. In their validation study of the ASSIST, the WHO suggested that scores of 1.5 or more best delineated cannabis use versus cannabis abuse (Humeniuk & Ali, 2006). Using this cut-off value, approximately 9% of the Ontario population screened positive for cannabis abuse.

Table 3.3.5

Cannabis Use in Ontario

Time Period	Freq.	Proportion (%)	[95% C.I.]
Lifetime	799	40.21	[38.31, 42.69]
Past 12 months	272	13.81	[12.33, 15.43]
Past three months	188	9.47	[8.24, 10.86]
Cannabis Abuse			
Yes	181	9.15	[7.93, 10.93]
No	1797	90.85	[89.47, 92.07]

3.3.6 Lifetime cocaine use in Ontario. Approximately 7.03% [5.96, 8.27] of the Ontario population have used cocaine at least once in their lifetime.

3.4 Description of the Study Population According to ADHD Screener Status

3.4.1 Prevalence of adult ADHD screener status. As previously mentioned, the ASRS v1.1 Screener is comprised of six questions reflecting the impairments common in adult ADHD. Response options are rated on a five-point Likert scale as follows: 0 'never', 1 'rarely', 2 'sometimes', 3 'often', and 4 'very often'. Summary scores have a possible range from 0-24. Kessler et al. (2007), the developers of the measure, proposed an optimal case finding threshold of 13/14, meaning that scores less than or equal to 13 are considered ADHD negative and scores of 14 or more are considered ADHD positive. Using this cut point, 3.47% [2.73, 4.40] of Ontario adults screened positive for ADHD on the ASRS v1.1 Screener.

3.4.2 The association of age and ADHD screener status. The average age of those who screened positive for adult ADHD was 35.59 years ($SD = 14.64$) and 46.84 years ($SD = 17.03$) for those who screened negative for ADHD. Positive ADHD Screener status was highest among those 18-24 years of age and lowest among those in the 65+ category. The relationship between ADHD Screener status and age was significant, χ^2 ($df = 3, N = 1926$) = 11.48, $p = .009$.

Table 3.4.2

Frequencies and Proportions for Age According to ADHD Screener Status

	ADHD Positive (N = 69)			ADHD Negative (N = 1857)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Age						
18-24	13	18.84	[10.79, 30.42]	197	10.61	[9.26, 12.12]
25-44	31	44.92	[33.10, 57.32]	716	38.56	[36.35, 40.82]
45-64	22	31.88	[21.47, 44.33]	624	33.60	[31.46, 35.81]
65 +	3	4.35	[1.13, 13.01]	320	17.23	[15.55, 19.04]

3.4.3 Sex and ADHD screener status. As shown in Table 3.4.3, the proportion of men and women in both the ADHD positive and ADHD negative groups were approximately equal. No significant relationship between sex and ADHD Screener status was found, χ^2 (df = 1, N = 1988) = 0.29, $p = .591$.

Table 3.4.3

Frequencies and Proportions for Sex According to ADHD Screener Status

	ADHD Positive (N = 70)			ADHD Negative (N = 1918)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Male	31	44.29	[32.60, 56.61]	912	47.55	[45.30, 48.81]
Female	39	55.71	[43.39, 67.40]	1006	52.45	[50.19, 54.70]

3.4.4 Marital status and ADHD screener status. The relationship between marital status and ADHD Screener status was significant, χ^2 (df = 2, $N = 1967$) = 12.34, $p = .002$. Based on examination of the standardized residuals, individuals with ADHD positive screen were more likely to have never been married (see Table 3.4.4 below).

Table 3.4.4

Frequencies and Proportions for Marital Status and ADHD Screener Status

	ADHD Positive ($N = 69$)			ADHD Negative ($N = 1898$)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Marital Status						
Married/ Partner	35	50.72	[38.51, 62.85]	1318	69.44	[67.33, 71.47]
Widowed/ Separated/ Divorced	9	13.04	[6.50, 23.82]	201	10.59	[9.26, 12.08]
Never Married	25	36.23	[25.25, 48.75]	379	19.97	[18.21, 21.86]

3.4.5 Education and ADHD screener status. The majority of individuals in both ADHD screening status groups reported having some post-secondary education or a university degree (see Table 3.4.5). Also, roughly 10% of both groups had not completed high school. No relationship between education and ADHD screener status was found, χ^2 (3, $N = 1971$) = 2.91, $p = .406$.

Table 3.4.5

Frequencies and Proportions for Education According to ADHD Screener Status

	ADHD Positive (<i>N</i> = 67)			ADHD Negative (<i>N</i> = 1904)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Education						
< High school	9	13.43	[6.70, 24.47]	184	9.66	[8.39, 11.10]
Completed high school	13	19.40	[11.12, 31.24]	385	20.22	[18.45, 22.11]
Some post-secondary	27	40.30	[28.72, 53.00]	660	34.66	[32.53, 36.85]
University degree	18	26.87	[17.11, 39.31]	675	35.45	[33.31, 37.65]

3.4.6 Employment and ADHD screener status. Table 3.4.6 shows the proportions for each category of employment according to ADHD Screener status. No significant difference in employment status between those who screened positive for ADHD and those who screened negative for ADHD was found, $\chi^2 (2, N = 1977) = 2.55, p = .279$.

Table 3.4.6

Frequencies and Proportions for Employment Status According to ADHD Screener Status

	ADHD Positive (N = 68)			ADHD Negative (N = 1908)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Employment Status						
Employed	48	68.57	[56.23, 78.85]	1233	64.66	[62.46, 66.80]
Unemployed	5	7.14	[2.66, 16.56]	80	4.20	[3.36, 5.22]
Other	17	24.29	[15.17, 36.27]	594	31.15	[29.09, 33.29]
Total	70	100.00	-	1907	100.00	-

3.4.7 Annual Household Income and ADHD screener status. As Table 3.4.7 indicates, similar proportions of individuals in both the ADHD positive (35%) and ADHD negative (39%) screen groups had an average household income of more than 80,000 dollars a year. No significant difference in average annual household income was found between the two groups, χ^2 (df = 4, N = 1986) = 4.67, $p = .323$.

Table 3.4.7

*Frequencies and Proportions for Annual Household Income According to ADHD**Screener Status*

	ADHD Positive (N = 69)			ADHD Negative (N = 1917)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Annual Household Income Range						
< 30,000	9	13.04	[6.50, 23.82]	160	8.35	[7.17, 9.70]
30,000-49,000	9	13.04	[6.50, 23.82]	214	11.16	[9.80, 12.68]
50,000-79,000	16	23.19	[14.22, 35.18]	352	18.36	[16.67, 20.18]
80,000 +	24	34.78	[23.98, 47.28]	740	38.60	[36.42, 40.83]
Don't know/ Refused	11	15.94	[8.60, 27.16]	451	23.53	[21.66, 25.51]

3.4.8 Household location and ADHD screener status. The majority of both the ADHD positive (87%) and ADHD negative (85%) groups lived in an urban area. No significant relationship between household location and ADHD Screener status was found, $\chi^2(1, N = 1986) = 0.237, p = .626$. Household location according to ADHD Screener status is presented in Table 3.4.8 below.

Table 3.4.8

Frequencies and Proportions for Household Location According to ADHD Status

	ADHD Positive (N = 69)			ADHD Negative (N = 1917)		
	Freq.	Proportion (%)	[95% C.I.]	Freq.	Proportion (%)	[95% C.I.]
Location						
Urban	60	86.96	[76.18, 93.50]	1626	84.82	[83.12, 86.38]
Rural	9	13.04	[6.50, 23.82]	291	15.18	[13.62, 16.88]

3.5 Descriptive Statistics and Population Proportions for Mental Health and Substance Use Variables by ADHD Screener Status

3.5.1 Previous ADHD diagnosis by ADHD screener status. Among those who screened positive for ADHD on the ASRS v1.1 Screener, 10.14% [4.52, 20.37] had previously been diagnosed with ADHD by a healthcare professional. In addition, 2.40% [1.78, 3.22] of those who screened negative for ADHD had previously been diagnosed with ADHD by a healthcare professional.

A separate cross-tabulation was conducted to disaggregate previous ADHD diagnosis according to age. Results showed that the vast majority of those previously diagnosed with ADHD were under the age of 44 with 49.02% [34.95, 63.23] belonging to the 25 to 44 year old age group, followed by 37.25% [24.47, 51.94] in the 18 to 24 year old age group, and 11.76% [4.87, 24.55] in the 45 to 64 year old age group. Only 1.96% [0.10, 11.79] of those who had previously been diagnosed with ADHD belonged to the 65 and over age group. Furthermore, previous ADHD diagnoses were most prevalent among those aged 18-24 years of age (9.05%) [5.68, 13.98], followed by those aged 25 to 44 years old (3.36%) [2.23, 4.99], those 45 to 64 years old (0.93) [0.38, 2.12], and those aged 65 and over (0.31) [0.02, 1.99].

3.5.2 Previous ADHD medication use and ADHD screener status. Of those previously diagnosed with ADHD, 83.33% [36.48, 99.12] were treated with prescription medication for the disorder. In addition, of those previously diagnosed and treated with prescription medication for ADHD, 66.67% [24.11, 94.00] had been treated with ADHD medication before the age of 18. Furthermore, none of these individuals had been treated

with ADHD medication in the past 12 months nor was anyone in the sample currently taking/prescribed ADHD medication.

3.5.3 Anti-anxiety and antidepressant medication use and ADHD screener status. As shown in Table 3.5.4, about 40% of the ADHD positive group and approximately 6% of the ADHD negative group had taken prescription medication to reduce anxiety and panic attacks in the past 12 months. Of those who had taken anti-anxiety medication in the past year, 20.00% [13.91, 27.78] had an ADHD positive screen. There was a significant association between anti-anxiety medication use over the past 12 months and ADHD screening status, $\chi^2(1, N = 1984) = 120.07, p = .000$. Individuals who screened positive had higher odds of past 12 month anti-anxiety medication use than those who screened negative for ADHD (OR = 10.73; 95% CI = 6.41, 17.95).

Antidepressant medication use in the last year according to ADHD screener status is also presented in Table 3.5.4. Approximately 36% of those who screened positive for ADHD reported taking prescription medication to treat depression in the past 12 months, whereas about 6% of those who screened negative for ADHD reported taking such medication in the past 12 months. Of those who had taken antidepressants in the last year, 17.86% [12.10, 25.43] screened positive for ADHD. Antidepressant medication use in the past 12 months differed significantly by ADHD status, $\chi^2(1, N = 1980) = 92.52, p = .000$, with the ADHD positive group having higher odds of antidepressant medication in the past year (OR = 8.87; 95% CI = 5.25, 15.01).

Table 3.5.3

Psychotropic Medication Use by ADHD Screener Status

	ADHD Positive			ADHD Negative			χ^2	df	p-value
	Freq.	(%)	[95% C.I.]	Freq.	(%)	[95% C.I.]			
Anti-anxiety									
Yes	28	40.00	[28.69, 52.41]	112	5.85	[4.86, 7.02]			
No	42	60.00	[47.59, 71.31]	1802	94.15	[92.98, 95.14]			
Total	70	100.00	-	1914	100.00	-			
							120.07	1	< .000
Antidepressants									
Yes	25	36.23	[25.25, 48.75]	115	6.00	[5.02, 7.21]			
No	44	63.77	[51.25, 74.75]	1796	93.08	[92.79, 94.98]			
Total	69	100.00	-	1911	100.00	-			
							92.52	1	< .000

3.5.4 Psychiatric distress according to ADHD screener status. Psychiatric distress differed significantly by ADHD Screener status, ($\chi^2 (1, N = 1986) = 90.10, p = .000$), with those in the ADHD positive group being more likely to have psychiatric distress than those in the ADHD negative group (43.48% [31.77, 55.92] versus 8.71% [7.50, 10.08] respectively). Moreover, those in the ADHD positive group had higher odds of psychiatric distress (OR = 8.06; 95% CI = 4.88, 13.31) compared to the non-ADHD group. Those who screened positive for ADHD represented 15.23% [10.67, 21.19] of all those who screened positive for psychiatric distress.

3.5.5 Antisocial Personality Disorder screener status and ADHD screener status. Of those who screened positive for ADHD, 8.82% [3.64, 18.85] screened positive for ASPD, whereas 0.42% [0.19, 0.86] of those who screened negative for ADHD screened positive for ASPD. Of all those who screened positive for ASPD, 42.86% [18.82, 70.35] also had ADHD. ASPD screening status differed significantly by ADHD screening status, $\chi^2 (1, N = 1951) = 64.98, p = .000$. Those who screened positive for ADHD had higher odds of screening positive for ASPD (OR = 22.68; 95% CI = 7.64, 67.35).

3.5.6 Hazardous alcohol use and ADHD screener status. As seen in Table 3.5.6, a significant association between ADHD screening status and hazardous alcohol use as measured by the AUDIT was found, $\chi^2 (1, N = 1938) = 10.25, p = .001$. The ADHD positive group had higher odds of hazardous alcohol use than the ADHD negative group (OR = 2.41; 95% CI = 1.38, 4.18). Of those in the ADHD positive group, approximately 26% screened positive for hazardous alcohol use on the AUDIT, whereas half that amount (about 13%) of the ADHD negative group screened positive for hazardous alcohol use on the AUDIT.

Table 3.5.6

Hazardous Alcohol Use (AUDIT 8+) and ADHD Screener Status

	ADHD Positive			ADHD Negative			χ^2	df	p-value
	Freq.	(%)	[95% C.I.]	Freq.	(%)	[95% C.I.]			
Hazardous Alcohol Use									
Yes	18	25.71	[16.74, 37.78]	235	12.58	[11.13, 14.19]			
No	52	74.29	[62.22, 83.66]	1633	87.42	[85.81, 88.87]			
							10.25	1	.001

3.5.7 Cannabis, marijuana and hash use and abuse according to ADHD

screener status. As shown in Table 3.5.7 below, roughly 73% of those who screened positive for ADHD and approximately 39% of those who screened negative for ADHD reported using cannabis, marijuana or hash in their lifetime. Lifetime cannabis, marijuana or hash use differed significantly by ADHD status, $\chi^2 (1, N = 1974) = 31.58, p = .000$. Those in the ADHD positive had higher odds of lifetime cannabis, marijuana or hash use compared to the ADHD negative group (OR = 4.15; 95% CI = 2.43, 7.08).

Cannabis use over the past 12 months is also presented in Table 3.5.7.

Approximately 26% of those in the ADHD positive group compared to 13% of the ADHD negative group have used cannabis, marijuana or hash in the past 12 months. Past 12 month cannabis, marijuana or hash use differed significantly by ADHD screener status, $\chi^2 (1, N = 1970) = 9.06, p = .003$, with those in the ADHD positive group having higher odds of past 12 month cannabis, marijuana or hash use compared to the ADHD negative group (OR = 2.29; 95% CI = 1.32, 3.98).

However, the association between cannabis, marijuana or hash use over the past three months and ADHD screener status was not significant, $\chi^2(1, N = 1985) = 3.49, p = .062$, with about 16% of the ADHD positive group and 9% of the ADHD negative group reported using cannabis, marijuana or hash in the past three months. About 15% of those who screened positive for ADHD and roughly 9% of those who screened negative for ADHD screened positive for cannabis abuse. Those who screened positive for ADHD were no more likely than those in those who screened negative for ADHD to screen positive for cannabis abuse, $\chi^2(1, N = 1978) = 2.61, p = .106$. Frequencies, proportions, and 95% confidence intervals for lifetime, past 12 month, and past three month cannabis, marijuana and hash use, and cannabis abuse stratified by ADHD screener status are presented in Table 3.5.7 below.

Table 3.5.7

Cannabis Use and ADHD Screener Status

	ADHD Positive			ADHD Negative			χ^2	df	p-value
	Freq.	Prop. (%)	[95% C.I.]	Freq.	Prop. (%)	[95% C.I.]			
Lifetime									
Yes	51	72.86	[62.22, 83.66]	748	39.29	[37.09, 41.53]			
No	19	27.14	[17.52, 39.30]	1156	60.71	[58.47, 62.91]			
							31.58	1	.000
Past 12 months									
Yes	18	26.09	[16.59, 38.28]	254	13.36	[11.88, 14.99]			
No	51	73.91	[61.72, 83.31]	1647	86.64	[85.01, 88.12]			
							9.06	1	.003
Past 3 months									
Yes	11	15.94	[8.60, 27.16]	177	9.24	[8.00, 10.65]			
No	58	84.06	[72.84, 91.40]	173	90.76	[89.35, 92.00]			
							3.49	1	.062
Cannabis Abuse									
Yes	10	14.71	[7.66, 25.85]	171	8.95	[7.73, 10.34]			
No	58	85.29	[74.15, 92.34]	1739	91.05	[89.66, 92.27]			
							2.61	1	.106

3.5.8 Lifetime cocaine use according to ADHD screener status. Approximately twenty-three percent (23.19%) [14.22, 35.18] of those who screened positive for ADHD

and 6.36% [5.33, 7.57] of those who screened negative for ADHD reported using cocaine in their lifetime. Also, 11.59% [6.98, 18.42] of those who used cocaine in their lifetime screened positive for ADHD. A significant relationship between lifetime cocaine use and ADHD screener status was found, $\chi^2(3, N = 1986) = 29.40, p = .000$, with the ADHD positive group having higher odds of lifetime cocaine use than the ADHD negative group (OR = 4.42; 95% CI = 2.45, 7.96).

3.6 Summary of Key Findings

The prevalence of positive adult ADHD screen was found to be approximately 3.47% using the ASRS v1.1 Screener. Descriptive analyses of demographic variables comparing those who screened positive for ADHD on the ASRS v1.1 Screener to those who did not revealed that men and women are equally likely to screen positive for ADHD and that the rates of a positive adult ADHD screen appear to be highest among those 18-24 years old and lowest among those 65 and over. Those who screen positive for ADHD appear to be more likely to have never been married. The rates of full-time employment were similar between the two groups, however, a larger proportion of those who screened positive for ADHD reported being unemployed or endorsed 'other' (being on social assistance or having an alternative form of income) as their category of employment. Furthermore, no significant differences in education, annual household income or household location were found.

Only about 10% of those who screened positive for ADHD reported being previously diagnosed with ADHD by a healthcare professional, the majority of who were under the age of 44. Most of those who had a previous diagnosis of ADHD reported being treated with prescription medication for the disorder, with treatment starting before

the age of 18 in a substantial proportion of cases. High rates of anti-anxiety and antidepressant medication use was found among those who screened positive for ADHD with correspondingly high rates of psychiatric distress being reported among the ADHD positive group as well. Significantly higher rates of ASPD positive screen were also reported in the ADHD positive group. Significantly elevated rates of hazardous alcohol use, lifetime and past 12 month cannabis use were also found among those who screened positive for ADHD. However, no significant differences in past three month cannabis use or cannabis abuse were found between the groups.

Chapter 4: Sex and ADHD Screener Status

4.1 Sex, ADHD Screener Status and Previous ADHD Diagnosis

No significant differences in previous ADHD diagnosis were found between men and women who screened positive for ADHD. Women who screened positive for ADHD however, were more likely to have a previous diagnosis of ADHD than women who screened negative for ADHD, $\chi^2(1, N = 1041) = 24.25, p = .000$. Among those who screened negative for ADHD, men were more likely to have previously been diagnosed than women, $\chi^2(1, N = 1909) = 17.87, p = .000$. No significant difference in previous ADHD diagnosis was found between men and women who screened positive for ADHD, $\chi^2(1, N = 70) = 0.01, p = .936$.

4.2 Sex and Previous ADHD Medication Use

Of those previously diagnosed, 74.36% [57.57, 86.40] of males and 76.92% [45.98, 83.84] of females were treated with ADHD medication and no significant difference in ADHD medication use between sexes was found, $\chi^2(1, N = 52) = 0.34, p = .853$. Of those previously diagnosed and treated with medication, none had been treated with medication in the past 12 months. Furthermore, of those previously diagnosed and treated with medication, 86.21% [67.43, 95.49] of men were treated with medication before the age of 18 and 54.55% [24.57, 81.87] of women were treated with ADHD medication before the age of 18. A significant difference in ADHD medication use prior to age 18 between men and women was found, $\chi^2(1, N = 4) = 4.59, p = .032$.

4.3. Sex, ADHD Screener Status and Anti-anxiety Medication Use

4.3.1 ADHD screener status and anti-anxiety medication use among men.

Past year anti-anxiety medication use was present among 30.00% [15.41, 49.56] of men

who screened positive for ADHD and 4.51% [3.29, 6.12] of men who screened negative for ADHD. Moreover, men who screened positive for ADHD represented 18.00% [9.05, 31.92] of those who had reported taking anti-anxiety medication in the past year. Past year anti-anxiety medication use among men differed significantly by ADHD screener status, $\chi^2 (1, N = 939) = 37.43, p = .000$. Men who screened positive for ADHD had higher odds of past year anti-anxiety medication use than men who screened negative for ADHD (OR = 9.07; 95% CI = 3.91, 21.05).

4.3.2 ADHD and anti-anxiety medication use among women. Among women in the positive ADHD screener group, 47.37% [31.31, 63.95] reported taking anti-anxiety medication in the past year compared to 52.63% [36.05, 68.69] who had not. Among women in the negative ADHD screener status group, 7.07% [5.60, 8.88] reported taking anti-anxiety medication in the past year compared to 92.93% [91.12, 94.40] that had not. Also, 20.22% [12.73, 30.33] of women who reported taking anti-anxiety medication in the past year screened positive for ADHD. A significant association was found between ADHD screener status and anti-anxiety medication use among women, $\chi^2 (1, N = 1042) = 76.11, p = .000$, with women who screened positive for ADHD having higher odds of past 12 month anti-anxiety medication use compared to women who screened negative for ADHD (OR = 11.83; 95% CI = 5.99, 23.37).

4.3.3 Anti-anxiety medication use among men and women who screened negative for ADHD. Of those who screened negative for ADHD and who reported taking anti-anxiety medication in the past year, 36.61% [27.86, 46.29] were men and 63.39% [53.71, 72.14] were women. A significant relationship was found between sex and past year anti-anxiety medication use among those who screened negative for

ADHD, $\chi^2 (1, N = 1913) = 5.68, p = .017$. Women who screened negative for ADHD had higher odds of past 12 month anti-anxiety medication use compared to men who screened negative for ADHD (OR = 1.61; 95% CI = 1.09, 2.39).

4.3.4 Anti-anxiety medication use among men and women who screened positive for ADHD. Thirty percent [15.41, 49.56] of men and 47.40% [31.31, 63.95] of women who screened positive for ADHD reported taking anti-anxiety medication in the past year. No significant difference in anti-anxiety medication use between men and women who screened positive for ADHD was found, $\chi^2 (1, N = 68) = 2.11, p = .146$.

4.4. Sex, ADHD Screener Status and Antidepressant Medication Use

4.4.1 ADHD screener status and antidepressant medication use among men.

Among men who screened positive for ADHD, 25.81% [12.54, 44.93] reported taking antidepressant medication in the past year and 4.29% [3.11, 5.87] of men who screened negative for ADHD reported taking antidepressant medication in the past year. Also, men who screened positive for ADHD represented 17.02% [8.14, 31.35] of all men who had reported taking antidepressants in the past year. There was a significant relationship between antidepressant medication use among men and ADHD screener status, $\chi^2 (1, N = 940) = 29.22, p = .000$. Men who screened positive for ADHD had higher odds of taking antidepressants in the past 12 months compared to men who screened negative for ADHD (OR = 7.76; 95% CI = 3.26, 18.45).

4.4.2 ADHD Screener status and antidepressant medication use among women. Antidepressant medication use over the past year was prevalent among 46.15% [30.43, 62.62] of women who screened positive for ADHD and 7.55% [6.03, 9.40] of women who screened negative for ADHD. In addition, women who screened positive for

ADHD represented 19.15% [12.04, 28.84] of all women who reported taking antidepressants in the past year. Past year antidepressant medication use among women differed significantly by ADHD screener status, $\chi^2 (1, N = 1041) = 67.98, p = .000$, with women who screened positive for ADHD having higher odds of past 12 month antidepressant use than women who screened negative for ADHD (OR = 10.44, 95% CI = 5.34, 24.44).

4.4.3 Antidepressant use among men and women who screened negative for ADHD. Past year antidepressant medication use was prevalent among 4.29% [3.11, 5.87] of men and among 7.55% [6.03, 9.40] of women who screened negative for ADHD. Of all those who screened negative for ADHD and who reported taking antidepressants in the past year, 33.91% [25.51, 43.40] were men and 66.09% [56.60, 74.49] were women. There was a significant association between sex and past year antidepressant use among those screening negative for ADHD, $\chi^2 (1, N = 1911) = 9.15, p = .002$, with women having higher odds of past 12 month antidepressant use than men (OR = 1.83; 95% CI = 1.23, 2.72).

4.4.4 Antidepressant medication use among men and women who screened positive for ADHD. Past year antidepressant use was prevalent among 25.81% [12.54, 44.93] of men and 46.15% [30.43, 62.62] of women who screened positive for ADHD. No significant difference in past year reported antidepressant use was found between men and women who screened positive for ADHD, $\chi^2 (1, N = 70) = 3.06, p = .080$.

4.5 Sex, ADHD Screener status and Psychiatric Distress

4.5.1 ADHD screener status and psychiatric distress among men. A larger proportion of men who screened positive for ADHD than men who screened negative for

ADHD screened positive for psychiatric distress (35.48% [19.83, 54.62] versus 7.46% [5.88, 9.41] respectively). Moreover, of all men who screened positive for psychiatric distress, 13.92% [7.48, 23.97] also screened positive for ADHD. Psychiatric distress, as measured by the GHQ-12, among men differed significantly by ADHD screener status, $\chi^2(1, N = 942) = 30.64, p = .000$, with men who screened positive for ADHD having higher odds of psychiatric distress than men who screened negative for ADHD (OR = 6.82; 95% CI = 3.14, 14.82).

4.5.2. ADHD screener status and psychiatric distress among women.

Psychiatric distress was present among 48.72% [32.71, 64.97] of women who screened positive for ADHD and in 9.84% [8.10, 11.89] of women who screened negative for ADHD. Of all women who screened positive for psychiatric distress, 16.10% [10.21, 24.26] also screened positive for ADHD. A significant association between psychiatric distress and ADHD screener status was found among women, $\chi^2(1, N = 1045) = 56.65, p = .000$, with women who screened positive for ADHD having higher odds of psychiatric distress than women who screened negative for ADHD (OR = 8.70; 95% CI = 4.49, 16.86).

4.5.3 Psychiatric distress among men and women who screened negative for ADHD. Approximately 9.84% [8.10, 11.89] of women who screened negative for ADHD and 7.46% [5.88, 9.41] of men who screened negative for ADHD had psychiatric distress. Among those who screened positive for psychiatric distress, 40.72% [33.27, 48.60] were men and 59.28% [51.40, 66.73] were women. No significant difference in psychiatric distress between men and women who screened negative for ADHD was found, $\chi^2(1, N = 7) = 3.40, p = .065$.

4.5.4 Psychiatric distress among men and women who screened positive for ADHD. Among those who screened positive for ADHD, 35.48% [19.83, 54.62] of men and 48.72% [32.71, 64.97] of women screened positive for psychiatric distress. Among all those who screened positive for psychiatric distress, 63.33% [43.90, 79.45] of men also screened positive for ADHD and 36.67% [20.55, 56.10] of women also screened positive for ADHD. No significant difference in psychiatric distress between men and women who screened positive for ADHD was found, $\chi^2(1, N = 70) = 1.24, p = .266$.

4.6 Sex, ADHD Screener Status and Antisocial Personality Disorder (ASPD)

Screener Status

4.6.1 ADHD screener status and ASPD screener status among men.

Approximately 20.00% [9.51, 37.31] of men who screened positive for ADHD also screened positive for ASPD, whereas 0.90% [0.42, 1.84] of men who screened negative for ADHD also screened positive for ASPD. Of those who screened positive for ASPD, 42.86% [18.82, 70.35] screened positive for ADHD and 57.14% [29.65, 81.18] did not. A significant association between ASPD screener status and ADHD screener status in men was found, $\chi^2(1, N = 915) = 70.23, p = .000$, with men who screened positive for ADHD having higher odds screen of screening positive for ASPD than men who screened negative for ADHD (OR = 27.41; 95% CI = 8.82, 85.14). No women screened positive for ASPD in this sample.

4.7 Sex, ADHD Screener Status and Hazardous Alcohol Use

4.7.1 ADHD screener status and hazardous alcohol use among men. Similar proportions of men who screened positive for ADHD and men who screened negative for ADHD screened positive for hazardous alcohol use as measured by the AUDIT (25.81%

[12.54, 44.93] versus 18.72% [16.22, 21.50], respectively). ADHD was prevalent among 4.65% [2.18, 9.26] of all men who screened positive for hazardous alcohol use. No significant difference in hazardous alcohol use between men who screened positive for ADHD and men who screened negative for ADHD was found, $\chi^2 (1, N = 907) = 0.98, p = .323$.

4.7.2 ADHD screener status and hazardous alcohol use among women.

Among women, 23.68% [12.02, 40.61] of those who screened positive for ADHD and 7.16% [5.67, 8.99] of those who screened negative for ADHD screened positive for hazardous alcohol use. Of all women who screened positive for hazardous alcohol use, 11.25% [5.59, 20.76] also screened positive for ADHD. Hazardous alcohol use among women differed significantly by ADHD screener status, $\chi^2 (1, N = 1030) = 13.95, p = .000$. Women who screened positive for ADHD had higher odds of hazardous alcohol use than women who screened negative for ADHD (OR = 4.03 95% CI = 1.84, 8.83).

4.7.3 Hazardous alcohol use among men and women who screened negative for ADHD. A larger proportion of men who screened negative for ADHD than women who screened negative for ADHD screened positive for hazardous alcohol use (18.72% [16.22, 21.50] versus 7.16% [5.67, 8.99]). Also, of all those who screened positive for hazardous alcohol use, 67.98% [63.42, 75.50] were men. A significant relationship between sex and hazardous alcohol use among men and women who screened negative for ADHD was found, $\chi^2 (1, N = 1868) = 56.57, p = .000$, with men having higher odds of hazardous alcohol use compared to women (OR = 2.99; 95% CI = 2.22, 4.01).

4.7.4 Hazardous alcohol use among men and women who screened positive for ADHD. A similar proportion of men who screened positive for ADHD (25.81%)

[12.54, 44.93] and women who screened positive for ADHD (23.68%) [12.02, 40.61] also screened positive for hazardous alcohol use. Of all those who screened positive for ADHD and hazardous alcohol use, 47.06% [23.86, 71.47] were men and 52.94% [28.53, 76.14] were women. No significant sex difference in hazardous alcohol use among those who screened positive for ADHD was found, $\chi^2(1, N = 69) = 0.04, p = .839$.

4.8 Sex, ADHD Screener Status and Cannabis Use and Abuse

4.8.1 ADHD screener status and cannabis use and abuse among men. A larger proportion of men who screened positive for ADHD (83.33%) [64.55, 93.69] than men who screened negative for ADHD (43.27%) [40.02, 46.57] reported using cannabis, marijuana, or hash in their lifetime. Of all men who reported using cannabis in their lifetime, 6.00% [4.00, 8.85] also screened positive for ADHD. Lifetime cannabis, marijuana or hash use among men differed significantly by ADHD Screener status, $\chi^2(1, N = 936) = 18.87, p = .000$. Men who screened positive for ADHD were more likely to report having used cannabis, marijuana or hash in their lifetime than men who screened negative for ADHD (OR= 6.56; 95% CI = 2.49, 17.28). However, no significant differences were found between men in both the positive and negative ADHD screener groups for cannabis, marijuana or hash use over the past 12 months, $\chi^2(1, N = 935) = 2.15, p = .143$, or over the past 3 months, $\chi^2(1, N = 940) = 2.21, p = .137$. No significant difference in cannabis abuse between men who screened positive for ADHD and men who screened negative for ADHD was found, $\chi^2(1, N = 934) = 2.63, p = .105$.

4.8.2 ADHD screener status and cannabis use among women. Of all women who screened positive for ADHD, 65.79% [48.58, 79.86] reported using cannabis in their lifetime compared to 35.67% [32.71, 38.74] of women who screened negative for

ADHD. Women who screened positive for ADHD represented 6.56% [4.37, 9.66] of all women who reported using cannabis, marijuana or hash in their lifetime. Lifetime cannabis, marijuana and hash use among women differed significantly by ADHD screener status, $\chi^2 (1, N = 1036) = 14.28, p = .000$, with women who screened positive for ADHD having higher odds of lifetime cannabis, marijuana or hash use than women who screened negative for ADHD (OR = 3.47; 95% CI = 1.75, 6.86). A larger proportion of women who screened positive for ADHD, 26.32% [13.98, 43.39], compared to women who screened negative for ADHD, 11.04% [9.20, 13.19], also reported using cannabis, marijuana or hash over the past 12 months. Women who screened positive for ADHD represented 8.33% [4.29, 15.16] of all women who reported using cannabis, marijuana or hash in the past 12 months. Past 12 month cannabis, marijuana or hash use among women differed significantly by ADHD screener status, $\chi^2 (1, N = 1034) = 8.32, p = .004$, with women who screened positive for ADHD having higher odds of past 12 month cannabis, marijuana or hash compared to women who screened negative for ADHD (OR = 2.88; 95% CI = 1.36, 6.08). Yet no significant difference in past three month cannabis, marijuana or hash use between women who screened positive for ADHD and women who screened negative for ADHD was found, $\chi^2 (1, N = 1045) = 1.53, p = .216$.

4.8.3 Cannabis use among men and women who screened negative for

ADHD. Lifetime cannabis marijuana or hash use was prevalent among 43.27% [40.02, 46.57] of men and 35.67% [32.71, 38.74] of women who screened negative for ADHD. Of those who had used cannabis, marijuana or hash in their lifetime, 52.41% [48.76, 56.03] were men and 47.59 [43.97, 51.24] were women. Past 12 month cannabis, marijuana or hash use was prevalent among 15.93% [13.64, 18.52] of men who screened

negative for ADHD and among 11.04% [9.20, 13.19] of women who screened negative for ADHD. Of those who had used cannabis, marijuana or hash in the past 12 months, 56.69% [50.34, 62.83] were men and 43.31% [37.17, 49.66] were women. Past 3 month cannabis, marijuana or hash use was prevalent among 11.21% [9.32, 13.43] of men who screened negative for ADHD and 7.46% [5.95, 9.30] of women who screened negative for ADHD. Of those who had used cannabis, marijuana or hash in the past 3 months, 57.63% [49.98, 64.94] were men and 42.37% [35.06, 50.02] were women. Men and women who screened negative for ADHD were found to differ on lifetime, past 12 month and past 3 month cannabis, marijuana or hash use, respectively ($\chi^2(1, N = 1904) = 11.49, p = .001$; $\chi^2(1, N = 1900) = 9.77, p = .002$; $\chi^2(1, N = 1916) = 8.03, p = .005$), with men who screened negative for ADHD having higher odds of lifetime cannabis, marijuana or hash use (OR = 1.38; 95% CI = 1.14, 1.65), past 12 month use (OR = 1.53; 95% CI = 1.17, 1.99) and past 3 month use (OR = 1.57; 95% CI = 1.15, 2.14) than women who screened negative for ADHD.

Approximately 10.62% [8.73, 12.86] of men who screened negative for ADHD and 7.46% [5.95, 9.30] of women who screened negative for ADHD met the cut-off for cannabis abuse. Of those who abused cannabis, 56.14% [48.36, 63.64] were men and 43.86% [36.36, 51.64] were women. A significant difference in cannabis abuse between men and women who screened negative for ADHD was found, $\chi^2(1, N = 1910) = 5.85, p = .016$, with men having higher odds cannabis abuse compared to women (OR = 1.48; 95% CI = 1.08, 2.02).

4.8.4 Cannabis use among men and women who screened positive for ADHD.

Among those who screened positive for ADHD, 83.33% [64.55, 93.69] of men and

65.79% [48.58, 79.86] of women reported using cannabis, marijuana or hash in their lifetime and no significant difference in lifetime cannabis, marijuana or hash use between men and women who screened positive for ADHD was found, $\chi^2 (1, N = 68) = 2.65, p = .103$. Regarding cannabis, marijuana or hash use over the past 12 months among those who screened positive for ADHD, 25.81% [12.54, 44.93] of men and 26.32% [13.98, 43.39] of women endorsed using cannabis over the past 12 months and no significant difference in past 12 month cannabis, marijuana or hash use between men and women who screened positive for ADHD was found $\chi^2 (1, N = 69) = 0.002, p = .962$. Moreover, 20.00% [8.40, 39.13] of men and 12.82% [4.82, 28.23] of women reported using cannabis, marijuana or hash over the past 3 months and again, no significant difference in past 3 month cannabis, marijuana or hash use between men and women who screened positive for ADHD was found, $\chi^2 (1, N = 69) = 0.65, p = .419$. Moreover men and women who screened positive for ADHD did not differ in rates of cannabis abuse, $\chi^2 (1, N = 68) = 1.20, p = .273$.

4.9 Sex, ADHD Screener Status and Lifetime Cocaine Use

4.9.1 ADHD and lifetime cocaine use among men. About 22.58% [10.28, 41.54] of men who screened positive for ADHD and 9.46% [7.67, 11.60] of men who screened negative for ADHD reported using cocaine in their lifetime. Men who screened positive for ADHD accounted for 7.53% [3.3.4, 15.40] of all men who had reported using cocaine in their lifetime. Lifetime cocaine use among men differed significantly by ADHD screener status, $\chi^2 (1, N = 940) = 5.79, p = .016$, with men who screened positive for ADHD having higher odds lifetime cocaine use than men who screened negative for ADHD (OR = 2.79; 95% CI = 1.17, 6.67).

4.9.2 ADHD screener status and lifetime cocaine use among women. Among women, 23.68% [12.02, 40.61] of those who screened positive for ADHD and 3.71% [2.66, 5.13] of those who screened negative for ADHD had reported using cocaine in their lifetime. Also, women who screened positive for ADHD accounted for 19.57% [9.87, 34.38] of all women who had reported using cocaine in their lifetime. A significant association between lifetime cocaine use among women and ADHD screener status was found, $\chi^2(1, N = 1036) = 34.43, p = .000$, with women who screened positive for ADHD having higher odds of lifetime cocaine use than women who screened negative for ADHD (OR = 8.06; 95% CI = 3.56, 18.24).

4.9.3 Lifetime cocaine use among men and women who screened negative for ADHD. In those who screened negative for ADHD, 9.46% [7.67, 11.50] of men and 3.71% [2.66, 5.13] of women reported using cocaine in their lifetime. Of all those who reported using cocaine in their lifetime, 69.92% [60.89, 77.68] were men and 30.08% [22.32, 39.11] were women. A significant association between sex and lifetime cocaine use among those who screened positive for ADHD was found, $\chi^2(1, N = 1907) = 26.10, p = .000$, with men having higher odds of lifetime cocaine use than women (OR = 2.71; 95% CI = 1.83, 4.03).

4.9.4 Lifetime cocaine use among men and women who screened positive for ADHD. Regarding lifetime cocaine use, 22.58% [10.28, 41.54] of men and 23.68% [12.02, 40.61] of women who screened positive for ADHD had reported using cocaine in their lifetime. Of those who had reported using cocaine in their lifetime, 43.75% [20.75, 69.45] were men and 56.25% [30.55, 79.25] were women. No significant difference in lifetime cocaine use between men and women who screened positive for ADHD was

found, $\chi^2(1, N = 69) = 0.01, p = .914$. Results from all analyses stratified by sex and ADHD screener status are presented in Table 4.1 below.

Table 4.1

Proportions and 95% Confidence Intervals for Variables Stratified by Sex

Variable	ADHD Men (%)	Positive Women (%)	ADHD Men (%)	Negative Women (%)
Previous ADHD Diagnosis	9.68 [2.53, 26.90]	10.26 [3.34, 25.16]	3.97 [2.83, 5.51]	1.00 [0.51, 1.89]
Anti-anxiety Med Use	30.00 [15.41, 49.56]	47.37 [31.31, 63.95]	4.51 [3.29, 6.12]	7.07 [5.60, 8.88]
Antidepressant Med Use	25.81 [12.54, 44.93]	46.15 [30.43, 62.62]	4.29 [3.11, 5.87]	7.55 [6.03, 9.40]
Psychiatric Distress	35.48 [19.83, 54.62]	48.72 [32.71, 64.97]	7.46 [5.88, 9.41]	9.84 [8.10, 11.89]
ASPD Screener Status	20.00 [9.51, 37.31]	0	0.90 [0.42, 1.84]	0
Hazardous Alcohol Use	25.81 [12.54, 44.93]	23.68 [12.02, 40.61]	18.72 [16.22, 21.50]	7.16 [5.67, 8.99]
Cannabis Lifetime	83.33 [64.55, 93.69]	65.79 [48.58, 79.86]	43.27 [40.02, 46.57]	35.67 [32.71, 38.74]
Cannabis Past 12 Months	25.81 [12.54, 44.93]	26.32 [13.98, 43.39]	15.93 [8.40, 39.13]	11.04 [9.20, 13.19]
Cannabis Past 3 Months	5.56 [2.28, 12.19]	12.82 [4.82, 28.23]	11.21 [9.32, 13.43]	7.46 [5.95, 9.30]
Cannabis Abuse	20.00 [8.40, 39.13]	10.53 [3.43, 25.75]	10.62 [8.73, 12.86]	7.46 [5.95, 5.30]
Cocaine Lifetime	22.58 [10.28, 41.54]	23.68 [12.02, 40.61]	9.46 [7.67, 11.60]	3.71 [2.66, 5.13]

Chapter 5: Hierarchical Logistic Regression Model

A logistic regression analysis was conducted to predict psychiatric distress in 1871 participants using age, sex, ASPD screener status, ADHD screener status, and substance use as predictors. Psychiatric distress was used as a screener for anxiety and depression. Lewis and Wessely (1990) found that the convergent validity between the GHQ-12 (psychiatric distress) and the Hospital Anxiety and Depression Scale was 0.74. In hierarchical logistic regression, variables are entered in steps (blocks) following theoretical or pragmatic reasoning about the sequential order of the variables that may predict a given outcome. Covariates were entered in three blocks as follows: demographic variables (block 1), psychiatric variables (block 2), and substance use variables (block 3).

Block 1 consisted of the demographic variables age and sex. These have been found to be associated to depression and anxiety; studies have shown that anxiety and depressive disorders are most prevalent at middle age and among women (Kroenke, Strine, Spitzer, Williams, Berry & Mokdad, 2009; Bland, 1997; Lehtinen & Joukamaa, 1997; Kessler, McGonagle, Swartz, Blazer & Nelson, 1993). Antisocial personality disorder (ASPD) screener status and ADHD screener status were entered in block 2. ASPD was included in the model due to its frequent co-occurrence with ADHD (Barkley, Fischer, Smallish, & Fletcher, 2004; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Satterfield & Schell, 1997; Weiss, Hechtman, Milroy & Perlman, 1985). As such, antisocial traits needed to be controlled for in order to isolate the effects of ADHD on psychiatric distress. Here, ADHD was the main predictor variable of interest hence its inclusion in the model was imperative. Additionally, according to *DSM-IV-TR*, ADHD is considered a disorder of early developmental origins, while depression is considered a

disorder of later age onset (Ostrander & Herman, 2006). Finally, hazardous alcohol use and cannabis use were entered into the model in block 3. Frequent alcohol and cannabis use has been shown to increase the risk of depression and anxiety and has also been found to be associated with ADHD (Horwood *et al.*, 2012; Boden & Ferguson, 2011; Biederman *et al.*, 1995; Biederman *et al.*, 1997). As such, the nature and magnitude of the contribution of these variables to the overall outcome was of interest.

Table 5.1 displays the results from the hierarchical logistic regression analysis for psychiatric distress. Results from block 1 showed that the addition of the variables age and sex added to the model (Model $\chi^2 = 12.20$, $p = .002$). Age accounted for about 7% of the variance in psychiatric distress (Wald $\chi^2 = 6.51$, $p = .001$). Younger individuals had higher odds of psychiatric distress than older individuals (OR = 0.99; 95% CI = 0.98, 1.00), with a 1.2% decrease in the odds of psychiatric distress with each one year increase in age. Sex was also a significant predictor of psychiatric distress (OR = 0.68; 95% CI = 0.48, 0.92), accounting for approximately 6% of the variance between those who were above threshold for psychiatric distress and those who were not (Wald $\chi^2 = 5.71$, $p = .017$). Furthermore, females were more likely to have psychiatric distress and being female was associated with a 31.60% increase in odds of psychiatric distress.

Psychiatric predictors were added following demographic predictors in block 2. The addition of psychiatric predictors was significant, Block $\chi^2 = 50.49$, $p = .000$. ASPD screener status did not significantly predict psychiatric distress. ADHD screener status however was found to significantly predict psychiatric distress and accounted for about 50% of the variability in psychiatric distress in the sample (Wald $\chi^2 = 49.84$, $p = .000$). Moreover, those who screened positive for ADHD had approximately 7 times the odds of

psychiatric distress (OR= 6.85; 95% CI = 4.00, 11.72). Furthermore, with the addition of the ADHD variable, age was no longer a significant predictor of psychiatric distress.

Two substance abuse variables, hazardous alcohol use and cannabis use were subsequently added in block 3. This block was non-significant; hazardous alcohol use and cannabis abuse did not significantly predict psychiatric distress, Block $\chi^2 = 0.06$, $p = .969$.

A test of the full model against a constant only model was statistically significant, indicating that the predictors as a set reliably distinguished between those who screened positive for psychiatric distress and those who did not (Model $\chi^2 = 62.75$, $p = .000$, $df = 6$). The full model resulted in the correct classification of 89.94% of the data. The Hosmer-Lemeshow goodness-of-fit test was greater than .05; therefore we failed to reject the null hypothesis that there is no difference between observed and model-predicted values, implying that the model's estimates fit the data at an acceptable level and is well-calibrated to predict cases from non-cases ($c = 0.81$). Sex and ADHD were the only two independent predictors of psychiatric distress, as the percentage of variance accounted for by each variable remained relatively unchanged in block 3. Sex was associated with increased odds of psychiatric distress and ADHD was associated with roughly a sevenfold increase in the risk of psychiatric distress.

Table 5.1

Hierarchical Binary Logistic Regression Analysis for Psychiatric Distress (N = 1871)

Variable	β	S.E.	Wald	df	Sig.	Exp. (β)	95% C.I.
Block 0							
(Constant)	-2.19	.08	801.27	1	.000	0.11	
Block 1							
Age	-.01	.01	6.51	1	.011	0.99	0.98, 1.00
Sex	-.38	.16	5.71	1	.017	0.68	0.50, 0.93
(Constant)	-1.49	.23	41.52	1	.000	0.23	
Block 2							
Age	-.01	.01	3.23	1	.072	0.99	0.98, 1.00
Sex	-.40	.16	5.97	1	.015	0.67	0.49, 0.92
ASPD screen	.90	.79	1.30	1	.255	2.47	0.52, 1.64
ADHD screen	1.92	.27	49.84	1	.000	6.88	4.03, 1.75
(Constant)	-1.77	.24	53.35	1	.000	0.17	
Block 3							
Age	-.01	.01	2.99	1	.084	0.99	0.98, 1.00
Sex	-.41	.17	5.94	1	.015	0.67	0.48, 0.92
ASPD screen	.90	.79	1.29	1	.255	2.46	0.52, 1.56
ADHD screen	1.92	.27	49.30	1	.000	6.85	4.00, 11.72
Cannabis Abuse	-.05	.28	0.04	1	.851	0.95	0.54, 1.66
AUDIT 8+	.05	.25	0.46	1	.830	1.05	0.65, 1.71
(Constant)	-1.98	.26	46.32	1	.000	0.17	

Note: Block 1 $\chi^2 (2) = 12.20, p = .002$, -2 Log likelihood = 1190.68; Block 2 $\chi^2 (4) = 62.69, p = .000$, -2 Log likelihood = 1140.19; Block 3 $\chi^2 (6) = 62.75, p = .000$, -2 Log likelihood = 1140.12.

Chapter 6: Discussion

6.1 Findings of Interest

The purpose of this study was to estimate the prevalence and correlates of adult ADHD in Ontario using population-based data, although it is important to point out that the results of this study are based on self-report screening tools and not psychiatric diagnosis. Thus, readers should be mindful that the results of this study may not reflect actual prevalence and correlates of diagnosed ADHD in the community. That said, the overall prevalence of adult ADHD in Ontario, using the ASRS Screener v1.1, was found to be approximately 3.5%. This estimate is in accordance with the WHO World Mental Health epidemiological studies that estimated the prevalence of adult ADHD to be approximately 3.4% in the total sample using the same screening measure (Fayaad *et al.*, 2007; Kessler *et al.*, 2006; Medina-Mora *et al.*, 2005), however it is slightly lower than population-based estimates from the U.S. also using the same measure (Kessler *et al.*, 2006). Approximately equal proportions of men and women screened positive for ADHD, which is in line with earlier evidence of an equalization of the disorder between the sexes in adulthood (Faraone & Biederman, 2005; Kessler *et al.*, 2005; McGough *et al.*, 2005).

Significant age differences were found in this study. Specifically, estimates of ADHD positive screen were highest among those 18-34 years old and declined with increasing age. These results are in keeping with earlier studies that reported a decline of ADHD symptoms with increasing age (Biederman *et al.*, 2000; Faraone, Biederman, Spencer, Wilens, Seidman, Mick & Doyle, 2000).

Only 10% of adults who screened positive for ADHD had previously been diagnosed with ADHD. Another recent epidemiological ADHD study found similarly low rates of previous ADHD diagnoses (de Zwaan, 2012). Furthermore, the vast majority (86.25%) of those who reported previous diagnosis with ADHD was under the age of 44 and reported ADHD diagnoses were most prevalent among those aged 18-34 year old, suggesting a cohort effect resulting from the lack of a consistent conceptualization of the disorder throughout history. In fact, it was not until relatively recently (the early 1980s) that objective diagnostic criteria for ADHD (then termed ADD) appeared in *DSM III*. Thus, those who were school-aged prior to the 1980s were much less likely to have been sent to a medical practitioner and even less likely to have received a diagnosis. Indeed it is much more probable that prior to the 1980s, children who we would now consider as having ADHD were simply labeled ‘hyperactive’, ‘defiant’ or their behaviour was excused on the premise that ‘boys will be boys’ and that was the end of it. The low diagnostic rate could also indicate that the ASRS Screener v1.1 is missing persons with diagnosed ADHD.

The prevalence of adult ADHD screener status was not found to differ between urban and rural areas as it has in previous studies that reported higher estimates of psychopathology in urban compared to rural areas (Peen, Shoevers, Beekman & Dekker, 2010). Variations in the definitions of what constitutes “rural” and “urban” areas between studies have been cited as a possible explanation for discrepant results in the literature (de Zwaan, 2012). Furthermore, divergent findings regarding ADHD and urbanization indicate that this association requires further investigation among adults.

A lower rate of ADHD positive screen was found among those who were married, while a higher rate of ADHD positive screen was found among those who had never been married. Results from the current study are consistent with those from previous population studies (Kessler *et al.*, 2006; de Zwaan, 2012) that have also examined the relationship between ADHD and marital status. These studies found that not only were those with ADHD more likely to have never been married, but they were also more likely to be separated or divorced- a finding that was not corroborated in the current study. Nonetheless, taken together, the tendency for those with ADHD to be less likely to be married and more likely to be previously or never married may reflect the interpersonal difficulties often reported in this population.

However, the effect of age must also be considered as a plausible explanation for our finding that those with ADHD were less likely to be married, as a large proportion of those who screened positive for ADHD were between the ages of 18 to 34 and hence are less likely to be married due to their younger age. This cohort effect may also explain why our study failed to support earlier work that found that those with ADHD were more likely to be previously married. Subsequent analyses should control for the effect of age on marital status in ADHD.

Our study did not find any differences in employment according to ADHD screener status. These results therefore do not support the association between ADHD and occupational challenges in adulthood found in the literature (Barkley, 2002). Previous studies have found that those with ADHD were more likely to experience a number of negative employment outcomes including being more likely to be terminated from a job, frequent job changes, and lower job performance ratings (Manuzza, Klein,

Bessler, Malloy & Hynes, 1997). However, the aforementioned studies were based on clinical samples that may display higher levels of impairment than individuals who screened positively with ADHD in the general population.

Moreover, the current study found that those who screened positive for ADHD did not differ from their non-ADHD counterparts in educational attainment or annual household income. The majority of both groups reported having some post-secondary education or a university degree, a finding that stands in stark contrast to the general consensus that ADHD is associated with low educational performance (Weiss *et al.* 1985; Manuzza *et al.* 1993; Murphy, Barkley & Bush, 2002). In fact, education is generally thought to be the area in which ADHD has the greatest impact (Barkley, 2002). Poor educational outcomes have been corroborated by numerous studies citing that 32-38% of those with ADHD do not complete high school, few people with ADHD enter college, and of those who do, only about 5% graduate (Fischer, Barley, Smallish & Fletcher, 2002).

Again, the discrepancy in findings between our study and the particularly poorer educational outcomes reported in previous studies could be due to important differences in methodology. Mainly, these studies were follow-up reports of clinically-referred samples and these samples, may be biased in that their impairments may be more severe than those found in the general population. Children who are referred to clinics by their parents and adults who are distressed to the point of seeking treatment represent only a subset of ADHD and this subset may in all likelihood, display greater functional impairment. On the other hand, the results of this study were based on a screening tool and not a clinical diagnosis and thus may not be accurate in its assessment of ADHD.

The current study used the GHQ-12 to ascertain current prevalence estimates of psychiatric distress. A large proportion (43.48%) of those who screened positive for ADHD also screened positive for psychiatric distress compared with 8.71% of those who screened negative for ADHD. Furthermore, those who screened positive for ADHD had higher odds of psychiatric distress than those who screened negative for ADHD (OR = 8.06). Men and women who screened positive for ADHD also had significantly higher odds of psychiatric distress compared to their same sex counterparts (OR = 6.82 and 8.70 respectively) with rates being highest among women who screened positive for ADHD.

These results are consistent with previous work with referred and non-referred groups of children, adolescents, and adults demonstrating additional mental health issues in ADHD, particularly where mood and anxiety disorders are concerned (Biederman *et al.*, 1993; Kooij, Buitelaar, van den Oord, Furer, Rijnders & Hodiament, 2004).

Furthermore, epidemiological studies have reported prevalence rates of psychiatric distress in the general population to be around 15%, whereas the rates of psychiatric distress are above 30% in ADHD (Shekim, Asarnow, Hess, Zaucha & Wheeler, 1990; Biederman *et al.*, 1991; Jensen, Shervette, Xenakis & Richters, 1993; Sobanski, 2006). Additionally, the equalization of depression and anxiety among men and women who screened positive for ADHD is indicative of increased psychological vulnerability associated with the disorder and also demonstrates similarities rather than differences in the expression of adult ADHD between sexes.

Results from our regression analysis demonstrate that ADHD contributes to a significant proportion of the variance in psychiatric distress. Some investigators have hypothesized that depression in ADHD is analogous to an adjustment disorder reflecting

demoralization resulting from chronic social and academic failure (Sobanski, 2006). Based on this theory, we speculate that ADHD and depression may be a function of the demoralizing effects of the disorder, possibly resulting from an absence of treatment in this population. These results are also consistent with the hypothesis that ADHD poses a biological predisposition toward adverse environmental and social factors as well as further mental health issues, although it is important to point out that the results of the regression were based on a cross-sectional database; thus directionality of prediction among variables cannot be determined.

The rates of anti-anxiety and antidepressant medication use were also notably elevated in those who screened positive for ADHD and did not differ significantly between sexes. A higher rate of anti-anxiety and antidepressant medication use in general is not surprising given the correlation between ADHD and other mental health issues. However, differential diagnosis is a prominent issue due to shared symptoms between ADHD and many other psychiatric disorders (Milberger, Biederman, Faraone, Murphy & Tsuang, 1995). For example, anxiety and depression often consist of symptoms of inattention and restlessness which are hallmarks of ADHD. However, this study cannot disaggregate ADHD from other psychiatric disorders.

The prevalence of an ASPD positive screen was significantly higher among men who screened positive for ADHD (8.82%) than among men who screened negative for ADHD (0.42%), however the sample size was notably small ($n = 6$). This finding is supported by the results of previous longitudinal studies with children and retrospective studies with adults that consistently report high rates of ASPD in adult ADHD and among men in general (Weiss *et al.*, 1985, Manuzza *et al.*, 1993; Barkley, Fischer, Smallish, &

Fletcher, 2004; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Satterfield & Schell, 1997; Weiss, Hechtman, Milroy & Perlman, 1985). The literature indicates that hyperactivity and impulsivity in ADHD can lead to early conduct problems resulting in ASPD and does predict greater delinquency and criminality in males but not in females (Babinski, 1999). However, other authors underscore the oversimplification of the widespread claims of poor outcomes in ADHD and of shared etiology for ADHD and ASPD and hypothesize that the association between ADHD and ASPD is a product of the overlap between ADHD and CD (Lilienfeld & Waldman, 1990). Hence, the association may simply reflect the persistence of antisocial behaviour (CD) from childhood into adulthood and not ADHD as such.

The current study also found significantly higher rates of harmful alcohol use among those with ADHD positive screen, which is supported by previous studies that reported a markedly higher incidence of alcohol abuse and dependence in ADHD compared to controls (Downey *et al.*, 1997; Biederman, Wilens, Mick, Faraone & Spencer, 1998; Ohlmeier *et al.*, 2008). Yet higher rates of alcohol consumption have also been found to be associated with younger age in general (Johnson, 1998; Caetano & Kaskutas, 1995; Fillmore, 1991) and those in the ADHD positive group were primarily of younger age than those in the ADHD negative group. Therefore, the higher rates of hazardous alcohol use found in this study could also be a consequence of age.

Various researchers have consistently reported an association between ADHD and substance use disorder (SUD), with up to 50% of adults with ADHD suffering from a concurrent SUD (Biederman *et al.*, 1995, 1998; Wilens, Biederman, Mick, Faraone &

Spencer, 1997). ADHD is also assumed to be associated with a twofold risk of SUD compared to the general population.

Our results showed that individuals who screened positive for ADHD had significantly elevated rates of lifetime and past 12 month cannabis use; however their use over the past 3 months did not differ from those who screened negative for ADHD. These results may suggest a propensity toward greater risk-taking behaviour in this population, as individuals with ADHD have shown a tendency to experiment more liberally with substance use than those without ADHD (Carroll & Rounsaville, 1993; Levin & Kleber, 1995; Wilens *et al.*, 1997; Biederman *et al.*, 1998). However our findings do not indicate elevated rates of recent cannabis use, or chronic cannabis use among those who screened positively for ADHD.

Sex-specific comparisons revealed no significant differences in hazardous alcohol use, cannabis use, or abuse between men who screened positive for ADHD and men who screened negative for ADHD. Women who screened positive for ADHD however had significantly higher rates of hazardous alcohol use, as well as lifetime and past 12 month cannabis use than women who screened negative for ADHD. Although cannabis use, abuse, and lifetime cocaine use were more likely among men in the general population, they did not differ significantly between men and women who screened positive for ADHD. These results stand in contrast to earlier findings that men with ADHD have a higher frequency of substance use than women with ADHD (Millstein, Wilens, Biederman & Spencer, 1997; Biederman *et al.* 2004; McGough *et al.* 2005), yet they corroborate earlier work by Biederman and colleagues (1994) indicating that ADHD

females may share a similarly increased risk for substance use with their male counterparts.

Although our findings showed higher odds for substance use in those who screened positive for ADHD, the vast majority of those who screened positive for ADHD did not engage in hazardous alcohol use (74.29%), had not used cannabis in the last year (73.91%) or in the past 3 months (84.06%), and most were likewise not cannabis abusers (85.29%). Many studies and reviews present a behavioural determinism perspective whereby ADHD is presented “firmly as a biological disorder” that “has such a profound effect on brain function that every aspect of the life of an affected individual may be permanently compromised” (Comings *et al.*, 2005). Yet this is not the whole story. This perspective may be attributable to the synthesis of the findings derived principally from highly impaired clinical samples. It would be important to examine what proportion of those who screen positively for ADHD in the CAMH Monitor would also receive a clinical diagnosis of ADHD, as our results show that those who screened positively for ADHD show a propensity for psychiatric distress and greater odds of substance use yet seem to be functioning reasonably well in areas such as education, employment, and annual household income.

6.2 Limitations

These results however must be considered in light of the limitations inherent in this study. Because this was a preliminary analysis of only one year of data, sample sizes were too small for more sophisticated analyses. Importantly, age was not controlled for because our sample did not meet the guidelines for multinomial logistic regression that indicate a minimum of 10 cases per independent variable (Schwab, 2002). Adjusted ORs

were not calculated in the current study and thus age may have undue influence on the results of bivariate relationships. Therefore findings must be interpreted with caution. For example, individuals with positive screen for ADHD had a lower likelihood of being married and higher rates of hazardous alcohol use; yet these findings may be an artifact resulting from the uncontrolled effect of age. Future studies using the adult ADHD screener of the CAMH Monitor dataset should examine multiple years of data and examine odds ratios that have been adjusted for age and sex in comparison to the crude odds ratios presented here.

The key limitation, as previously mentioned, is that this study examined prevalence and correlates of a number of screening tools. Subsequent research should sample and conduct a psychiatric assessment of a cohort of participants who screened positive and negative on the ASRS-v1.1 Screener in order to assess the sensitivity, specificity and other important measures of degree of overlap between the screening tools and actual psychiatric diagnoses. A related key limitation in this study was the overlap between the items on the ASRS-v1.1 Screener and the GHQ-12 screening instruments. The similarities between items in these screening measures make it difficult to ascertain whether the ASRS is actually assessing ADHD symptomatology and not some ambiguous conglomerate of psychological symptoms applicable to a wide range of psychiatric disorders. Therefore, one cannot label those who screened positive for ADHD with ADHDs and this fact must be taken into consideration when interpreting our findings. Thus, the use of screening measures such as the ASRS-v1.1 Screener, the GHQ-12, the AUDIT, the ASSIST and the MINI-APD as opposed to full-length assessments and clinical diagnoses is an important methodological limitation of this study.

Another important limitation of this work involves the generalizability of the study results. The CAMH Monitor is only representative of non-institutionalized Ontarians age 18 and older ($n = 9,118,084$ from 2001 Ontario Census) (Ialomiteanu & Adlaf, 2007). The data collection method used in this study has several limitations, chiefly that 1) the survey is based on households with telephones 2) the survey is based on self-report and 3) the presence of interview barriers.

Excluded by design are Ontario households that are phoneless, which represent 1% of Ontario residents (Statistics Canada, 2011). Also excluded are those too ill or aged to be interviewed and those unable to communicate on the telephone or in English. In addition, household surveys are limited to those residing in conventional households and are not intended as a sample of all possible adults. As such, those in prisons, hospitals, military establishments, and transient populations such as the homeless are excluded. Importantly, these excluded groups often contain an especially large number of drug and alcohol users (Rossi, 1989). However, the coverage error depends firstly upon the difference in drug use and mental health status between those surveyed and those not surveyed, and secondly, the size of the group missed (Ialomiteanu & Adlaf, 2011). Impairments may be substantially higher in the excluded group than are those in the sampled group, yet if the size of the excluded group is small relative to the total population, the bias is usually minimal (Kandel, 1991; Trinkoff, Ritter, & Anthony, 1990). One common deficit of telephone surveys is that they often over-represent those with higher education and under-represent those with lower education (Trewin & Lee, 1988).

Furthermore, survey estimates are susceptible to self-report errors that are influenced by the conditions under which the survey is conducted. One limitation of the CAMH Monitor in this regard is its reliance on self-reports. Reviews of self-report methods for alcohol and drug use suggest that although surveys tend to underestimate true usage, they are still deemed the best available method to estimate such behaviours (Harrison, Haaga, & Richards, 1993; Turner, Lessler, & Gfroerer, 1992). One of the most important sources of bias to consider is social desirability bias, which is the tendency of respondents to answer questions in a manner that will be viewed favorably by others (Krosnick, 1999).

Given that this was a preliminary assessment of adult ADHD screening status using the first year of data collection in a multi-year study, the sample size was relatively small. Specifically, cell counts were too low for more refined disaggregated effects and analyses may have had insufficient power to detect statistical significance. Additionally, missing data on the annual household income variable (> 5%) was also a limitation in this study as imputation methods were not used to correct for this. Furthermore, the design effect created by the 2 stage probability sampling was not accounted for in this study. Therefore for the aforementioned reasons it is most important that caution be taken when generalizing the results of this study to the entire population of Ontario.

6.3 Directions for Future Research

Directions for future research would include using combined data from multiple waves of the CAMH Monitor in order to further investigate disaggregated results. Many analyses were not possible in the current study due to the low sample size; hence

amassing several years of data would not only improve sample size and statistical power, but would also allow for more extensive analysis of the data.

Future research could stratify results by age group in view of providing further insight into the similarities and differences in descriptive information, mental health correlates and substance use across all stages of adulthood. Also, preferential drug use in ADHD could be examined using a Canadian sample, as this was not possible due to low cell counts for non-prescribed ADHD medication use, prescription analgesic use, opiate use, and past year cocaine use. Furthermore, since such a small portion of the sample of those who screened positive for ADHD had been previously diagnosed, potential studies examining predictive factors for ADHD diagnoses are needed. Prospective follow-up studies of young adults who screened positive for ADHD may also lend to the ASRS-v1.1 as a valid predictor of ADHD, as well as ADHD as a risk factor for later outcomes.

ADHD is characterized by three symptom domains: inattention, hyperactivity and impulsivity. The current theory of adult ADHD describes an attenuation of hyperactivity and a persistence of inattention in adulthood. As such, the ASRS-v1.1 Screener primarily emphasizes symptoms of inattention, with four out of the six ASRS-v1.1 Screener items representing inattention and the remaining two items pertaining to hyperactivity. Importantly the Screener does not contain a single item related to impulsivity. Therefore, future studies using items representing all symptom domains are recommended. Also, examining symptom domains in relation to demographic, psychiatric and substance use variables may also reveal important differences between these groups. Since ADHD and additional mental health issues display unique profiles and trajectories, comparing outcomes between profiles would be an important priority for future research. Also,

further studies of not only risk but resiliency factors in ADHD are also warranted. Finally, as previously mentioned, it would be very important to conduct clinical assessments of a cohort of respondents who screened positively and negatively on the ASRS-v1.1 Screener, in order to determine the performance of the screener against clinical diagnoses.

6.4 Summary and Conclusions

ADHD positive screen was significantly associated with greater rates of psychiatric distress and antidepressant and anti-anxiety medication use. ADHD positive screen was also found to be associated with higher rates of lifetime and past 12 month cannabis use, however the greater majority of those who screened positively with ADHD had not used cannabis in the past 3 months. Moreover, despite higher odds of cannabis use disorders in ADHD, the vast majority did not screen positive for cannabis abuse. Inconsistent with the results of previous studies, individuals who screened positively for ADHD did not differ from those who screened negatively for ADHD in educational attainment, employment status or annual household income. These findings are intriguing and require further investigation.

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Appendix A Adult ADHD Self-Report Scale version 1.1

Questions	Never 0	Rarely 1	Sometimes 2	Often 3	Very Often 4
1. In the past 6 months, have you had trouble finishing a project?					
2. In the past 6 months, have you had difficulty getting things in order?					
3. In the past 6 months, have you had difficulty remembering appointments?					
4. In the past 6 months, have you delayed projects that required a lot of thought?					
5. In the past 6 months, how often have you fidgeted when sitting for a long time?					
6. In the past 6 months, have you felt overly active and compelled to do things?					

Appendix B General Health Questionnaire 12 (GHQ-12)

1. Over the past few weeks, have you been able to concentrate on whatever you're doing?	Better than usual	No more than usual	Rather more than usual	Much more than usual
2. Over the past few weeks, have you felt that you are playing a useful part in things?	Not at all	No more than usual	Rather more than usual	Much more than usual
3. Over the past few weeks, have you felt capable of making decisions about things?	More so than usual	Same as usual	Less than usual	Much less
4. Over the past few weeks, have you been able to enjoy your normal day-to-day activities?	More so than usual	Same as usual	Less than usual	Much less
5. Over the past few weeks, have you been able to face up to your problems?	Not at all	No more than usual	Rather more than usual	Much more than usual
6. Over the past few weeks, all things considered, have you been feeling reasonably happy?	Not at all	No more than usual	Rather more than usual	Much more than usual
7. Over the past few weeks, have you lost much sleep because of worry?	More so than usual	Same as usual	Less than usual	Much less
8. Over the past few weeks, have you felt constantly under strain?	More so than usual	Same as usual	Less than usual	Much less
9. Over the past few weeks, have you felt you could not overcome your difficulties?	Not at all	No more than usual	Rather more than usual	Much more than usual
10. Over the past few weeks, have you been feeling unhappy and depressed?	Not at all	No more than usual	Rather more than usual	Much more than usual
11. Over the past few weeks, have you been losing confidence in yourself?	Not at all	No more than usual	Rather more than usual	Much more than usual
12. Over the past few weeks, have you been thinking of yourself as a worthless person?	More so than usual	Same as usual	Less than usual	Much less

**Appendix C Antisocial Personality Disorder Module from the MINI
Neuropsychiatric Interview Version 5.0.0**

<i>Before you were 15 years old, did you:</i>		
1. Repeatedly skip school or run away from home overnight?	Yes	No
2. Repeatedly lie, cheat, "con" others, or steal?	Yes	No
3. Start fights or bully, threaten, or intimidate others?	Yes	No
4. Deliberately destroy things or start fires?	Yes	No
5. Deliberately hurt animals or people?	Yes	No
<i>Since you were 15 years old, have you:</i>		
6. Repeatedly behaved in a way that others would consider irresponsible, like failing to pay for things you owed, deliberately being impulsive or deliberately not working to support yourself?	Yes	No
7. Done things that are illegal even if you didn't get caught (for example, destroying property, shoplifting, stealing, selling drugs, or committing a felony)?	Yes	No
8. Been in physical fights repeatedly (including physical fights with your spouse or children)?	Yes	No
9. Often lied or "conned" other people to get money or pleasure, or lied just for fun?	Yes	No
10. Exposed others to danger without caring?	Yes	No
11. Felt no guilt after hurting, mistreating, lying to, or stealing from others, or after damaging property?	Yes	No

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Appendix D Alcohol Use Disorders Identification Test (AUDIT)

1. How often do you have a drink containing alcohol?	Never	Monthly or less	2 – 4 times a month	2-3 times a week	4 or more times a week
2. How many drinks do you have on a typical day when you are drinking?	1 or 2	3 or 4	5 or 6	7 to 9	10 or more
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 unable to stop drinking once you had started?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
5. How often during the past year have you failed to do what was normally expected of you because of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
6. How often during the past year have you needed a drink in the morning after a night of drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
7. How often during the past year have you had a feeling of remorse or guilt after drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
8. How often during the past year have you been unable to remember what has happened the night before because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily
9. Have you or someone else been injured because of your drinking?	No	-	Yes, but not in the last year	-	Yes, during the last year
10. Has a relative, friend, doctor or 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 E Alcohol, Smoking and Substance Involvement Screening Test (ASSIST)
for Cannabis Only**

1. How often have you used cannabis, marijuana or hash during the PAST THREE months?	Never 0	Once or Twice 2	Monthly 3	Weekly 4	Daily or Almost Daily 6
2. During the PAST 3 MONTHS, how often have you had a strong desire or urge to use cannabis, marijuana or hash?	Never 0	Once or Twice 3	Monthly 4	Weekly 5	Daily or Almost Daily 6
3. During the PAST 3 MONTHS, how often has your use of cannabis, marijuana or hash led to health, social, legal or financial problems?	Never 0	Once or Twice 4	Monthly 5	Weekly 6	Daily or Almost Daily 7
4. During the PAST 3 MONTHS, how often have you failed to do what was normally expected of you because of your use of cannabis, marijuana or hash?	Never 0	Once or Twice 5	Monthly 6	Weekly 7	Daily or Almost Daily 8
5. Has a friend, relative, a doctor or anyone else ever expressed concern about your use of cannabis, marijuana or hash?	Never 0	Yes, not past 3 months 3	Yes, past 3 months 6	-	-
6. Have you ever tried and failed to control, cut down or stop using cannabis, marijuana or hash?	Never 0	Yes, not past 3 months 3	Yes, past 3 months 6	-	-

Appendix F Diagnostic Statistics

Variable	<i>N</i>	Range	Min.	Max.	<i>M</i>	<i>SD</i>	Skewness	Kurtosis
Age	1926	3	1	4	2.563	0.895	0.059	-0.783
Sex	1986	1	0	1	0.474	0.499	0.103	-1.990
Marital Status	1968	2	1	3	1.518	0.813	0.109	-0.600
Education	1970	3	1	4	2.954	0.971	-.550	-0.731
Employment Status	1977	7	1	8	3.250	3.203	0.784	-1.347
Annual Household Income	1986	4	1	5	3.567	1.203	-0.685	-0.423
Household Location	1986	1	0	1	0.849	.359	-1.946	1.789
ADHD Screening Status	1986	1	0	1	0.035	.183	5.08	23.78
Previous ADHD Diagnosis	1979	1	0	1	0.030	.161	5.878	32.589
Antidepressant Use	1980	1	0	1	0.071	0.257	3.350	9.299
Psychiatric Distress	1986	1	0	1	0.099	0.299	2.681	5.195
ASPD Screening Status	1952	1	0	1	0.007	0.084	11.740	135.973
Hazardous Alcohol Use	1937	1	0	1	0.130	0.337	2.197	2.828

Cannabis Lifetime	1974	1	0	1	0.405	0.491	0.389	-1.351
Cannabis Past 12 Months	1970	1	0	1	0.138	0.345	2.098	2.405
Cannabis Past 3 Months	1985	1	0	1	0.094	0.293	2.770	5.681
Cocaine Lifetime	1976	1	0	1	0.070	0.255	3.370	9.365

Curriculum Vitae

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September 2011-August 2012

- Ontario Graduate Scholarship (OGS)
- Western Graduate Research Scholarship (WGRS)

September 2010- August 2012

- Schulich Graduate Scholarship (SGS)

Education

September 2010- December 2012

London, ON

Western University, Schulich School of Medicine and Dentistry**Master of Science in Epidemiology (in progress)**

September 2006- April 2010

Hamilton, ON

McMaster University**Honours Bachelor of Arts in Psychology, Neuroscience and Behaviour****Work Experience**

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UWO Department of Epidemiology and Biostatistics**Teaching Assistant- Survey Research Methods (Epi9547B)**

September 2010- December 2012

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UWO Department of Family Medicine, Population and Community Health Unit**Research Assistant****Poster Presentations**

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