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Effects of Puberty Onset on Attention Deficit/ Hyperactivity Disorder (ADHD) Symptoms in Female University Students

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EFFECTS OF PUBERTY ONSET ON ATTENTION DEFICIT/HYPERACTIVITY DISORDER (ADHD) SYMPTOMS IN FEMALE UNIVERSITY STUDENTS

By

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ABSTRACT

Despite the growing evidence that circulating sex hormones during puberty may help explain the subtle sex differences that exist in the symptom profile, neuropathology and clinical sequelae of ADHD, there is limited research in this area. The current study investigated how the timing of female pubertal maturation influences the extent of ADHD symptoms in a non-clinical female undergraduate sample (*N*=253). Participants completed a set of self-report rating scales examining pubertal onset, and ADHD symptoms and related deficits. Using logistic regression models, difficulties in attention, emotion regulation, psychosocial functioning and more risky behaviour were shown to significantly help classify those who reported having an earlier pubertal onset relative to their peers. That is, early puberty was associated with increased symptom endorsement on a variety of ADHD-related variables. Findings highlight the potential role of sex hormones during puberty in explaining the differences in gender prevalence rates of ADHD and symptom profiles.

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LIST OF ABBREVIATIONS

ADHD	Attention Deficit/Hyperactivity Disorder
BAARS-IV	Barkley Adult ADHD Rating Scale
BDEFS	Barkley Deficits in Executive Functioning Scale
BFIS	Barkley Functional Impairment Scale
DERS	Difficulties in Emotion Regulation Scale
DSM	Diagnostic and Statistical Manual of Mental Disorders
EF	Executive Functioning
EM	Expectation-Maximization
FSH	Follicle stimulating hormone
GnRH	Hypothalamic gonadotropin releasing hormone
HPG	Hypothalamic-pituitary-gonadal axis
LH	Luteinizing hormone
MANCOVA	Multivariate Analysis of Covariance
MAR	Missing at Random
MCAR	Missing Completely at Random
PDS-RV	Pubertal Developmental Scale – Retrospective Version
PFC	Pre-Frontal Cortex
RTBQ	Risk Taking Behaviour Questionnaire

CHAPTER I

Introduction

Attention deficit/hyperactivity disorder (ADHD) is a neurobehavioural developmental disorder typically diagnosed in childhood (APA, 2013; Miller, 2012). Although prevalence rates vary widely due to methodological issues (e.g., sampling techniques, diagnostic criteria), ADHD continues to be the most common childhood psychiatric diagnosis affecting approximately 5.5% of children worldwide (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Over the last couple of decades, there has been increasing awareness that the symptoms observed in childhood often continue into adulthood, with many children with ADHD not "outgrowing" the disorder (Barkley, Fischer, Edelbrock, & Smallish, 1990; Kalbag & Levin, 2005; Halperin, Trampush, Miller, Marks, & Newcorn, 2008), although there is debate over the extent of symptom preservation (Hill & Schoener, 1996; Mannuzza et al., 1991). Nevertheless, data on prevalence rates in adolescence and adulthood are limited and likely echo both under-diagnosis and lack of clinical attention given to this age range (Kalbag & Levin, 2005; Miller, 2012). According to Fayyad and colleagues (2007), ADHD affects approximately 3-4% of the adult population worldwide. Such a remarkable statistic supports the fact that adult ADHD requires more attention and needs to be studied more thoroughly.

To receive an adult diagnosis of ADHD, several symptoms present before age 12 must persist into adulthood (APA, 2013a). However, late adolescents and young adults with ADHD and their parents have limited recall of childhood ADHD symptoms, questioning the validity and use of such retrospective data when making a diagnosis (Barkley, Knouse, & Murphy, 2011; Miller, Newcorn, & Halperin, 2010). Relatedly,

there is a tendency for adults to not connect ADHD symptoms and associated impairments to adult ADHD if they were not diagnosed as children (Kalbag & Levin, 2005), and rather to attribute them to personality or character traits (Barkley & Brown, 2008). Finally, while it has been suggested that the symptoms required by the Diagnostic and Statistical Manual of Mental Disorders (DSM) may not be developmentally appropriate, the current DSM has not made changes to the diagnostic criteria to appropriately address the changes in developmental norms across the life span (APA, 2013b; Barkley, Murphy, & Fischer, 2008; Miller, 2010). Gaps in our current understanding of adult ADHD, and poor consensus regarding what might constitute appropriate diagnostic criteria highlight the need for research investigating the persistence of this disorder into adulthood, with the goal of developing new criteria that incorporates symptoms more relevant to the challenges encountered by young adults.

Across clinical settings ADHD is reported to be more common in males than in females, with male childhood rates approximately two times larger than females (APA, 2013a). Notably, studies examining ADHD symptoms, including those based on university samples, overwhelmingly sample males (Biederman et al., 1993, as cited in Rodriguez & Span, 2008). Consequently, the manifestation of ADHD in females has been neglected in the literature, as have sex differences in ADHD (Arnold, 1996). It has been speculated that this paucity in the literature, in addition to sex differences in the outward display of ADHD symptoms, has led to the sex discrepancy in clinical referrals and sampling bias (Mahone, 2010; Sciutto, Nolfi, & Bluhm, 2004). For instance, even when the expression of ADHD symptoms is equal, teachers are still more likely to refer boys than girls for treatment (Sciutto et al., 2004). Whereas childhood prevalence rates

continuously suggest greater ADHD rates for males than females, there is some indication that the male bias in ADHD is eliminated by adulthood (DuPaul et al., 2001; Nussbaum, 2012). Importantly, females with ADHD are equally susceptible to life course impairments as are males (Lee, Lahey, Owens, & Hinshaw, 2008), and may even be more prone to particular disorders (e.g., eating disorders; Biederman et al., 2010). Thus, this neglect is a public health concern affecting hundreds of thousands of affected females (Arnold, 1996).

CHAPTER II

Review of Literature

Organization of Review

This chapter begins with a review of the relevant literature on attention deficit hyperactivity disorder (ADHD), followed by a discussion on human pubertal development. This is then followed by the presentation of literature supporting a link between female pubertal development and sex-specific ADHD symptom manifestation. Finally, an outline of the purpose of the proposed study and the research questions is given.

Attention Deficit Hyperactivity Disorder

The most current edition of the DSM (DSM-5) defines ADHD as a "persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development" (APA, 2013a, p. 59). The DSM-5, like the DSM-IV-TR, distinguishes four subtypes of ADHD: Combined type, Predominantly Inattentive, Predominantly Hyperactive-Impulsive, and Other Specified ADHD. The Combined type is the most common, accounting for 50-75% of diagnoses, and requires at least a total of six symptoms from both the hyperactive-impulsive and inattentive domains for diagnosis (APA, 2013a; Miller, 2012). Notably, new to the DSM-5, the symptom threshold for those aged 17 and older has been lowered from six to five symptoms (APA, 2013a). Some symptoms indicative of an attention deficit are inability to 'sustain attention', 'easily distracted' and 'often forgetful' (APA, 2013a). Examples of symptoms falling under the hyperactivity-impulsivity domain include, but are not limited to, 'often fidgets', 'runs about or climbs excessively in situations where inappropriate', 'talks excessively',

and has ‘difficulty awaiting turn’ (APA, 2013a). Predominantly Inattentive and Predominantly Hyperactive-Impulsive subtypes account for 20-30% and less than 15% of diagnoses, respectively (Miller, 2012). The diagnosis of either subtype requires that the six symptoms (or five for older adolescents and adults) cluster within either the inattention or hyperactive-impulsive domains, although there may also be subclinical features present that would fall under the other category (APA, 2013a). Finally, the last subtype, Other Specified ADHD, is the subtype chosen when in the presence of significant impairment and distress there are prominent symptoms present yet they do not sufficiently meet all the criteria (APA, 2013a). Evident from the examples given above and in line with the criticism of the current DSM diagnostic criteria described above, this continues to be a very child-centric definition of ADHD. The DSM-5 committee attempted to remedy this by providing clinicians with examples of the types of behaviours that might manifest in older adolescents and adults with ADHD (APA, 2013b). The manifestation of symptoms in adults is different, and characterized more by symptoms such as ‘racing thoughts’, ‘restlessness’, ‘making careless mistakes when work on a boring or difficult project’, ‘problems remembering appointments’, ‘feeling overly energetic and compelled to do things’, and ‘disrupting others when they are occupied’ (Barkley, 2011; Kessler, 2005). It is important to note that whereas the use of these subtypes is common practice in North America, there is some indication that these subtypes are developmentally unstable (Lahey, Pelham, Loney, Lee, & Willcutt, 2005; Todd et al., 2008). Moreover, population-based behaviour genetic studies examining preferential familial clustering in the study of complex genetic traits, such as ADHD, suggest that ADHD is a single dimensional phenotype that varies in severity across

humans, and that when two dimensions are found, they are highly correlated (Acosta, Arcos-Burgos, & Muenke, 2004). Thus the utility of subtyping ADHD may be limited.

ADHD has been predominantly conceptualized as a disorder of executive functioning, and thus commonly linked to a dysfunction of neural circuits in the prefrontal cortex (PFC) and the catecholamine neurotransmitter systems (Barkley, 1997; Halperin & Schulz, 2006; Nussbaum, 2012). In line with this conceptualization, children and adults with ADHD have shown impairment on a number of neuropsychological measures of executive function, including inhibitory control, self-regulation, planning, working memory, and shifting sets (Halperin & Schulz, 2006; Nussbaum, 2012). To explain the executive functioning deficit present in ADHD, Barkley (2011d) conceptualizes the disorder as age-inappropriate behaviour in two domains of neuropsychological functioning that parallel the DSM subtypes categorization: Hyperactivity-Impulsivity (Inhibition) and Inattention (Meta-Cognition). Symptoms within the domain of hyperactivity-impulsivity reflect poor inhibition and are characterized by impairment in verbal and motor inhibition, impulsive decision making, inability to delay gratification, greater disregard for future consequences, excessive task-irrelevant movement and emotional impulsiveness (Barkley, 1997; Barkley, 2011d; Brown, 2006). Conversely, the ADHD deficit in attention is exemplified by impairment in resistance to responding to distraction, low persistence toward goals or tasks, poor working memory, difficulty re-engaging in a task following disruptions and poor emotional self-regulation (Barkley, 1997; Barkley, 2011d; Brown, 2006). It is important to note that this conceptualization of ADHD has not been unequivocally supported. Halperin and Schulz (2006) proposed that ADHD is not due to dysfunction of the PFC,

but rather due to subcortical neural dysfunction present during early development. As such, symptom diminution is due to the degree which the developing PFC is able to compensate for early neural deficit via its descending regulatory influence on more caudal neural structures. Moreover, the delay aversion model (Sonuga-Barke et al., 1996) and the cognitive-energetic model of ADHD (Sergeant, 2005) provide alternative explanations for the symptoms and behaviours present in ADHD, with the former implicating dysfunction to the neurobiological system linking present behaviour and future reward/punishments, and the latter attributing the symptoms to dysregulation of arousal centers.

The neuroimaging literature has implicated three primary neural circuits in ADHD: fronto-striatal, fronto-cerebellar and fronto-limbic (Barkley, 2011d; Nigg & Casey, 2005). Fronto-striatal functioning is thought to be important in the detection of unpredicted reward or novel functioning and is associated with difficulties in response suppression, ability to maintain concentration, working memory, planning, and organization (Barkley, 2011d; Nigg & Casey, 2005). Alerting to, monitoring, and detecting the mistiming of events is thought to rely on fronto-cerebellar circuitry, and is associated with problems with timing, motor coordination and 'timeliness of behaviour'(Barkley, 2011d; Nigg & Casey, 2005). Finally, the fronto-limbic circuit has also been implicated in ADHD, and is thought to underlie the detection and evaluation of emotionally significant events or situations and reinforcement learning. In turn, this circuit has been associated with the following symptoms: hyperactivity-impulsivity, emotional impulsivity, motivational difficulties, and propensity to aggression (Barkley, 2011d; Nigg & Casey, 2005). These circuits are important in basic learning that forms the

foundation for behavioural, cognitive and emotional control, as well as adjusting to changes in the environment (Nigg & Casey, 2005). There is also recent evidence showing that the trajectory of early abnormal brain development within ADHD is sex dependent. For example, 4.5 and 8 year old boys with ADHD have less right-lateralized frontal alpha asymmetry than typically developing boys, whereas girls with ADHD show a more right-lateralized asymmetry pattern than typically developing age-mates (Mahone & Wodka, 2008). Similarly, Hermens and colleagues (2005, as cited in Mahone & Wodka, 2008) showed that irrespective of ADHD subtype, female adolescents with ADHD have focal frontal increase in theta and electrodermal activity, while male adolescents with ADHD show a widespread increase in theta activity. Whereas certain circuits have been hypothesized to be impaired in ADHD, and neural abnormalities have been reported, it is noteworthy to remember that there is no neuroanatomical profile that is consistent across all individuals with ADHD (Miller, 2012).

As previously mentioned, DSM-5 criteria continue to fail to account for the developmental changes in symptoms over time, although examples more appropriate for older ages have been provided (APA, 2013a). This limits the identification of many adolescents and adults who have ongoing and significant impairment due to ADHD despite not meeting full diagnostic criteria for the disorder (Miller, 2012). This has also been used to explain the lower persistance rates of the disorder into late adolescence and adulthood (Barkley, Murphy, & Fischer, 2008). Relatedly, a common diagnostic issue is whether the presence of less than the required number of symptoms warrants clinical attention (Kalbag & Levin, 2005). The current polythetic model of ADHD, in which various combinations of at least six symptoms are required for diagnosis, implies that

ADHD symptoms form a continuum rather than marking a categorical boundary between having and not having the disorder (Lubke et al., 2009). Moreover, for the symptoms to be diagnostic of ADHD they have to be developmentally inappropriate and result in impairment in major life activities (Barkley, Murphy & Fischer, 2008). By extension, given that the ADHD symptoms reflect extremes on the developmental continuum that are maladaptive and inconsistent with development level, it is possible to categorize all individuals somewhere on this spectrum. That is, some have suggested that ADHD reflects "an extreme on the quantiative manifestation of normal behaviour" (Acosta et al., 2004, p. 3) allowing for the investigation of ADHD symptoms in a nonclinical sample. Past research has examined the severity of ADHD symptoms in clinical (e.g., Barkley et al., 2006) and nonclinical (e.g., Rodriguez & Span, 2008) samples, with more severe symptoms of ADHD being positively correlated with impairment in daily life activities and drinking habits, respectively.

Presently, there are no differences in recommendations for ADHD with respect to diagnostic cut points according to the sex of the individual as per the DSM-5 (APA, 2013a). Further, the current set of symptoms neglects to mention certain central features of ADHD, such as emotional impulsiveness, poor emotional self-regulation, and poor working memory. Applying results from the UMass study of clinic-referred adults with ADHD, Barkley, Fischer and Murphy (2008) encourage the use of nine symptoms based largely on the construct of impulse control and attention in identifying adults with ADHD, rather than the DSM-5 symptom list. Examples from this alternative list of symptoms are: 'often have poor follow through on promises or commitments I make to others', 'start a project or task without reading or listening to instructions' and 'often

have difficulty performing things in proper order' (Barkley et al., 2008). Finally, the current cutoffs and pattern of symptoms reflect research conducted mainly on males, and thus may need to be adjusted accordingly to reflect female-specific symptom profiles (Nadeau & Quinn, 2002; Rasmussen & Levander, 2009). A meta-analysis conducted by Gaube and Carlson (1997) suggested that women have a different pattern of symptoms, more dominated by inattention and less by hyperactivity, and present with higher rates of internalizing symptoms (e.g., mood and anxiety problems) than externalizing symptoms (e.g., conduct disorder) that are more common in males. Applying Barkley's broadband domains, meta-cognitive symptoms linked to dysfunction within the inattention domain (e.g., emotional self-regulation, planning/problem solving, and working memory) may be more affected in females with ADHD. Exemplifying the potential sex inappropriateness of current diagnostic criteria, females are more likely to have greater impairment than average when assessed using behaviour ratings of ADHD symptoms and yet not meet the diagnostic threshold (Waschbusch & King, 2006, as cited in Mahone, 2010). Thus, the relative neglect of incorporating these symptoms in the diagnosis of ADHD might explain the sex differences in prevalence. Notably, some studies do not report these sex discrepancies in the prevalence of ADHD inattention and hyperactivity/ impulsivity symptoms (Biederman, Faraone, Monuteaux, Bober, & Cadogen, 2004; Rasmussen & Levander, 2009), although their results may be due to sampling bias inherent in studies utilizing self-referral/treatment-seeking individuals. Further, whereas Biederman and colleagues (2004) report similar phenotypic features in both males and females with ADHD, they do highlight that females with ADHD had significantly higher inattentive scores than their male counterparts. In line with this finding, Fedele and colleagues

(2012) report that college-aged ADHD females significantly endorse greater number of inattentive and hyperactive symptoms than college males with ADHD. Moreover, they show that even after controlling for symptom severity, emerging adult females with ADHD report having greater levels of impairment.

Human Pubertal Development

The lack of a clear and consistent pattern of results across studies examining symptom, behaviour, neuropsychological and neuroanatomical profiles of individuals with ADHD can at least partly be attributed to developmental factors (Halperin & Schulz, 2006; Mahone, 2010). Adolescence marks a major developmental milestone, involving dramatic changes in physical, psychological, and social maturity (Paus, Keshavan, & Giedd, 2008). These developmental changes make adolescence a "period of vulnerability and adjustment" (Casey, Jones, & Hare, 2008, p. 111). Reflecting this vulnerability, this developmental period is a time of increased prevalence of several psychiatric illnesses (e.g., mood disorders and eating disorders; Paus et al., 2008), and risky behaviours (e.g., drug and alcohol use and unprotected sex; Casey & Jones, 2010). Fundamental to the changes occurring in adolescence are sex specific effects presumed to be caused, at least in part, by the increase in secretion of circulating sex steroids with the onset of puberty (Cahill, 2006).

Puberty, functionally coupled with adolescence, is defined as a period of elevated secretion of gonadal steroid hormones. The onset of puberty marks the start of a 'sensitive period' in the development of and changes to the structural organization of the nervous system (Sisk & Zehr, 2005; Zehr, Culbert, Sisk & Klump, 2005;). In humans, pubertal maturation begins with hypothalamic gonadotropin releasing hormone (GnRH)

secretion, which activates the hypothalamic-pituitary-gonadal (HPG) axis (Palmert & Boepple, 2001; Sisk & Zehr, 2005). This period is "characterized by a gradual increase in the frequency and amplitude of intermittent episodes of GnRH release" (Sisk & Zehr, 2005, p. 164). In turn, GnRH stimulates the production and secretion of luteinizing hormone (LH) and follicle stimulating hormone (FSH), pituitary gonadotropins, which promote release of sex steroid hormones (i.e., estradiol in females and testosterone in males), and completion of gamete development (i.e., egg and sperm; Sisk & Foster, 2004; Sisk & Zehr, 2005). The higher levels of estrogen and androgen in turn, trigger the development of secondary sex characteristics (e.g., breast development in females and growth of facial hair in males; Sisk & Zehr, 2005). It is important to remember that while a hallmark of puberty is the production and secretion of gonadal steroids, puberty is not only a gonadal event (Sisk & Zehr, 2005). Rather, it should be viewed as a "brain event"; a period when sex hormones interact with the developing adolescent nervous system (Sisk & Zehr, 2005, p. 164). In fact, puberty-related changes continue into the third decade of life, thus corresponding to the lengthy maturation of the brain, in particular of the frontal cortex that continues to develop well into the twenties (Gogtay et al., 2004; Sisk & Zehr, 2005). Relatedly, the nervous system has a reciprocal influence on gonadal development and maturation (Sisk & Zehr, 2005).

The developing adolescent brain is highly receptive to the effects of gonadal steroid hormones. Circulating steroids (e.g., estradiol and progesterone) act in a time-sensitive and graded manner to shape adolescent brain development during a protracted process that spans more than a decade (Sisk & Zehr, 2005; Zehr et al., 2007). This process is highly individualized. As such, variation in the age of puberty onset

contributes to individual differences in developmental course and behavioral maturation (Sisk & Zehr, 2005). Relatedly, differences in the timing of puberty will contribute to the diversity of adult psychological characteristics, behaviours, and relative risks for psychopathology (Sisk & Zehr, 2005; Zehr et al., 2007). Finally, given the permanent organizational influence of gonadal hormones, effects dependent on the timing of puberty are likely to be permanent and observable in adulthood (Sisk & Zehr, 2005; Zehr et al., 2007).

The start of puberty in females is defined by the appearance of breast development, with a median age of onset of 10.0 years (Papadimitriou et al., 2008; Parent et al., 2003). Yet most studies examining the effects of timing of puberty onset typically use age at menarche as the marker of puberty onset, which has a median age of onset of 12.5 years (Dorn & Biro, 2011; Parent et al., 2003). Although the physiology of puberty is common to all individuals, its onset occurs across a wide range of ages in the normal population (i.e., 11 years or earlier to 14 years or older; Palmert & Boepple, 2001; Parent et al., 2003). Several pathologic conditions, such as central nervous system tumors or systemic illnesses, can influence timing of puberty (Palmert & Boepple, 2001). Moreover, the decrease in the age of pubertal onset over recent decades had been attributed to changes in family structure (e.g., father absence), better nutrition, increased obesity in childhood, and reductions in levels of childhood illness (DiVall & Radovick, 2009; Downing & Bellis, 2009). Yet, most variation in pubertal timing has no known etiology, and much of this variation stems from differences in the reactivation of the HPG axis (Dorn & Biro, 2011; Parent et al., 2003).

Variation in sex steroid exposure has been used to explain sex differences in neuroanatomy and cognitive function (Sisk & Zehr, 2005). For instance, it has been reported that females undergo an earlier peak in brain volume (Lenroot et al., 2007), greater growth in some structures over males (e.g., hippocampus; Sisk & Zehr, 2005), and less white matter growth than males during adolescence (Lenroot et al., 2007; Perrin et al., 2008). Given that circulating sex hormones influence virtually all mechanisms involved in the remodeling of the adolescent brain (e.g., dendritic elaboration, synaptic pruning, and axonal sprouting), it is not surprising that estrogen also plays a role in modulating cognition in the developing human brain (Sisk & Zehr, 2005). Relevantly, typical behavioural and cognitive changes noted in adolescence, such as risk-taking, reward sensitivity, sensation/novelty seeking, and basic cognitive abilities have been linked to pubertal maturation (Casey et al., 2008). For example, imbalance with the frontolimbic circuitry has been used to account for the greater prevalence of risky behaviours among adolescents and young adults, with only subcortical structures being directly linked to pubertal maturation (Steinberg et al., 2008). Whereas the cognitive functions most likely to be affected will be those linked to neuroanatomical areas with the highest concentration of estrogen receptors, the scientific community remains uncertain regarding the role of sex hormones in cognition.

Attention Deficit/Hyperactivity Disorder and Female Pubertal Development

As discussed above and in common with many neurodevelopmental disorders, the prevalence of ADHD differs in males and females. In addition to the limitations inherent in the DSM-5 nomenclature and proposed inadequacy of current rating scales in capturing symptom severity among females, this sex discrepancy may, in part, be driven

by hormonal influences. ADHD in females presents at a later onset and with more subtle clinical symptoms, often of the predominantly inattentive subtype (Keltner & Taylor, 2002). The direct assessment of subtype differences is essential when investigating the hormonal influences on ADHD symptom manifestation (Mahone, 2010). It has been suggested that while females may be protected to some extent from the symptoms of ADHD pre-puberty because of their earlier brain maturation, increased release of estrogen with puberty, and corresponding increase in dopamine receptors, may lead to a subsequent increase in ADHD symptoms (Fink, Rosie, Grace, & Quinn, 1996, as cited in Nussbaum, 2012; Keltner & Taylor, 2002). That is, deficits in cognitive control may be the result of the direct influence of sex hormones on the dopaminergic neural circuitry in the nucleus accumbens, striatum, and prefrontal cortex (Martel, Klump, Nigg, Breedlove, & Sisk, 2009). Animal models reveal female specific modulatory effects of estrogen and progesterone on dopamine in the striatum and nucleus accumbens (Xia & Becker, 1994, as cited in Martel et al., 2009). Similarly, higher levels of extracellular estrogen during the estrous cycle in female rats are accompanied by greater dopamine release in the striatum. It is also interesting to note that the amygdala, hippocampus, and orbital and medial prefrontal cortices, and the hypothalamic-pituitary-adrenal axis are targets of estradiol at puberty. Therefore, previous reports of remitting symptoms in ADHD into adolescence and young adulthood may be more reflective of the trajectory of male ADHD symptoms. Conversely, just when male symptoms begin to diminish, female symptoms begin to be more apparent and reported (Keltner & Taylor, 2002). Relatedly, it has been noted that increased hormonal fluctuations throughout the phases of the menstrual cycle are associated with increased symptomatology (Nadeau, Littman, &

Quinn, 2006). Further supporting the existence of a link between hormones, particularly estrogen, and ADHD in females is the existence of ADHD comorbidities known to be influenced by pubertal onset (e.g., eating disorders, anxiety, substance use and depression; Zehr et al., 2007). That is, given that the manifestation of many of the known common comorbidities in females with ADHD have been shown to be affected by pubertal timing (Bijlenga et al., 2011; Klump et al., 2012; Westling, Andrews, Hampson, & Peterson, 2008) it seems highly plausible that a correlation between pubertal onset and ADHD exists. Finally, similar to the imbalance noted within the frontolimbic circuitry used to account for the greater prevalence of risky behaviours among adolescents and young adults (Steinberg et al., 2008), it is likely that the neural circuits implicated in the inattentive symptoms and emotional dysregulation of ADHD (i.e., frontal-striatal and frontal-limbic circuits) would also be affected by the puberty-dependent imbalance in maturation between subcortical and cortical regions.

The Present Study

The literature reviewed above indicates that puberty is a key time for neuroanatomical changes and that circulating sex steroids likely play a significant role. Moreover, data suggest that circulating sex steroids modulate cognition, especially those cognitive functions that are underpinned by anatomical structures richest in estrogen receptors, such as the frontal cortex. The frontal lobes subserve various functions (e.g., affective regulation, attention/arousal, and impulse control), and the prefrontal area, particularly involved in executive function, has been chiefly implicated in ADHD (Nussbaum, 2012). There is growing evidence that subtle sex differences exist in the symptom profile, neuropathology and clinical sequelae of ADHD, and that hormonal

factors may play an important role in understanding ADHD in females. Yet, to date, there has been little research on this topic. The present study sought to address the current gaps in our understanding of how female pubertal maturation influences the extent of ADHD symptoms in a nonclinical female sample. This was primarily an exploratory study. Nevertheless, given the noted negative consequences of early puberty onset, such as disordered eating and anxiety (Zehr et al., 2007), sexual risk taking, substance use and anti-social behaviour (Downing & Bellis, 2009), it was predicted that aberrations from typical pubertal onset, specifically early maturation relative to peers, would be associated with elevated levels of ADHD symptoms, impairments in daily functioning, and difficulties in emotion regulation. The findings from the study were aimed to add to the general understanding of the relationship between puberty onset and executive functioning. Further, the study was designed to add to our understanding of ADHD prevalence rates among females, and the potential female-specific adolescent onset of presenting symptoms.

CHAPTER III

Methods

Design of Study

A quantitative, cross-sectional non-experimental design was used to examine the predictive value of constructs associated with ADHD (e.g., executive functioning) on the pubertal timing of typically developing female emerging adults. The study was exploratory in nature, with limited past research to guide hypothesis generation. That said, it was predicted that early pubertal onset would be associated with a greater number of ADHD symptoms and related impairments.

Participants

An invitation to participate in the study was sent via the existing participant pool system within the Department of Psychology at the University of Windsor. Participation was open to females aged 18 to 25 years within the pool. There were no restrictions based on race, socio-economic status, marital status, or neighbourhood of residence. Individuals who reported being unable to read, speak or write in English were excluded from participation. Finally, individuals with a self-reported history of traumatic brain injury were also excluded.

At the outset, an estimated required sample size was determined by a power analysis using the G-power program. A thorough literature review did not reveal prior studies upon which a hypothesized effect size could be estimated. As such, a small effect size value of 0.20 – 0.25 was chosen for the power analysis (Cohen, 1988). An alpha level = 0.05, effect size = 0.20 to 0.25, power level = 0.80, and one covariate suggested a sample size range of 158 to 244 participants. Correspondingly, the aim was to recruit

approximately 200 participants, however, recruitment proved easier than expected and information from a total of 254 female students was collected. One case was removed from the analysis because the same participant completed the study at two different time-points, for a final total of 253 participants.

The mean age of the total sample was 20.19 years (± *SD* = 1.69). As described in greater detail below, participants were grouped based on timing of pubertal onset: early (mean age ± *SD* = 20.58 ± 1.88), on-time (mean age ± *SD* = 20.09 ± 1.65) and late (mean age ± *SD* = 19.98 ± 1.52). The majority of the sample (65.2%) self-identified as Caucasian, with 9.88% Asian/Asian-descent, 9.09% African-Canadian/Black, 1.19% Hispanic/Latino, 0.39% Aboriginal and 13.8% mixed-race or other. 78.3% of the sample identified English as their primary language and 24.9% reported being able to speak another language. In terms of marital status, 59.3% reported that they were single, 37.2% were in a romantic relationship and 3.56% were married or cohabiting. In addition to their studies at the university, 64.0% of the sample reported that they were employed outside the home.

Parental education level was used as a proxy for socioeconomic status (SES). Specifically, 69.9% of participants reported that their mothers completed more than 12 years of formal education (range = 1 to 22 years of formal education). A similar rate was obtained for paternal education level, with 68.1% of participants reporting that their fathers completed some level of post-secondary education (range = 8 to 22 years of formal education). These rates are comparable to those reported by Statistics Canada in 2007 for the city of Windsor (62.04% of the population completed some level of post-secondary education; Statistics Canada, 2007).

With regards to disclosure of relevant medical history, two participants identified history of head injury accompanied with a loss of consciousness, seven participants disclosed history of seizures, six participants identified that they had a diagnosis of a learning disorder, and 15 participants reported having a current mental disorder (e.g., depression, anxiety). In an attempt to increase the generalizability of the findings, only information provided by individuals with a reported history of head injury was excluded from analysis, reducing the sample size to 251. Finally, it should be noted that the pubertal onset groups, described in detail below, did not significantly differ on any of these demographic variables.

Procedure

Once the participants arrived at the testing session and following the informed consent process, they were asked to complete a series of questionnaires. They were reminded that the session would last approximately one hour and that they would receive one psychology course bonus point following the completion of the questionnaires. If they agreed to continue, the following questionnaires were presented for completion: (1) demographics form, (2) Pubertal Development Scale-Retrospective Version, (3) Barkley Adult ADHD Rating Scale – IV (BAARS-IV), (4) Barkley Deficits in Executive Functioning Scale (BDEFS), (5) Difficulties in Emotion Regulation Scale (DERS); (6) Barkley Functional Impairment Scale (BFIS); (7) Risk-Taking Behavior questionnaire. The participants completed the questionnaires in randomized order.

Measures

Demographics questionnaire. Demographic information was collected via a form completed by all participants. The form requested general information about

identity, including date of birth, race/ethnicity, marital status, parental education and employment, handedness, height, weight, days since last menstrual period, in addition to information about the participants' medical (including current contraceptive use), and developmental histories. Appendices contain copies of all questionnaires administered.

Pubertal Development Scale – Retrospective Version (PDS-RV). Participants were asked to retrospectively answer questions on pubertal development using a modified version of the Pubertal Development Scale (PDS) obtained from Dr. Cheryl Sisk at Michigan State University. The original version of the PDS was designed for use with adolescents to assess current pubertal development of a number of secondary sex characteristics. Dr. Sisk and colleagues revised the modified version of the PDS in order to assess timing of pubertal development in post-pubertal adults. Initially used with an undergraduate sample at a large Midwestern university, Sisk and colleagues (2007) report that the modified measure has good psychometric properties. Specifically, they reported good internal consistency ($\alpha = 0.80$ in females; $\alpha = 0.84$ in males) and excellent test-retest reliability ($r = 0.87$ for females; $r = 0.83$ for males). For most items, the participants were asked to recall the timing of pubertal development relative to their peers (i.e., "much earlier than others" (1), "somewhat earlier" (2), "about the same time" (3), "somewhat later" (4), "much later" (5), or "do not know"). For other items, the participants were asked to estimate the age at which an event occurred. The total PDS-RV score was calculated by summing the scores from the following 6 items: "In general, do you think your development was any earlier or later than most other girls?"; "Do you think your first period was any earlier or later than most other girls?"; "Do you think your breasts developed any earlier or later than most other girls?"; "Would you say that your

growth in height was any earlier or later than other girls?"; "Would you say that your growth of body hair was any earlier or later than other girls?"; "Would you say your skin changed any earlier or later than other girls?" Smaller summed total scores reflected earlier pubertal timing. All other items on the scale were informational, and were included to exclude outliers or identify potential new covariates.

The mean PDS-RV total score for all participants was calculated to be 17.26 (*SD* = 4.084), with the scores ranging from 6.00 to 29.0. The PDS-RV total score was also examined per pubertal onset group, with the early pubertal onset group having a mean score of 12.21 (*SD* = 1.966, range = 6.00 – 14.75), the on-time onset pubertal group having a mean score of 17.22 (*SD* = 1.314, range = 15.00 – 19.18), and the late onset pubertal group having a mean score of 22.74 (*SD* = 2.156, range = 19.89 – 29.00). Cronbach's alpha was calculated as a measure of internal consistency. This analysis revealed a good alpha value of 0.81.

Barkley Adult ADHD Rating Scale-IV (**BAARS-IV**; *Barkley, 2011a*)**.** Designed with consideration of DSM-IV diagnostic criteria, the BAARS-IV is a self-report questionnaire in which the participants were asked to report their current ADHD symptoms. Specifically, with regards to the current symptoms interview, the participants were asked to indicate to what extent each item described their behavior during the past six months. The possible response for the questionnaire ranged from "Never/Rarely" (1) to "Very Often" (4). A total ADHD score and symptom count was calculated by adding up the scores for each item answered. From the BAARS-IV – current symptoms questionnaire, 4 subscale totals were calculated related to the four recognized ADHD symptom dimensions: Inattention, Hyperactivity, Impulsivity, and Sluggish Cognitive

Tempo (SCT). The internal consistency, construct validity, discriminant validity and criterion validity are all reported to be satisfactory (Barkley, 2011a).

Examination of the total scores for the BAARS-IV current symptoms questionnaire for all study participants revealed a mean total score of 27.24 (*SD* = 6.498, range = 18.0 – 51.0), out of a possible maximum score of 108. Relatedly, analyzing the scores independently for each pubertal onset group revealed that the early pubertal onset group had a mean total score of 28.28 (*SD* = 6.846, range = 19.0 – 50.0), the on-time pubertal onset group had a mean total score of 26.37 (*SD* = 5.860, range = 18.0 – 51.0), whereas the late pubertal onset group had a mean total score of 27.95 (*SD* = 7.229, range = 18.0 – 49.0). Table 1 displays the mean, standard deviation and range of scores for each subscale separately per pubertal onset group. The internal consistency for all 27 items was excellent, with an alpha value of 0.90. Calculation of Cronbach's alpha for the inattention, hyperactivity, impulsivity and sluggish cognitive tempo subscales revealed alpha values of 0.82, 0.59, 0.69, and 0.87, respectively.

Barkley Deficits in Executive Functioning Scale (**BDEFS**; *Barkley, 2011b*). The BDEFS is an 89-item Likert-type rating scale designed to evaluate the variety of behavioural, emotional, and motivational symptoms linked to executive functioning deficits. More specifically, this self-report measure aims to capture self-regulation ability within five domains: Self-Management to Time, Self-Organization/Problem Solving, Self-Restraint (Inhibition), Self-Motivation and Self-Regulation of Emotion. Correspondingly, the scale provides a global measure of deficits in executive functioning, as well as subscale scores for each of the five domains. Individuals are asked to indicate how frequently they experienced each of the problems in the past six months, with

possible responses ranging from “Never or Rarely” (1) to “Very Often” (4). Developed for use in a variety of settings, including research, the measure has been shown to have satisfactory reliability and validity (Barkley, 2011b).

The mean BDEFS total score for all participants was 148.90 (*SD* = 34.64, range = 91 – 273), out a possible maximum total score of 356. Table 1 includes the score statistics for each BDEFS subscale for all participants and per pubertal onset group. Internal consistency for the total score was evaluated to be excellent, with an alpha value of 0.97. Relatedly, the Cronbach’s alpha values for each subscale were also evaluated to be satisfactory (Self-Management to Time = 0.94; Self-Organization/Problem = 0.94; Self-Restraint = 0.85; Self-Motivation = 0.84; and Self-Regulation of Emotion = 0.92).

Difficulties in Emotion Regulation Scale (**DERS**; *Gratz & Roemer, 2004*)**.** The DERS is a self-report measure designed to evaluate clinically significant difficulties in emotion regulation. The measure was originally developed with a large undergraduate sample at the University of Massachusetts – Boston (Gratz & Roemer, 2004), and has since been used with substance abusers and other clinical populations (Gratz et al., 2008). Participants were asked questions reflecting difficulties in four domains of emotion regulation, including (1) awareness and understanding of emotions; (2) acceptance of emotions; (3) the ability to use goal-directed behaviour and control impulsive behaviour when having negative emotions, and (4) ability to use effective emotion regulation strategies. They were asked to indicate how often the items apply to them, with responses ranging from “almost never” (1) to “almost always” (5). The total DERS score was calculated by summing the scores for all 36 items, with possible scores ranging from 36 to 180. Gratz and Roemer (2004) report preliminary findings suggesting that the DERS

has high internal consistency, good test-retest reliability, and adequate construct and predictive validity.

The mean DERS total score for all study participants was 81.80 (*SD* = 21.98), with the scores ranging from 39.0 to 153.0. Please refer to Table 1 for the mean, standard deviation and ranges for the score breakdown per pubertal onset group and per DERS subscale. The internal consistency for all 36 items of the DERS was excellent, with an alpha value of 0.94. Cronbach's alphas were also calculated for each DERS subscale, revealing the following: Nonacceptance of Emotional Responses subscale (α = 0.89), Difficulties Engaging in Goal-Directed Behaviour subscale (α = 0.91), Impulse Control Difficulties (α = 0.88), Lack of Emotional Awareness (α = 0.84), Limited Access to Emotion Regulation Strategies (α = 0.89), and Lack of Emotional Clarity (α = 0.82).

Barkley Functional Impairment Scale (**BFIS**; *Barkley, 2011c*)**.** The BFIS is a self-report measure that was used to ask participants to rate the degree of psychosocial impairment they believe they are experiencing in 15 major domains of adult life activities (i.e., home-family, home-chores, work, social- strangers, social-friends, community activities, education, marriage/cohabiting/dating, money management, driving, sexual relations, daily relations, daily responsibilities, self-care routines, health maintenance, and childrearing; Barkley, 2011c). Participants were asked to indicate how much difficulty they have functioning effectively in each of the major life activities during the past six months, with possible responses ranging from "Not at all" (0) to "Severe" (9). A summed total score for the questionnaire was calculated by adding up the individual item scores, excluding those items that were answered "Does not apply" (99). The scale is

reported to have satisfactory internal consistency, test-retest reliability, and validity (Barkley, 2011b).

The mean impairment score for all participants was 2.071 (SD = 1.583) out of a possible maximum mean impairment score of 9. The scores for the scale ranged from 0 to 7.69. See Table 1 for the mean impairment score, standard deviation and range for each pubertal onset group. Cronbach's alpha for the BFIS is excellent in this sample, with an alpha value of 0.96.

The Risk-Taking Behavior Questionnaire (RTBQ). Participants were asked to complete a self-report measure of their engagement in and frequency of specific risky behaviours across five domains, including driving, drugs/alcohol/cigarettes, law breaking, family, and sexual behaviour. This is an unpublished measure originally developed to survey risk-taking behaviour in a related study of younger adolescents and has been used extensively with university students. Preliminary analyses suggest that this measure has adequate psychometric properties (Miller, White, Knezevic, Ostojic, & Niemasik, manuscript submitted for publication). Participants were asked how frequently they engaged in the listed behaviours over the past six months, with possible responses ranging from zero ("Never") to four ("11 or more times in the past six months"). The final of the sexual behaviour items, the total lifetime number of sexual partners, was scored slightly differently (0 = no sexual partners, 1 = 1-2 sexual partners, 2 = 3-5 sexual partners, and 3 = 6 or more sexual partners. Despite the fact that the RTBQ is differentiated into five domains, only the summed total score is interpreted. The summed total score for the RTBQ was calculated by adding up the individual item scores, with a possible maximum score of 99.

The mean RTBQ total score for all participants was calculated to be 14.63 (*SD* = 9.144), with the scores ranging from 0.00 to 44.0. The RTBQ total score was also examined per pubertal onset group, with the early pubertal onset group having a mean score of 16.61 (*SD* = 9.757, range = 1.00 – 42.0), the on-time onset pubertal group having a mean score of 13.76 (*SD* = 8.635, range = 0.00 – 42.0), and the late onset pubertal group having a mean score of 14.34 (*SD* = 9.386, range = 0.00 – 44.0). Internal consistency, evaluated by calculating Cronbach's alpha using all 23 items, was adequate with an alpha value of 0.79.

Data Analyses

All analyses were performed using the Statistical Package for Social Science (SPSS) for Mac, Version 21.0. Prior to conducting the main analyses the data were assessed for patterns of missingness and missing data was subsequently imputed using the expectation-maximization procedure. A one-way multivariate analysis of covariance (MANCOVA) was initially chosen as the statistical test of choice with puberty onset as the independent variable with three factor levels: early, on-time, and late puberty onset. MANCOVA was chosen because it was speculated that given the related content of the questionnaires the outcome variables would likely be correlated. Further, the number of days since the last menstrual period was suspected to be a covariate (K. Milne, personal communication, March, 2012). Days since last menstrual period was initially chosen as the covariate because fluctuating sex hormone levels during the menstrual cycle have been shown to affect performance in a wide range of domains, including fine motor performance (Bayer & Hausmann, 2012), prepulse inhibition (Kumari et al., 2010), learning and memory (Gasbarri et al., 2008), inhibitory output control (Colzato, Hertsig,

van den Wildenberg, & Hommel, 2010), attention (Colzato, Pratt, & Hommel, 2012), and female dominance motivation and behaviour (Stanton & Schultheiss, 2007). Moreover, it has been argued that ignoring menstrual cycle status of female respondents may bias self-reporting and clinician's judgments about numerous syndromes and disorders (Endicott & Shea, 1989). This continuous variable was split into two groups: less than or equal to 14 days or more than 14 days since last menstrual period; with former corresponding to the follicular phase of the menstrual cycle and the latter, the luteal phase (Barnett et al., 2004; Butt, 1979). Notably, however, this variable was found to not be associated with the outcome variables, and thus was removed from all further analyses.

Relatedly, as described below in greater detail, before proceeding with the analysis, verification of the statistical assumptions for MANCOVA was done; revealing that the assumptions were not met (described below). All subsequent attempts to remedy this problem were unsuccessful. As a result, the decision to proceed with a different statistical test (i.e., multinomial logistic regression) was made. Multinomial logistic regression was chosen because this statistical procedure is thought to be robust to violations of normality and homogeneity of variance-covariance matrices, and makes no assumption that the predictors have to be linearly related (Tabachnich & Fidell, 2007, p. 437). Further advantage of this technique is that it allows for the selection of a reference group for all comparisons (Field, 2009, p. 301). In contrast to the MANCOVA, however, the *outcome* variable in the multivariate logistic regression was timing of puberty, with three categories: early, on-time, and late puberty onset, whereas the subscale and composite totals on the questionnaires served as the *predictors*. As was done by Zehr and colleagues (2007), quartiles for the summed total score of pubertal development scale

were used to categorize early (lowest quartile), on-time (middle-two quartiles), and late (highest quartile) pubertal onset. Next, logistic regression analysis was conducted to allow for a direct comparison between the early pubertal onset group and the on-time pubertal group. Finally, this was followed up by descriptive analyses. A $p < 0.05$ was selected as the statistical significance level for all analyses. This is the standard alpha value utilized most often by researchers (Field, 2009, p. 281) and was considered appropriate given the exploratory nature of the study. Interestingly, Tabachnick & Fidell (2007; p. 455) report that Hosmer & Lemeshow (2000) suggest using a less stringent cut-off, in the range of 0.15 to 0.20, for the inclusion of a variable in order to avoid erroneously removing a predictor that may be involved in a suppressor effect. Notably, however, as described below the entry method chosen for the step-wise logistic regression is designed to minimize Type II error.

Missing Data

Investigation of the absence of data, including the pattern of missingness was done in order to address the fact that missing data may bias the analysis and result findings (McKnight, McKnight, Sidani, & Figueredo, 2007). Computing the proportion of missing data using the sparse matrix method yielded an overall item-non-response rate of 0.184%, whereas calculation of the response rate using the complete case method for the 253-participant dataset was 18.9%. Correspondingly, the sparse-matrix-to-complete-case ratio, was calculated to be 0.00976, signifying that the average amount of data missing per incomplete case is approximately 0.976%. Further, the messiness index was found to be 0.813, indicating a messy missing data pattern in which each participant has her own pattern of missing data (McKnight et al., 2007, p. 109). Importantly, a messy

missing data pattern suggests that the data is missing completely at random (MCAR).

Closer inspection of the individual outcome questionnaires (i.e., BAARS-IV, BDEFS, DERS, and RTBQ) further supported the conclusion of a scattered pattern of missingness, with each questionnaire having an overall item non-response rate less than 1%. It should be noted that apart from one participant not completing the entire DERS, there were no cases in which participants left more than a few items incomplete per questionnaire. With regards to the Puberty Development Scale-Retrospective version (PDS-RV), the 6 items used to calculate the total PDS-RV score, and the PDS-RV item reflecting age of first menstruation were analyzed for missingness. It should be noted that data was considered missing if the individual responded "Don't Know" or if they left the item blank. The overall item non-response rate was calculated to be 3.29% with the greatest number of missing items concentrated on items pertaining to growth of body hair relative to peers (14.2%) and timing of skin changes relative to peers (7.87%). Pertaining to the three demographic variables used to impute PDS-RV missing values, race, weight and height, with the latter two used to calculate a Body Mass Index score, only one case was missing in the sample for each variable.

To examine the impact of the missing data and to ensure that the missingness was not conditional on the other variables in the data set, Little's MCAR test was conducted (Missing Completely at Random; Little, 1988). Given that the null hypothesis for the test is that the data are MCAR, the non-significant result from the Little's MCAR test (x^2 =7868.937; df = 7830; p = 0.376) suggested that the data are at least Missing at Random (MAR), and therefore, the missingness is not dependent on the other values in the data set (Little, 1988). In line with the visual inspection of the missing data, and taking into

account the existing selection bias in the sample (i.e., sample consisted of those participants who voluntarily signed up for the study and completed the questionnaires), the conclusion that the data are likely at least missing at random is justified.

Imputation of Missing Data

Expectation-Maximization (EM) was used to replace missing values. As per Tabachnick and Fidell (2007, p. 71), EM method provides the "simplest and most reasonable approach to imputation of missing data", when there is evidence that the data are at least missing at random. As such, given the minimal amount of missing data, and evidence that the values are missing at random, use of EM was deemed appropriate. To obtain less biased estimates of the missing data, imputation of missing data was done separately for the PDS-RV and the outcome measures. Missing PDS-RV items were imputed using the key PDS-RV items (i.e., PDS items: 1, 2, 4, 6, 13, 17, and 21), a calculated body mass index (BMI) score and race. The latter two variables were chosen because both the BMI score and race have been associated with the timing of pubertal onset (Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001; Lee et al., 2007). Inclusion of the other PDS-RV items (i.e., PDS items: 8, 11, 15, 19, 23) led to a failure to converge after 100 iterations. Consequently, these items were not included because the lack of convergence would lead to unstable estimates. With regards to imputing data for the outcome measures using EM, the missing values for the BAARS-IV Current scale, BDEFS, BFIS and DERS were done together. The RTBQ items were not included because the EM algorithm conducted with these items failed to converge after 25 iterations, even though Little's MCAR test was not significant. Again, given that the lack of convergence would lead to unstable estimates, missing values for the RTBQ were

imputed independently of any other variables in the dataset. The individually imputed datasets were combined and all subsequent analyses were conducted using this new dataset. Notably, however, although the results presented reflect analysis done using the imputed datasets, given the small amount of missing data the original and imputed datasets were very similar.

Analysis of the Assumptions of MANCOVA

Prior to conducting the main analyses as planned using a MANCOVA, the data were first examined to see if the assumption for MANCOVA were met. Although not a direct assumption of MANCOVA, examination of univariate normality for the independent variable, PDS-RV total score measured as a continuous variable, was done to ensure that break-down into groups based on quartiles would be justified and unbiased. The assumption of normality for the PDS-RV total score was assessed using tests of normality and examination of kurtosis and skewness values. Although, the Kolmogorov-Smirnov test was significant (p<0.000), the Shapiro-Wilks test was not (p = 0.096), nor did kurtosis and skewness values exceed ±2 and ±3, respectively. Further, examination of the histogram and Q-Q plots suggested a normal distribution for the PDS total score.

The assumption of multivariate normality for the outcome variables was assessed through examination of the skewness and kurtosis values, the Shapiro-Wilk statistic, and bivariate scatterplots. Given that a necessary, but not sufficient, condition for multivariate normality to hold, is to ensure that observations on each dependent variable must follow a normal distribution in each group, univariate normality was first examined for each variable independently (personal communications, D. Jackson, January 2013; Tabachnick & Fidell, 2007, p. 78). This is especially important if inference is an aim

(Tabachnick & Fidell, 2007, p. 79). Examination of univariate normality for the BAARS-IV current scale by visual inspection of Q-Q plots and histogram showed a distribution with a positive skew. This conclusion was supported as both tests of normality were significant at the $p<0.001$ level. A positive skew was also observed for all subscale totals. Attempts to correct normality by conducting square root and logarithmic transformations as recommended by Tabachnick and Fidell (2007; p. 88) were not successful. Similarly, examination of univariate normality for the BDEFS and BFIS also revealed non-normal distribution of scores; a conclusion supported by significant tests of normality (i.e., Kolmogorov-Smirnov ($p<0.001$) and Shapiro-Wilks tests ($p<0.001$)) and visual inspection of the histograms and Q-Q plots. Further, the assumption of normality was also not met for all of the associated subscale totals. In contrast to the BAARS-IV current scale, conducting a logarithmic transformation did improve normality for the BDEFS total score as indicated by a non-significant Kolmogorov-Smirnov test ($p = 0.200$) and visual inspection of the histogram. It should be noted, however, that the Shapiro-Wilks test was still significant for the total score ($p = 0.030$) after the logarithmic transformation, and neither transformation improved normality for the BDEFS subscales. Relatedly, performing a square root transformation did improve normality for the BFIS as indicated by non-significant Kolmogorov-Smirnov ($p = 0.200$) and Shapiro-Wilks tests ($p<0.119$), and visual inspection of the histogram and normal Q-Q plots. Notably, logarithmic transformation yielded a negatively skewed distribution. With regards to the DERS, examination of normality for the total score revealed a slight positive skew, and tests of normality supported conflicting conclusions (Kolmogorov-Smirnov ($p = 0.075$) and Shapiro-Wilks ($p = 0.002$)). Moreover, evaluation of the kurtosis and skewness

values showed that they did not exceed their respective cutoffs of ±2 and ±3. Notably, visual inspection of the histogram and Q-Q plots did suggest that the distribution was positively skewed. Again, in attempt to fix this slight deviation from normality, square root and logarithmic transformations were done (Tabachnick & Fidell, 2007, p. 88) The square root transformation made the distribution normal as indicated by non-significant tests of normality: Kolmogorov-Smirnov (p=0.200) and Shapiro-Wilks (p = 0.489). Further, as depicted on the Q-Q plots and histogram, the data distribution appeared normal. No outliers were identified on the box-plot. In line with this finding, the z scores for the DERS total score before and after the square root transformation did not exceed ±3.29. Further, examination of normality for each PDS-RV group showed that the assumption of normality was met. In contrast, the subscales on the DERS were not normally distributed, and both square root and logarithmic transformations were not successful in correcting this deviation from normality. Finally, the assumption of univariate normality was also not met for the RTBQ, and conducting square root and logarithmic transformations did not help improve normality. Therefore, given that univariate normality was violated in nearly all cases, a necessary condition for meeting the assumption of multivariate normality, the assumption of a multivariate normal distribution was also violated (Stevens, 2009). Fortunately, Stevens (2009) notes that this assumption is robust with respect to type I error.

The assumption of homogeneity of variance-covariance was tested using Box's M test. Box's M was significant for all predictor variables, indicating that this assumption was violated. Box's M value remained significant after removal of the outliers, further supporting the retention of all usable cases. Notably, this test is very sensitive to

violations of normality. Further, it is conditionally robust if the group sizes are equal or approximately equal (largest/smallest <1.5; Stevens, 2009), which was not the case in the present analysis. Therefore, one can be confident in the conclusion that this assumption was also violated.

To ensure that the assumption of independence of observations was met, the data collection process was designed to minimize any chance that the responses for one person would affect the ratings on the measures for another. For instance, participants were always spaced far enough apart while completing the questionnaires to not allow for copying of responses, and they were not given the opportunity to communicate or share their responses with one another. That said, the shared school environment participants have may limit complete independence of observations. Further, each case reflects the responses of a unique participant. In the one case that a participant completed the study twice, the responses from their second time was removed from all analyses. Therefore, it can be argued that the assumption of independence of observation was met. This is especially important given that the assumptions of multivariate normality and homogeneity of variance-covariance matrices were not met.

Finally, the assumption of adequate sample size was assessed. Including all identified outliers, and after removing the repeated case and the two individuals who reported a history of head injury, a final sample of 251 was used for subsequent analyses. Using quartiles to create pubertal timing categories led to unequal group sizes, with the early pubertal onset group having 64 participants, the on-time pubertal onset group had 127 participants, and the late pubertal onset group had 60 participants. The total sample size/number of variables ratio was calculated to be 41.8:1, which met the criteria that the

ratio should be at least 20:1 (Stevens, 2009). Thus, the assumption of adequate sample size holds, and one can be more confident in the interpretation of the results. It should be noted that the late pubertal onset group was removed for the logistic regression analysis described below, bringing the final sample size for this analysis to 191 participants.

Attempting to improve normality, identification and removal of outliers and influential observations was done by entering all variables of interest in a linear regression model (personal communication, D. Jackson, May 2013). In order to detect univariate outliers, standardized residuals were examined. Using $z = \pm 3.29$ standard deviations from the mean as the cut-off, no outliers were detected. Mahalanobis distance (D^2) was calculated in order to detect multivariate outliers. Using the D^2 cut-off value of 49.728 ($df = 23$, $k = 23$, $\alpha = 0.001$), 11 cases were detected. These 11 outliers were removed and the assumption of multivariate normality and homogeneity of variances was reassessed. The assumption of multivariate normality was still violated, and no significant improvements in skewness and kurtosis values were observed following the removal of the cases. Similarly, bivariate scatterplots revealed no change in normality, and thus the outliers on *y* were retained. Finally, no influential observations were found in the data, as no Cook's distance value came close to 1. Further justifying keeping these cases, when data is not normally distributed it is difficult to identify if those cases are not from the population of interest.

Justification for Conducting Multinomial Logistic Regression and Logistic Regression

As a result of violating both the assumption of normality and homogeneity of variance, and given that the only assumptions met were adequate sample size and

independence of observations, conducting a parametric test, such as a MANCOVA, is not justifiable. Consequently, the analysis needed to be conducted using either a non-parametric test, or a test not requiring the strict adherence to these assumptions. Thus, multinomial logistic regression, given that it is robust to violations of normality and homogeneity of variance-covariance matrices, was chosen (Tabachnick & Fidell, 2007, p. 437). Notably, logistic regression does assume independence of observations, linearity, and absence of multicollinearity (Tabachnich & Fidell, 2007, p. 443; Field, 2009, p. 273). As described above, the assumption of independence of errors/observations was met. Similarly, for all but two predictors (i.e., BDEFS Self Management of Time Score and BDEFS Self-Regulation of Emotion Score) the assumption of linearity of the logit was met. This assumption was evaluated by examining "if the interaction term between the predictor and its log transformation was significant" when entered in the logistic regression model (BDEFS Self Management of Time Score: $p = 0.036$ and BDEFS Self-Regulation of Emotion Score: $p = 0.035$; Field, 2009, p. 273). Finally, testing for multicollinearity revealed no violation of this assumption as the tolerance values for all of the predictors were not less than 0.1, nor were VIF values greater than 10 (Field, 2009, p. 297).

In addition to being more flexible than other techniques, multinomial logistic regression allows for the selection of a reference group for all comparisons (i.e., on-time pubertal onset; Field, 2009, p. 302), as does logistic regression. Stepwise multinomial logistic regression utilizes statistical criteria to include and remove predictors from the equation (Tabachnick & Fidell, 2007, pp. 454). Although criticized for this, this methodology serves a good purpose in screening or hypothesis generating (Tabachnick &

Fidell, 2007, p. 454), which is appropriate given the exploratory nature of this study. Although underutilized in social science research, both multinomial and binary logistic regression is a very useful method when the outcome variable is categorical and the predictors are continuous and/or categorical (Davis & Offord, 1997). Stepwise is defensible when conducting analyses in an area where there is limited or no existing research (Field, 2009, p. 272). Moreover, it allows for the selection of those variables that are significantly associated with pubertal onset, while concurrently removing predictors that have less of an impact. When using a stepwise method, the backward method is preferred over the forward method because it allows for the detection of suppressor effects, and thus is less likely to cause a Type II error (Field, 2009, p. 272). Finally, with regards to the test statistic to be used in the stepwise method, the likelihood ratio method is preferred, given that the Wald statistic may produce inaccurate results under certain conditions (Field, 2009, p. 272). For these reasons, multinomial logistic regression and logistic regression were both done using the step-wise backward entry method, with the likelihood ratio statistic used as the criterion for comparing the models with and without the predictor. This procedure was later followed-up with step-wise forward entry method, to see if the same set of predictors have significant score statistics.

Multinomial logistic regression was done first because it allowed for the comparison of both the early and late pubertal onset groups to the on-time pubertal onset group. This analysis revealed that any significant differences on the administered questionnaires only existed between the early and on-time pubertal onset groups. Consequently, and in concordance with the hypothesis predicting the negative consequences of early pubertal onset, this analysis was followed up with logistic

regression, comparing these two groups. This analysis paralleled the findings from the multinomial logistic regression and were the findings that were interpreted.

CHAPTER IV

Results

Logistic Regression Model

For each logistic regression analysis, interpretation of the goodness-of-fit test using the "-2 Log-Likelihood Statistic" was performed in order to assess how well the logistic regression model fit the data. Further, examination of the B-value (unstandardized regression coefficient), odds ratio, and significance of the Wald statistic (measure of the unique contribution of each predictor) was done to determine how well each predictor in the model fit the data (Field, 2009, p. 284-89). In this study, the potential explanatory variables were entered in groups based on their corresponding questionnaires. That is, all subscales of one questionnaire were entered independently of the subscale totals corresponding to the other questionnaires. Relatedly, in order to avoid issues of multicollinearity, symptom count totals and total scores were not entered along with the matching subscale totals in the same logistic regression analysis. Instead, symptom counts corresponding to the same questionnaire were entered together, whereas all questionnaire total scores were entered and analyzed together to determine whether or not they are significant predictors in the model. Finally, all variables that were identified as significant in the previous analyses were entered in the same logistic regression analysis.

Outcome Variable in the Logistic Regression Analysis

The outcome variable for all of the logistic regression analyses was pubertal timing, determined by the participant's score on the PDS-RV questionnaire. As described above, pubertal onset had three factor levels: early, on-time and late pubertal

onset, and quartiles for the summed total score of pubertal development were used to categorize early (lowest quartile), on-time (middle-two quartiles), and late (highest quartile) pubertal timing. For the purposes of comparing early to on-time pubertal onset using logistic regression, pubertal onset was operationalized as a dichotomous variable, with those characterized as having on-time pubertal onset given a value of 0 and those identified as having early pubertal onset given a value of 1. This coding was chosen in order to aid in interpretation by labeling the on-time pubertal onset category as the reference group. As such, the odds ratio was interpreted at the ratio of the probability of membership in the early pubertal onset group occurring and the probability of this not to occur when the predictor variable increases by one. After removing two participants from the analysis because they disclosed having a head injury resulting in a loss of consciousness, the number of participants categorized in the on-time and early pubertal onset groups was 127 and 64, respectively.

Independent Variables in the Logistic Regression Analysis

Barkley Adult ADHD Rating Scale – IV: Self-report – Current Symptoms. The four subscale totals of the BAARS-IV self-report of current symptoms questionnaire (i.e., Inattention, Hyperactivity, Impulsivity and Sluggish Cognitive Tempo) were entered in step-wise backward entry logistic regression model. Assessment of model fit was done by examining the change in the maximum likelihood statistic when the predictors were added to the model (i.e., examination of the model chi-square statistic). This showed that the addition of the four subscales did not significantly improve model fit over the model when only the constant was included, although the chi-square value did approach significance (initial -2 log likelihood = 243.608; Goodness of fit = 240.037; $\chi 2$ (1) =

3.571, $p = 0.059$). Interestingly, the Hosmer-Lemeshow test produced a non-significant chi-square value ($\chi 2$ (8) = 2.913, $p = 0.940$), indicating an improvement in the model, as a good model produces a non-significant chi-square value (Tabachnick & Fidell, 2007, p. 459). With regards to the unique contribution of each predictor, none of the variables were identified as making a significant contribution to the prediction of pubertal timing membership, although as depicted in Table 2, the Wald statistic for the Inattention subscale total approached significance. Notably, the odds ratio is similar to those predictors that were significant. Thus, the effect size is comparable despite the non-significance. Additional measures of effect were provided with the Cox and Snell's measure ($R^2 = 0.019$) and Nagelkerke's adjusted value ($R^2 = 0.026$). It is important to note that the Cox and Snell value takes into account sample size and underestimates effect size as it cannot achieve a maximum value of one (Tabachnick & Fidell, 2007, p. 460). Finally, follow-up analysis using a step-wise forward entry logistic regression method did not produce a model.

In contrast to the non-significant finding noted above, when symptom counts for the four subscales were entered in a logistic regression analysis (stepwise backward entry), the current Inattention Symptom count score was identified as a significant predictor. Examination of the model chi-square statistic revealed an improvement in model fit with the addition of this variable (initial -2 log likelihood=243.608; Goodness of fit=237.989; $\chi 2$ (1) = 5.619, $p = 0.018$), and the Hosmer-Lemeshow test produced a non-significant chi-square value ($\chi 2$ (2) = 1.403, $p = 0.496$). Inspection of the measures of effect sizes, Cox and Snell ($R^2 = 0.029$) and Nagelkerke's ($R^2 = 0.040$) values suggested stronger effects. Further, entering the variables as symptom counts instead of

totals improved classification accuracy to 68.1%. Table 3 includes regression coefficients in the final model.

As depicted in Table 3, the odds of correctly predicting group membership are increased by 27.0% with the addition of the Inattention Symptom count to the model. In short, as the inattentive symptom count increases, the more likely one will have had an earlier pubertal onset.

Barkley Deficits in Executive Functioning Scale. As was done for BAARS-IV scales, the five BDEFS subscale totals (i.e., Self-Management to Time, Self-Organization/Problem Solving, Self-Restraint, Self-Motivation, and Self-Regulation of Emotion) were simultaneously entered in the logistic regression analysis, using the backward step-wise method. A significant model chi-square statistic (initial -2 log likelihood = 243.608; Goodness of fit = 237.928; $\chi 2$ (1) = 5.680, p = 0.017) revealed an improvement in model fit with the addition of the Self-Regulation of Emotion subscale total to the model. Further, the Hosmer-Lemeshow test produced a non-significant finding ($\chi 2$ (8) = 6.581, p = 0.582) indicating an improvement in the model (Tabachnick & Fidell, 2007, p. 459). With the inclusion of the Self-Regulation of Emotion subscale total the model classified 66.5% of participants correctly. Table 4 includes regression coefficients in the final model.

As illustrated in Table 4, as the variable Self-Regulation of Emotion score increases by one, participants are 1.053 times more likely to be classified in the early pubertal onset group. That is, the odds of correctly predicting classification increased by 5.3%. Cox and Snell's measure (R^2=0.029) and Nagelkerke's adjusted value (R^2=0.041) provide measures of effect size. Follow-up analysis using step-wise forward entry method

confirmed that Self-Regulation of Emotion subscale score is making a significant contribution to the prediction of the timing of pubertal onset.

The BDEFS form also allows for the calculation of an executive-function (EF) symptom count that is a measure of the number of items that were answered with a sufficiently rare response suggesting the presence of clinical symptoms of an executive functioning deficit. Unlike the BAARS-IV scales, the BDEFS does not have symptom counts for the individual subscales. As such, only the EF symptom count was entered as a continuous variable in the logistic regression analysis (step-wise: backward entry). Assessment of model fit after the addition of this variable suggested an improvement in model fit (initial -2 log likelihood=243.608; Goodness of fit=238.941; $\chi 2$ (1) = 4.667, p = 0.031). Further, as shown in Table 5, the variable was identified as a significant predictor, although its odds ratio was relatively small. With the addition of this variable the accuracy of the classification improved to 67%, with the odds of correctly predicting a participant's timing of pubertal onset increased by 2.6%. This suggests that a participant is more likely to have had an early pubertal onset if they reported having a higher EF symptom count.

Difficulties in Emotion Regulation Scale. The DERS yields a total score and six subscale scores: (1) Nonacceptance of emotional responses (Nonacceptance), (2) Difficulties engaging in goal directed behaviour (Goals), (3) Impulse control difficulties (Impulse), (4) Lack of emotional awareness (Awareness), (5) Limited access to emotion regulation strategies (Strategies), (6) Lack of emotional clarity (Clarity). The six subscale totals were entered in the step-wise logistic regression analysis using the backward entry method. Examination of model fit revealed a significant model chi-square statistic (initial

-2 log likelihood = 243.608; Goodness of fit = 239.251; $\chi 2$ (1) = 4.357, p = 0.037) indicating an improvement in model fit with the addition of the Impulse subscale total to the model. Relatedly, the non-significant Hosmer-Lemeshow test ($\chi 2$ (7) = 7.867, p = 0.344) also suggested an improvement in the model (Tabachnick & Fidell, 2007, pp. 459). With the inclusion of the Impulse subscale total the model classified 66.5% of participants correctly. As depicted in Table 6, the odds ratio for the Impulse subscale is 1.070, indicated that the odds of correctly predicting classification increases by 7.0% with the addition of this variable. In other words, as the Impulse subscale score increases by one, participants are 1.070 times more likely to be classified in the early pubertal onset group. Cox and Snell's measure (R^2=0.023) and Nagelkerke's adjusted value (R^2=0.031) provide measures of effect size. Again, follow-up analysis using a step-wise forward entry method also identified the Impulse subscale as making a significant contribution to improving model fit of the data. Table 6 includes regression coefficients in the final model.

All Total Scores. The total scores from all six questionnaires were entered in the step-wise logistic regression analysis using the backward entry method. Examination of model fit revealed a significant model chi-square statistic (initial -2 log likelihood = 239.192; Goodness of fit = 231.491; $\chi 2$ (2) = 7.701, p = 0.021) indicating an improvement in model fit. Further suggesting an improvement in the model with the addition of the predictors is the non-significant Hosmer-Lemeshow test finding ($\chi 2$ (8) = 12.305, p = 0.138; Tabachnick & Fidell, 2007, p. 459). In fact, with the inclusion of the Barkley's Functional Impairment Scale total and the Risk Taking Behaviour Questionnaire total scores the overall accuracy of the classification improved to 67.7%.

Table 7 includes regression coefficients in the final model.

As displayed in Table 7, using the backward-entry method neither predictor left in the final model had a significant Wald statistic, although both approached significance. This is especially the case for the BFIS Total score as it had an odds ratio of 1.210, indicating that the odds of correctly predicting classification increases by 21% with the addition of this variable. Notably, when the same set of predictors were entered in a forward step-wise entry method, the BFIS total score was identified as making a significant contribution to the prediction of group membership. That is, an individual's score on the BFIS significantly predicted whether they were classified in the early or on-time pubertal onset group (see Table 8).

As indicated by the odds ratio displayed in Table 8, as the BFIS increases by one unit, participants are 1.239 times more likely to be classified in the early pubertal onset group. Thus, the odds of correctly predicting classification increased by 23.9%.

For three of the six questionnaires used in the study it was possible calculate symptom counts: BAARS-IV Childhood symptom count, BAARS-IV Current symptom count and BDEFS-EF symptom count. These were entered in the step-wise logistic regression analysis using the backward entry method. Examination of model fit following the addition of these variables revealed an improvement in model fit as illustrated by a significant model chi-square statistic (initial -2 log likelihood = 243.608; Goodness of fit = 237.223, $\chi 2$ (1) = 6.385, p = 0.012). Estimates of effect size were provided with the Cox and Snell's measure (R^2 = 0.033) and Nagelkerke's adjusted value (R^2 = 0.046). Further, inspection of the classification table revealed that the adjusted model was able to correctly classify 67.5% of participants. Table 9 includes regression coefficients in the

final model.

As shown in Table 9, the odds ratio for the BAARS-IV current total symptom count is 1.182, indicated that the odds of correctly predicting classification increases by 18.2% with the addition of this variable. That is, as the number of BAARS-IV current symptoms increases by one, participants are 1.182 times more likely to be classified in the early pubertal onset group than the on-time pubertal onset group. Follow-up analysis using a step-wise forward entry method confirmed this finding.

CHAPTER V

Discussion

Review of Research Question

The aim of the current study was to investigate how the timing of female pubertal maturation is associated with the extent of ADHD symptoms and related impairments in a non-clinical female emerging adult sample. Despite the growing evidence that circulating sex hormones during puberty may help explain the subtle sex differences that exist in the symptom profile, neuropathology and clinical sequelae of ADHD, there is limited research in this area. Consequently, this work was primarily an exploratory study, with minimal past research to guide hypothesis generation. That said, it was predicted that early pubertal onset would be associated with higher levels of ADHD symptoms, difficulties in emotion regulation, impairments in daily functioning, and risk-taking behaviour.

Overview of Research Findings

Using logistic regression models, significant contributory factors were identified to classify those who reported having an earlier pubertal onset relative to their peers. Overall, the present findings suggest that early puberty is associated with more current symptoms of ADHD and greater impairment on a variety of ADHD-related factors. These factors may be categorized in four domains: (1) deficits in attention, (2) difficulties in emotion regulation, (3) psychosocial impairment, and (4) risky behaviour. As may be predicted from existing literature suggesting that ADHD females present with more inattentive rather than hyperactive/impulsive symptoms (Gaube & Carlson, 1997; Nussbaum, 2011), early pubertal timing in this non-clinical female sample was not

associated with current levels of hyperactivity and impulsivity, nor with self-regulation difficulties within domains such as self-restraint and self-motivation.

General Discussion

Deficits in attention. The present findings suggest that pubertal onset is associated with current level of inattentive symptoms, such as having difficulties in sustaining attention, difficulty following instructions, experiencing frequent forgetfulness in daily activities, and losing things. Specifically, as the count of current inattentive symptoms increases, the odds of an individual reporting they matured earlier than their peers increase. This finding provides empirical support for the influence of pubertal timing on deficits in inattention, and is consistent with the hypothesis that the rise in inattention symptoms may be the consequence of the changes in the hormonal milieu during puberty; a developmental trajectory that may be unique to females (Nussbaum, 2012). This may be especially important if considered in light of the fact that many adult females who exhibit impairments in attention are often misdiagnosed as having other psychopathology (e.g., dysthymia when inattentive symptoms are present alongside low levels of arousal; Nadeau & Quinn, 2002a, 2002b as cited in Nussbaum, 2012; Wender, Wolf & Wasserstein, 2001), and comorbidities for which a role of puberty onset has been suggested, such as Major Depressive Disorder (Martel et al., 2009).

Difficulties in emotion regulation. Exploration of the association between the timing of pubertal onset and deficits in emotion regulation revealed a significant contribution of emotion regulation factors in predicting pubertal onset group membership. Specifically, as individuals reported greater deficits in self-regulation of emotion, by endorsing items such as “Overreact emotionally”, “Having trouble calming

myself down once I am emotionally upset", and "Unable to manage my emotions in order to accomplish my goals successfully or get along well with others", the more likely they would be to be classified in the early pubertal onset group. Similarly, reporting more impulse control difficulties (e.g., disclosing having difficulty controlling behaviours and emotions when upset) was associated with higher odds of having matured earlier relative to other females. Again, in line with these findings, affect lability and emotional impulsivity have been implicated in ADHD (Barkley, 2009; Barkley & Murphy, 2010; Sobanski et al., 2010), and emotional regulation difficulties have been linked to the rise in internalizing symptoms in girls with ADHD post-puberty (Lee & Hinshaw, 2006). These findings help highlight the importance of considering emotional impulsivity and poor emotional self-regulation as central features of ADHD in females. There continues to be a need for further studies examining deficits in emotional regulation in an ADHD sample (Surman et al., 2013).

Psychosocial impairment. Deficits in psychosocial functioning are a hallmark of many mental disorders, including ADHD (Biederman et al., 1993). Correspondingly, those who reported higher levels of psychosocial impairment across several domains were more likely to experience puberty earlier than their age-mates. Graber and colleagues (2004) demonstrate a similar finding, showing that girls with earlier pubertal onset report having poorer adjustment than girls who met pubertal milestones 'on-time'. Specifically, they reported difficulties with the quality of their interpersonal relationships, having smaller social support networks and lower life satisfaction. This is interesting given that there is overwhelming evidence that both children and adults with ADHD show impairments in numerous areas, such as school/work and in interpersonal

relationships (Barkley et al., 2002); a finding also noted for females with ADHD (Biederman et al., 1999). Specifically, Biederman and colleagues (1999) report impaired scores in global, academic and family functioning for girls with ADHD. Further, this is in line with the finding from the present study, showing a significant positive correlation between the total current number of ADHD symptoms ($r = 0.452$, $p < 0.001$) and mean functional impairment. The under-identification of females with ADHD means that many girls with considerable functional difficulties will continue to have their difficulties unacknowledged and untreated. This finding supports the need for clinicians to pay close attention to impairments in several domains of psychosocial functioning, especially in girls who begin to mature earlier than others.

Risky behaviour. In line with previous reports noting the negative consequences of early puberty onset on adolescent risk-taking behaviour (Downing & Bellis, 2009), early pubertal onset were associated with greater report of risk-taking behaviour in this sample of young adults. This finding corroborates previous cross-sectional and longitudinal studies that have linked early maturation with higher prevalence of risky behaviour, including unhealthy substance use and risky sexual behaviour (Downing & Bellis, 2009; Witt, 2007). For instance, Biehl and colleagues (2006) report an association between early pubertal maturation and higher alcohol use and heavy drinking in late adolescence and young adulthood. In contrast, a study exploring the relationship between the age at menarche with current smoking, and heavy use of alcohol and other drugs in the past year among 14-15 year-old Canadian girls sampled from the National Longitudinal Survey of Children and Youth, revealed no association (Al-Sahab, Ardern, Hamadeh, & Tamim, 2012). Interestingly, Wichstrom (2001) reports the use of the PDS

over other measures of pubertal timing reveals higher correlations between pubertal timing and adolescent substance use. The present study adds to the current literature by providing evidence to suggest that early pubertal timing is also associated with risky behaviour in domains that have been explored to a lesser extent, such as risky driving behaviour or rule breaking. It has been argued that elevations in hormone secretion at puberty alter reward circuits during adolescence, influencing reward seeking behaviour, such as alcohol consumption (Witt, 2007). Reciprocally, drinking behaviour during post-pubertal development in turn influences brain development, including the effects of sex hormones, via mechanisms not yet understood (Witt, 2007). It is important to note, however, that the relationship between pubertal timing and subsequent substance use has been explained with reference to other hypotheses, such as the Deviance Hypothesis (Petersen & Taylor, 1980, as cited in Wichstrom, 2001).

Interpreting Current Findings Within a Neurobiological Conceptual Framework.

It has been argued that, in contrast to reports that ADHD is more common in males than in females during childhood, there is less of a discrepancy in adult ADHD prevalence rates between males and females (Nussbaum, 2012). This increase in representation of females with ADHD in adolescence and adulthood suggests a potential organizational influence of sex hormone exposure during puberty (Nussbaum, 2012). In turn, these organizational effects of sex hormones on psychological symptoms and behaviour, which are dependent on the timing of puberty onset, should be observable post-puberty (Sisk & Zehr, 2005; Zehr et al., 2007). This assertion is supported by the current findings suggesting that alterations in brain development as a consequence of earlier hormonal exposure during adolescence are associated with ADHD related

symptomology and psychosocial functioning in emerging adulthood. Relatedly, as an individual reports an increasing number of clinical symptoms of an executive functioning (EF) deficit, the odds of them having an earlier pubertal onset increase. "Hormone-dependent remodeling" of neural circuitry during puberty has been suggested as the potential explanatory mechanism (Sisk & Zehr, 2005; Zehr et al., 2007).

As described above, deficits in the prefrontal cortex and/or neural circuits linking the prefrontal cortex to subcortical regions have been implicated in ADHD (Biederman, 2005; Nigg & Casey, 2005). Notably, these brain regions are associated with catecholamine neurotransmitter systems (e.g., dopamine) that have also been linked to ADHD and have been the target of stimulant medication (Biederman, 2005; Halperin & Shulz, 2006). Increase in circulating sex hormones, specifically estrogen, is thought to induce an increase in the number of dopamine receptors (Fink, Rosie, Grace, & Quinn, 1996 as cited in Nussbaum, 2012). This increase in dopamine receptors with puberty is hypothesized to at least partly explain the increase in symptoms post-pubertal adolescents and young adults (Nussbaum, 2012). In animal models, male rats show an increase in dopamine receptors pre- and post-puberty, and approximately half-fold decrease by adulthood, whereas females rats do not display this pattern of over-production and elimination of dopamine receptors (Andersen & Teicher, 2000). Andersen & Teicher (2000) suggest that the lack of pre-programed elimination of dopamine receptors in the striatum in ADHD females may explain why there is persistence of problems into adulthood. Further, they hypothesize that delayed pruning of dopamine receptors in the pre-frontal cortex, may explain why motoric symptoms of ADHD tend to diminish post-puberty, whereas attentional difficulties persist (Andersen & Teicher, 2000). This has

been suggested as an explanation for the gender differences in the timing of symptom presentation and clinical sequelae. Importantly, these neural circuits develop into early adulthood, and it is likely that sex hormones influence the structural and neurochemical development in many other complementary ways (i.e., influencing synaptogenesis, dendritic elaboration, and myelination), via mechanisms not yet completely understood (Sisk & Zehr, 2005).

In applying this explanation for the present findings, it is important to remember that early puberty onset needs to be considered within the context of family history, racial and/or ethnic differences, pre-pubertal body mass index (BMI), geographic and meteorological context, social environment, and medical history (Posner, 2006; Wales, 2011). That is, other factors may mediate or moderate the observed relationship between the timing of puberty and ADHD-related symptoms and behavioural impairments in adulthood. In fact, when current BMI, which is usually associated with pre-pubertal BMI (Must et al., 2005), was entered into each logistic regression model, the effect size for each model increased while the individual predictors listed above remained significant.

Although a hormonal mechanism may play a part in explaining both the current findings and gender differences in ADHD symptom onset and presentation, alternative explanations are also worth noting. For instance, reports of higher adult ADHD prevalence rates among females may be a reflection of poor symptom recognition among girls by parents and teachers and the inadequacy of current diagnostic criteria in identifying females with ADHD (Nussbaum, 2012). Other neurobiological mechanisms, independent of hormonal influences, may also be responsible, such as neurogenesis and synaptogenesis. Goldstein and Gordon (2003; as cited in Nussbaum, 2012) write that this

increase in prevalence post-puberty may be suggestive of a "less severe form of the disorder in younger females". Further, it is important to remember that the changing social norms and expectations accompanying pubertal onset may also play a role in the current presentation of ADHD symptoms and related impairments. It has been argued that early maturing girls have fewer resources to successfully adjust to the changes accompanying puberty than later maturing girls, and as a consequence are more likely to experience emotional and behavioural problems (Mendle, Turkheimer, & Emery, 2007). For instance, early maturing girls are more susceptible to early substance use (Bratberg, Nilsen, Holmen, & Vatten, 2005; Wiesner & Ittel, 2002), sexual behaviour (Flannery, Rowe, & Gulley, 1993), delinquency (Mendle et al., 2007), In turn, these experiences may affect current symptom presentation via their direct or indirect impact on the development of the nervous system post-puberty.

Limitations of the Present Study

The findings from the present study are limited by several factors. First, by using a non-clinical university sample, one is limited in the conclusions that are warranted with regards to the nature of ADHD in females and in the generalizability of the findings. Notably, however, use of only clinic-referred ADHD females may also not be justified given that it has been argued that clinic-referred female ADHD samples may not be representative of the typical manifestation of ADHD in females (Gaub & Carlson, 1997). Thus, future studies should aim to capture as large of a community sample of females with ADHD as possible. Further limiting generalizability, the sample in the current study did not have an equal representation of all race/ethnic groups to allow for an accurate evaluation of any racial/ethnic differences that have been associated with pubertal timing,

but the breakdown accurately represented the local population. Second, it may be argued that the use of a retrospective, self-report measure does limit the accuracy of the conclusions that can be made. Yet, there is evidence to suggest that retrospective estimates of pubertal timing are relatively accurate (Dubas, Graber, & Petersen, 1991). Relatedly, while collateral information was not collected to corroborate self-report of symptoms, Barkley, Knouse and Murphy (2011) report moderate to high agreement between adult self-report and others-report with regards to current symptoms and impairment. Third, the use of a cross-sectional design does not warrant causational conclusions regarding the effect of pubertal timing on ADHD symptoms in emerging adulthood. Finally, the choice of using logistic regression analysis in this study may be methodologically confusing given that one is retrospectively predicting from current symptoms. It is worth noting, however, that other studies examining factors predictive of early puberty have utilized this technique (Downing & Bellis, 2009).

Unique Contributions of the Present Study

There are several strengths of the present research worth noting. First, the literature is limited with studies exploring the relationship between pubertal timing and ADHD related symptoms and impairments later in life. In fact, the author is unaware of any similar studies that have been conducted within the Canadian context. Curbing this gap is important in addressing an overlooked public health concern (i.e., the lack of clinical and research attention paid to symptom presentation and functional difficulties of females with ADHD). Second, as per the expert recommendations (personal communication with Russell Barkley, October 2011), the study takes advantage of self-report behaviour rating scales that have been suggested to be more sensitive in capturing

female-specific difficulties (Mahone, 2010). Similarly, the measure of pubertal timing used in the study incorporates multiple developmental aspects of pubertal status, and not just age at first menarche, providing a more comprehensive estimation of pubertal onset. Third, although logistic regression analysis is commonly used in cross-sectional epidemiological studies, many fewer studies in the social sciences take advantage of this technique. This statistical method proved to be an appropriate technique in this circumstance when the violation of several statistical assumptions made the use of other techniques unjustified.

Clinical Implications

The findings from the current study lend support for the argument that there is a need for further research examining gender differences in ADHD across the lifespan. Notably, although it does not speak directly to the need for modifying diagnostic thresholds, or using gender-specific diagnostic criteria to address potential gender differences, it does highlight the potential influence of sex hormones in symptom presentation and clinical sequalae of ADHD. This research is especially warranted when considered in light of the fact that relative to males with ADHD, adult females with ADHD report having a greater number of problems, yet fewer assets (Arcia & Conners, 1998, as cited in Rasmussen & Levander, 2009). Further, exploration of the impact of early pubertal onset on cognitive and psychosocial function is justified given that there is a trend toward girls beginning puberty at earlier ages (Al-Sahab, Ardern, Hamadeh, & Tamim, 2010; Gluckman & Hanson, 2006; Tanner, 1991, as cited in Posner, 2006). By improving our current understanding of the presentation of ADHD in females, we might

be better able to identify and improve quality of care given to females with ADHD, and thus potentially aiding in the establishment of sex specific interventions.

Conclusion and Future Directions

The present findings do not provide evidence for a causational relationship between pubertal timing and ADHD symptoms in emerging adulthood. They do, however, provide preliminary evidence for a relationship between these two variables. That is, hormonal-dependent organizational influences during puberty seem to be associated with ADHD symptoms and behaviours later in life, with earlier pubertal onset linked to more deficits in attention, emotion regulation, and psychosocial functioning, and greater reports of risky behaviour. It should be noted, however, that none of these symptoms are specific to ADHD, and many individuals with other psychopathology will present with some subset of these symptoms and impairment. Nevertheless, these findings provide support for a potential organizational effect of sex hormones during pubertal development.

The study sheds light on the impact of early puberty and its findings help pave the way for future research. Given that this is an unexplored research area, the possibilities for future studies are numerous. This includes conducting a longitudinal study examining symptom presentation across development, with special attention given to girls who experience precocious puberty. This would not only overcome the limitations of a cross-sectional design, but would also alleviate any limitations that may be due to the use of retrospective measures of pubertal onset. Relatedly, it is important to remember that organizational effects of sex hormones are not necessarily exclusive or independent of the potential transient activational effects of sex steroids. As such, future work should

examine symptom presentation during pubertal development, allowing for a better understanding of both short-term and long-term effects of pubertal timing.

Despite the fact that ADHD symptoms reflect extremes on the developmental continuum allowing for the investigation of ADHD symptoms in a nonclinical sample, use of a clinical sample would more directly address the research question. This would require access to a community sample of females with ADHD, which has been noted to be a difficult population to recruit. Relatedly, it would be interesting to examine if there are any subtype differences with regards to the influence of sex hormones during puberty.

Future studies could also add to the growing literature by examining or controlling for the influence of factors known to impact pubertal timing, such pre-pubertal BMI, socioeconomic status, and number of childhood illnesses (Downing & Bellis, 2009). These factors may mediate or moderate the identified relationship. Further, future studies should examine the interplay between hormonal influence and growing social demands on symptom presentation in emerging adulthood. It would also be interesting to further explore how the identified deficits in attention and emotion regulation in emerging adulthood contribute to the reported psychosocial functional impairments and higher risk taking behaviour in females with ADHD.

Overall, the present research aids in the understanding of the impact of differential pubertal timing and the role hormonal exposure during puberty may play in explaining the differences in gender prevalence rates of ADHD and symptom profiles. Future studies are needed to further examine the influence that elevations in sex hormones during puberty may have in increasing ADHD symptom presentation, and how and to what extent these effects are permanent and observable in adulthood.

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Table 1

Mean, Standard Deviation and Range for Each Questionnaire by Pubertal Onset Group

Questionnaire	Pubertal Onset Group	Mean	Standard Deviation	Range
PDS-RV (maximum score = 30)	Early	12.21	1.966	6.00 – 14.8
	On-time	17.22	1.314	15.0 – 19.2
	Late	22.74	2.156	19.9 – 29.0
	All Participants	17.26	4.084	6.00 – 29.0
BAARS-IV Current Symptoms Total Score (maximum score = 108)	Early	28.28	6.846	19.0 – 50.0
	On-time	26.37	5.860	18.0 – 51.0
	Late	27.95	7.229	18.0 – 49.0
	All Participants	27.24	6.498	9.00 – 31.0
BAARS-IV Current Inattention Subscale Score (maximum score = 36)	Early	14.83	4.410	9.00 – 31.0
	On-time	13.69	3.574	9.00 – 26.0
	Late	14.27	4.313	9.00 – 31.0
	All Participants	14.12	3.995	9.00 – 31.0
BAARS-IV Current Hyperactivity Subscale Score (maximum score = 20)	Early	7.672	2.055	5.00 – 14.0
	On-time	7.080	2.062	5.00 – 14.0
	Late	7.733	2.503	5.00 – 17.0
	All Participants	7.387	2.187	5.00 – 17.0
BAARS-IV Current Impulsivity Subscale Score (maximum score = 16)	Early	5.781	2.027	4.00 – 12.0
	On-time	5.606	1.691	4.00 – 14.0
	Late	5.950	2.086	4.00 – 14.0
	All Participants	5.730	1.877	4.00 – 14.0
BAARS-IV Current Sluggish Cognitive Tempo Subscale Score (maximum score = 36)	Early	17.44	5.363	9.00 – 32.0
	On-time	16.72	4.547	9.00 – 30.0
	Late	17.23	5.261	9.00 – 33.0
	All Participants	17.03	4.929	9.00 – 33.0
BDEFS Total Score (maximum score = 356)	Early	154.84	38.17	97.9 – 268.0
	On-time	145.10	32.41	91.0 – 273.0
	Late	150.61	34.83	94.0 – 214.9
	All Participants	148.90	34.64	91.0 – 273.0
BDEFS Self-Management to Time Subscale Score (maximum score = 84)	Early	40.52	12.89	21.0 – 74.0
	On-time	37.88	10.63	21.0 – 72.0
	Late	40.77	11.39	24.0 – 68.0
	All Participants	39.08	11.60	21.0 – 74.0
BDEFS Self-Organization/Problem Solving Subscale Score (maximum score = 96)	Early	41.69	13.59	25.0 – 94.0
	On-time	40.04	11.32	24.0 – 81.0
	Late	40.77	11.39	24.0 – 68.0
	All Participants	40.64	11.92	24.0 – 94.0
BDEFS Self-Restraint Subscale Score (maximum score = 76)	Early	30.29	7.128	19.0 – 50.0
	On-time	28.43	6.300	19.0 – 48.0
	Late	28.53	6.749	19.0 – 48.0
	All Participants	28.93	6.648	19.0 – 50.0

BDEFS Self-Motivation Subscale Score (maximum score = 48)	Early	18.23	5.349	11.9 – 39.0
	On-time	17.23	4.694	12.0 – 37.0
	Late	17.68	4.070	12.0 – 28.0
	All Participants	17.59	4.733	12.0 – 39.0
BDEFS Self-Regulation of Emotion Subscale Score (maximum score = 52)	Early	24.11	8.169	13.0 – 47.0
	On-time	21.52	6.280	13.0 – 43.4
	Late	23.53	7.761	13.0 – 42.0
	All Participants	22.66	7.232	13.0 – 47.0
DERS Total Score (maximum score = 180)	Early	85.02	22.86	46.0 – 153.0
	On-time	79.13	20.35	39.0 – 146.0
	Late	84.05	23.94	41.0 – 142.0
	All Participants	81.80	21.98	39.0 – 153.0
DERS Nonacceptance of Emotional Responses Subscale Score (maximum score = 30)	Early	13.25	5.798	6.00 – 30.0
	On-time	13.06	5.269	6.00 – 29.0
	Late	13.80	5.967	5.06 – 30.0
	All Participants	13.29	5.564	5.06 – 30.0
DERS Difficulties Engaging in Goal-Directed Behaviour Subscale Score (maximum score = 25)	Early	16.22	4.971	5.00 – 25.0
	On-time	15.49	5.315	5.00 – 25.0
	Late	16.08	4.906	6.00 – 25.0
	All Participants	15.82	5.125	5.00 – 25.0
DERS Impulse Control Difficulties Subscale Score (maximum score = 30)	Early	12.00	5.401	6.00 – 28.0
	On-time	10.50	4.150	6.00 – 28.0
	Late	11.60	5.578	6.00 – 28.0
	All Participants	11.14	4.881	6.00 – 28.0
DERS Lack of Emotional Awareness Subscale Score (maximum score = 30)	Early	14.45	4.731	6.00 – 25.0
	On-time	13.26	4.405	6.00 – 30.0
	Late	13.81	4.882	6.00 – 26.0
	All Participants	13.70	4.614	6.00 – 30.0
DERS Limited Access to Emotion Regulation Strategies Subscale Score (maximum score = 40)	Early	18.38	7.704	8.00 – 36.0
	On-time	16.57	6.286	8.00 – 35.0
	Late	17.81	6.664	8.00 – 31.0
	All Participants	17.33	6.781	8.00 – 36.0
DERS Lack of Emotional Clarity Subscale Score (maximum score = 25)	Early	10.72	3.627	6.00 – 21.0
	On-time	10.24	3.394	5.00 – 19.0
	Late	10.94	3.880	5.00 – 25.0
	All Participants	10.53	3.573	5.00 – 25.0
BFIS Mean Impairment Score (maximum score = 9)	Early	2.408	1.867	0.00 – 6.90
	On-time	1.860	1.393	0.00 – 7.08
	Late	2.158	1.589	0.00 – 7.69
	All Participants	2.071	1.583	0.00 – 7.69
RTBQ Total Score (maximum score = 99)	Early	16.61	9.757	1.00 – 42.0
	On-time	13.76	8.635	0.00 – 42.0
	Late	14.39	9.386	0.00 – 42.0
	All Participants	14.63	9.144	0.00 – 44.0

Table 2

Regression Coefficients for Model Variables: BAARS-IV Current Symptoms

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
Inattention Score	0.073 (.039)	3.541	1	0.060	0.997	1.076	1.162
Constant	-1.729 (.580)	8.871	1	0.003			

Note. R^2 = 0.940 (Hosmer & Lemeshow), 0.019 (Cox & Snell), 0.026 (Nagelkerke).

Table 3

Regression Coefficients for Model Variables: BAARS-IV Current Symptom Counts

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
Inattention Symptom Count	0.239 (.102)	5.492	1	0.019	1.040	1.270	1.552
Constant	-0.911 (.185)	24.309	1	0.000			

Note. R^2 = 0.496 (Hosmer & Lemeshow), 0.029 (Cox & Snell), 0.040 (Nagelkerke).

Table 4

Regression Coefficients for Model Variables: Barkley's Deficits in Executive Functioning Scale

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
Self-Regulation of Emotion Score	0.051 (.022)	5.569	1	0.018	1.009	1.053	1.099
Constant	-1.856 (.526)	12.448	1	0.000			

Note. R^2 = 0.091 (Hosmer & Lemeshow), 0.026 (Cox & Snell), 0.036 (Nagelkerke).

Table 5

Regression Coefficients for Model Variables: Barkley's Deficits in Executive Functioning Scale – Symptom Count

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
Executive Function Symptom Count	0.025 (.012)	4.602	1	0.032	1.002	1.026	1.050
Constant	-1.013 (.221)	21.038	1	0.000			

Note. R^2 = 0.163 (Hosmer & Lemeshow), 0.024 (Cox & Snell), 0.033 (Nagelkerke).

Table 6

Regression Coefficients for Model Variables: Difficulties in Emotion Regulation Scale

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
Impulse Score	0.068 (.033)	4.313	1	0.038	1.004	1.070	1.141
Constant	-1.444 (.402)	12.939	1	0.000			

Note. R^2 = 0.344 (Hosmer & Lemeshow), 0.023 (Cox & Snell), 0.031 (Nagelkerke).

Table 7

Regression Coefficients for Model Variables: Total Scores (Backward entry)

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
BFIS Total Score	0.191 (.100)	3.657	1	0.056	0.995	1.210	1.472
RTBQ Total Score	0.029 (.017)	2.852	1	0.091	0.995	1.030	1.065
Constant	-1.565 (.362)	18.711	1	0.000			

Note. $R^2 = 0.138$ (Hosmer & Lemeshow), 0.040 (Cox & Snell), 0.056 (Nagelkerke).

Table 8

Regression Coefficients for Model Variables: Total Scores (Forward entry)

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
BFIS Total Score	0.214 (.098)	4.776	1	0.026	1.022	1.239	1.502
Constant	-1.171 (.265)	19.459	1	0.000			

Note. R^2 = 0.568 (Hosmer & Lemeshow), 0.025 (Cox & Snell), 0.035 (Nagelkerke).

Table 9

Regression Coefficients for Model Variables: Total Scores – Symptom Counts

	B (SE)	Wald	*df*	*P*	95% CI for Odds Ratio		
					Lower	Odds Ratio	Upper
BAARS-IV Current Total Symptom Count	0.167 (.068)	6.006	1	0.014	1.034	1.182	1.351
Constant	-0.984 (.199)	24.339	1	0.000			

Note. R^2 = 0.058 (Hosmer & Lemeshow), 0.033 (Cox & Snell), 0.046 (Nagelkerke).

APPENDICES

APPENDIX A

Demographic Information

Participant's Name: _____

Participant's Student Number: _____ Current Year: _____ Major: _____

Home Address: _____

Home Phone: _____

Cell Phone: _____

Email: _____

Name/phone number for another person who will know how to find you if we cannot reach you:

_____

May we contact you again in the future for other studies? YES _____ NO _____

Instructions: The items in this questionnaire address issues pertaining to your personal identity, medical history, and family background. For questions that include numbered choice options, please circle the number(s) that best describes your answer. Other items will provide you with space(s) to provide a written response. Be sure to read each item carefully, and direct any questions to a member of the research staff. Please try to answer each item as best you can, however, if you feel uncomfortable with any question, you do not need to answer it. Please know that your answers will be kept completely confidential. ***Please do not write your name on any page but this front page. (This cover page will be detached and stored with your consent forms to protect your confidentiality.)***

(FOR PROJECT USE ONLY – ID # _____)

(PROJECT USE ONLY – ID # _____)

1. DEMOGRAPHIC INFORMATION

Date of Birth (MM/YY): ___/___ Today's Date (DD/MM/YY): ___/___/___
Age _____

Race/ethnic background: (*please circle*)

[1] ABORIGINAL

[2] ASIAN OR ASIAN DESCENT

[3] HISPANIC/LATINO

[4] NON-HISPANIC BLACK OR AFRICAN DESCENT

[5] NON-HISPANIC WHITE OR CAUCASIAN

[6] OTHER/MIXED (please describe) _____

[7] PREFER NOT TO ANSWER

Marital status: (*please circle*)

[1] SINGLE

[2] IN A ROMANTIC RELATIONSHIP

[3] MARRIED/CIVIL UNION/COHABITING

[4] WIDOWED

[5] PREFER NOT TO ANSWER

Body Weight: _____ (POUNDS (lb) / KILOGRAMS (kg))
(*please circle one*)
Height: _____

Days since last menstrual periods: _____ days
Typical length of menstrual period: _____ days

Are you currently pregnant? (*please circle*) [1] NO [2] YES
Is there a chance you could be pregnant? (*please circle*) [1] NO [2] YES

Do you write with your right or left hand? (*please circle*)
[1] RIGHT [2] LEFT [3] BOTH

Are you employed? (*please circle*) [1] NO [2] YES

IF YES, what is your job title _____

(PROJECT USE ONLY – ID # _____)

2. HOUSEHOLD INFORMATION/FAMILY INFORMATION

Total number of household members (including you): _____

# of children (*under 18 years of age*): _____ # of adults: _____

Parent Information:

PARENT	RELATION TO YOU Please indicate if BIOLOGICAL, STEP, FOSTER, OR ADOPTIVE	AGE	OCCUPATION	HIGHEST GRADE COMPLETED

Did your mother have any miscarriages? [1] NO [2] YES

IF YES, how many _____ and the sex (*if known*): ____ MALE ___ FEMALE
(*indicate number*)

Sibling Information

SEX	AGE	RELATION TO YOU If not FULL siblings indicate relatedness: FULL, STEP, HALF, ADOPTIVE

(PROJECT USE ONLY – ID # _____)

3. MEDICAL HISTORY

Were you ever diagnosed with ADHD or did you ever take a stimulant medication, such as Ritalin? (*please circle*) [1] NO [2] YES

Are you currently taking medicine prescribed for ADHD? (*please circle*) [1] NO [2] YES

Have you ever had any kind of head injury? (*please circle*) [1] NO [2] YES

IF YES, what happened _____

IF YES, was there a loss of consciousness? (*please circle*) [1] NO [2] YES

IF YES, for how long (in hours)? _____

Have you ever had a seizure? (*please circle*) [1] NO [2] YES

IF YES, specify type:

[1] FEBRILE, specify # of times _____

[2] PETIT MAL/ABSENCE, specify # of times _____

[3] GRAND MAL/TONIC-CLONIC, specify # of times _____

[4] OTHER, please specify type _____ and # of times _____

IF YES, were you ever medicated for seizures? (*please circle*) [1] NO [2] YES

Specify when and type of medication:_____

Have you ever been diagnosed with a learning disorder? [1] NO [2] YES

IF YES, please specify what kind _____

Have you ever been diagnosed with any mental disorder? [1] NO [2] YES

IF YES, please specify what kind _____

Are you currently taking any form of medication (except as indicated above)?

[1] NO [2] YES IF YES, please specify what kind _____

4. DEVELOPMENTAL HISTORY

Has anyone ever told you that you:

Started talking late?	[1] NO	[2] YES
Crawled or walked late?	[1] NO	[2] YES
Were difficult to manage as a young child?	[1] NO	[2] YES
Were late in being toilet trained?	[1] NO	[2] YES
Had problems getting along with other children?	[1] NO	[2] YES
Were aggressive toward others?	[1] NO	[2] YES

(PROJECT USE ONLY – ID # _____)

5. ACADEMIC HISTORY

Current Year? _____ Major? _____

Current CGPA? _____

Do you like school? [1] NO [2] YES

Are you having any difficulty in school? [1] NO [2] YES

IF YES, please describe

_____

_____

_____

Do you receive any special services at school? [1] NO [2] YES

If YES, please specify

_____

_____

THANK YOU FOR TAKING THE TIME TO COMPLETE THIS QUESTIONNAIRE !!

APPENDIX B

Pubertal Development Scale (PDS) - Women

Retrospective Version
7/28/04 CS, JZ, KK

Women show a large range of ages during which pubertal growth and development occur. We are interested in learning more about this range of individual differences in pubertal development. We ask you to help us get this information by answering some questions about what you remember about your own pubertal development.

When you are answering these questions, it is important to remember that no one will see your answers other than the researchers doing this study, and that your name will not be listed with your responses (just your identification number will be). Therefore, please be as honest as possible since your honest answers will help us learn about variability in these measures.

For many questions, we will ask you to estimate the age at which an event occurred. Many people find it useful to think about other events that were occurring around the same time to help them remember their age. For example, you may remember that an event occurred in the fourth grade, but not immediately recall the age. In these cases, you may be able to estimate your age based on these other memories and the grade you were in at the time. For additional assistance throughout this questionnaire, please also use the "grade level: age" conversion chart provided below.

Grade Level: Age Conversion Chart

Grade 1: 6 years old
Grade 2: 7 years old
Grade 3: 8 years old
Grade 4: 9 years old
Grade 5: 10 years old
Grade 6: 11 years old
Grade 7: 12 years old
Grade 8: 13 years old
Grade 9: 14 years old
Grade 10: 15 years old
Grade 11: 16 years old
Grade 12: 17 years old

Please remember to read the directions and each question carefully.

Section I: In the following section, please pick the answer that best fits your developmental profile. Please choose only **ONE** answer for each question.

(1) In general, do you think your development was any earlier or later than most other girls?

1. much earlier
2. somewhat earlier
3. about the same
4. somewhat later
5. much later
6. don't know

(2) Do you think your first period was any earlier or later than most other girls?

1. much earlier
2. somewhat earlier
3. about the same
4. somewhat later
5. much later
6. don't know

(3) Do you remember the approximate age or grade at which you first menstruated?

1. yes
2. no

If you answered "yes":

(4) How old were you when you had your first period?

a) _____ years and _____ months old
b) don't know

(5) What grade were you in when you had your first period?

a) _____ grade
b) don't know

(6) Do you think your breasts developed any earlier or later than most other girls?

1. much earlier
2. somewhat earlier
3. about the same
4. somewhat later
5. much later
6. don't know

(7) Do you remember the approximate age or grade in which you first noticed changes in your breasts or breast development?

1. yes
2. no

If you answered "yes":

(8) How old were you when you first noticed breast development?
a) _____ years and _____ months old
b) don't know

(9) What grade were you in when you first noticed breast development?
a) _____ grade
b) don't know

(10) Do you remember the approximate age or grade in which you wore your first training bra?
1. yes
2. no

If you answered "yes":

(11) How old were you when you wore your first training bra?
a) _____ years and _____ months old
b) don't know

(12) What grade were you in when you wore your first training bra?
a) _____ grade
b) don't know

(13) Both girls and boys go through a growth spurt before puberty, during which time they rapidly increase in height, outgrowing their shoes and clothes. Would you say that your growth in height was any earlier or later than other girls?
1. much earlier
2. somewhat earlier
3. about the same
4. somewhat later
5. much later
6. don't know

(14) Do you remember the approximate age or grade at which you went through a growth spurt?
1. yes
2. no

If you answered "yes":
(15) How old were you when you went through a growth spurt?
a) _____ years and _____ months old
b) don't know

(16) What grade were you in when you went through a growth spurt?
   a) _____ grade
   b) don't know

(17) Body hair ("body hair" meaning underarm and pubic hair) also begins to grow during puberty. Would you say that your growth of body hair was any earlier or later than other girls?
   1. much earlier
   2. somewhat earlier
   3. about the same
   4. somewhat later
   5. much later
   6. don't know

(18) Do you remember the approximate age or grade at which you began to grow body hair?
   1. yes
   2. no

If you answered "yes":

(19) How old were you when you began to grow body hair?
   a) _____ years and _____ months old
   b) don't know

(20) What grade were you in when you began to grow body hair?
   a) _____ grade
   b) don't know

(21) Hormonal changes during puberty can dramatically change the chemistry of the facial skin, causing pimples and acne. Would you say your skin changed any earlier or later than other girls?
   1. much earlier
   2. somewhat earlier
   3. about the same
   4. somewhat later
   5. much later
   6. don't know

(22) Do you remember the approximate age and grade in which your skin started changing?
   1. yes
   2. no

If you answered "yes":

(23) How old were you when your skin started to change?
a) _____ years and _____ months old
b) don't know

(24) What grade were you in when your skin started to change?
a) _____ grade
b) don't know

(25) Birth control pills and other types of hormonal contraceptives (e.g., Depo-Provera) are prescribed by doctors prior to the age of 18 for a variety of reasons, including but not limited to acne, irregular menstrual cycles, and as a form of pregnancy prevention. Are you currently taking hormonal contraceptives?
1. yes (specify brand, if known):_____
2. no

(Did you take any of these types of hormonal contraceptives before the age of 18?
1. yes
2. no
3. don't know

If you answered "yes," please answer the following questions:

Hormonal contraceptive(s) taken:
___ birth control pills (specify brand, if known): _____
___ birth control injections (e.g. Depo-Provera, specify type if known):_____
___ other (specify): _____

Age start: I was _____ years and _____ months old when I began taking hormonal contraceptives.

Grade start: I was in the ____ grade when I began taking hormonal contraceptives.

Between the start age and the age of 18, did you take hormonal contraceptives?
3. continuously
4. stopped and started (had more than one period of use)
5. stopped after a single period of use

If you stopped after a single period of use, what age were you when you stopped taking hormonal contraceptives?

Age: I was _____ years and _____ months old when I stopped taking hormonal contraceptives.

Grade: I was in the ____ grade when I stopped taking hormonal contraceptives.

APPENDIX C

BAARS-IV: Self Report - Current

Instructions

For the first 27 items, please circle the number next to each item below that best describes your behavior **DURING THE PAST 6 MONTHS**. Then answer the remaining three questions.

	Never or rarely	Some-times	Often	Very often
1. Fail to give close attention to details or make careless mistakes in my work or other activities	1	2	3	4
2. Difficulty sustaining my attention in tasks or fun activities	1	2	3	4
3. Don't listen when spoken to directly	1	2	3	4
4. Don't follow through on instructions and fail to finish work or chores	1	2	3	4
5. Have difficulty organizing tasks and activities	1	2	3	4
6. Avoid, dislike, or am reluctant to engage in tasks that require sustained mental effort	1	2	3	4
7. Lose things necessary for tasks or activities	1	2	3	4
8. Easily distracted by extraneous stimuli or irrelevant thoughts	1	2	3	4
9. Forgetful in daily activities	1	2	3	4
10. Fidget with hands or feet or squirm in seat	1	2	3	4
11. Leave my seat in classrooms or in other situations in which remaining seated is expected	1	2	3	4
12. Shift around excessively or feel restless or hemmed in	1	2	3	4
13. Have difficulty engaging in leisure activities quietly (feel uncomfortable, or am loud or noisy)	1	2	3	4
14. I am "on the go" or act as if "driven by a motor" (or I feel like I have to be busy or always doing something)	1	2	3	4
15. Talk excessively (in social situations)	1	2	3	4
16. Blurt out answers before questions have been completed, complete others' sentences, or jump the gun	1	2	3	4
17. Have difficulty awaiting my turn	1	2	3	4
18. Interrupt or intrude on others (butt into conversations or activities without permission or take over what others are doing)	1	2	3	4

	Never or rarely	Some-times	Often	Very often
19. Prone to daydreaming when I should be concentrating on something or working	1	2	3	4
20. Have trouble staying alert or awake in boring situations	1	2	3	4
21. Easily confused	1	2	3	4
22. Easily bored	1	2	3	4
23. Spacey or "in a fog"	1	2	3	4
24. Lethargic, more tired than others	1	2	3	4
25. Underactive or have less energy than others	1	2	3	4
26. Slow moving	1	2	3	4
27. I don't seem to process information as quickly or as accurately as others	1	2	3	4

28. Did you experience any of these 27 symptoms at least "Often" or more frequently (Did you circle a 3 or a 4 above)? **No** **Yes** (*Circle one*)

29. If so, how old were you when those symptoms began? (*Fill in the blank*)
I was _____ years old.

30. If so, in which of these settings did those symptoms impair your functioning? *Place a check mark (√) next to all the areas that apply to you.*

_____ School

_____ Home

_____ Work

_____ Social Relationships

APPENDIX D

BDEFS-LS: Self Report

Instructions

How often do you experience each of these problems? Please circle the number next to each item that best describes your behavior **DURING THE PAST 6 MONTHS**.

	Never or rarely	Some-times	Often	Very often
1. Procrastinate or put off doing things until the last minute	1	2	3	4
2. Poor sense of time	1	2	3	4
3. Waste or mismanage my time	1	2	3	4
4. Not prepared on time for work or assigned tasks	1	2	3	4
5. Fail to meet deadlines for assignments	1	2	3	4
6. Have trouble planning ahead or preparing for upcoming events	1	2	3	4
7. Forget to do things I am supposed to do	1	2	3	4
8. Can't seem to accomplish the goals I set for myself	1	2	3	4
9. Late for work or scheduled appointments	1	2	3	4
10. Can't seem to hold in mind things I need to remember to do	1	2	3	4
11. Can't seem to get things done unless there is an immediate deadline	1	2	3	4
12. Have difficulty judging how much time it will take to do something or get somewhere	1	2	3	4
13. Have trouble motivating myself to start work	1	2	3	4
14. Have difficulty motivating myself to stick with my work and get it done	1	2	3	4
15. Not motivated to prepare in advance for things I know I am supposed to do	1	2	3	4
16. Have trouble completing one activity before starting into a new one	1	2	3	4
17. Have trouble doing what I tell myself to do	1	2	3	4
18. Difficulties following through on promises or commitments I may make to others	1	2	3	4
19. Lack self-discipline	1	2	3	4
20. Have difficulty arranging or doing my work by its priority or importance; can't "prioritize" well	1	2	3	4
21. Find it hard to get started or get going on things I need to get done	1	2	3	4

	Never or rarely	Some-times	Often	Very often
22. I do not seem to anticipate the future as much or as well as others	1	2	3	4
23. Can't seem to remember what I previously heard or read about	1	2	3	4
24. I have trouble organizing my thoughts	1	2	3	4
25. When I am shown something complicated to do, I cannot keep the information in mind so as to imitate or do it correctly	1	2	3	4
26. I have trouble considering various options for doing things and weighing their consequences	1	2	3	4
27. Have difficulties saying what I want to say	1	2	3	4
28. Unable to come up with or invent as many solutions to problems as others seem to do	1	2	3	4
29. Find myself at a loss for words when I want to explain something to others	1	2	3	4
30. Have trouble putting my thoughts down in writing as well or as quickly as others	1	2	3	4
31. Feel I am not as creative or inventive as others of my level of intelligence	1	2	3	4
32. In trying to accomplish goals or assignments, I find I am not able to think of as many ways of doing things as others	1	2	3	4
33. Have trouble learning new or complex activities as well as others	1	2	3	4
34. Have difficulty explaining things in their proper order or sequence	1	2	3	4
35. Can't seem to get to the point of my explanations as well as others	1	2	3	4
36. Have trouble doing things in their proper order or sequence	1	2	3	4
37. Unable to "think on my feet" or respond as effectively as others to unexpected events	1	2	3	4
38. I am slower than others at solving problems I encounter in my daily life	1	2	3	4
39. Easily distracted by irrelevant events or thoughts when I must concentrate on something	1	2	3	4
40. Not able to comprehend what I read as well as I should be able to do; have to reread material to get its meaning	1	2	3	4
41. Cannot focus my attention on tasks or work as well as others	1	2	3	4
42. Easily confused	1	2	3	4

	Never or rarely	Some-times	Often	Very often
43. Can't seem to sustain my concentration on reading, paperwork, lectures, or work	1	2	3	4
44. Find it hard to focus on what is important from what is not important when I do things	1	2	3	4
44. I don't seem to process information as quickly or as accurately as others	1	2	3	4
45. Find it difficult to tolerate waiting; impatient	1	2	3	4
46. Make decisions impulsively	1	2	3	4
47. Unable to inhibit my reactions or responses to events or others	1	2	3	4
48. Have difficulty stopping my activities or behaviour when I should do so	1	2	3	4
49. Have difficulty changing my behaviour when I am given feedback about my mistakes	1	2	3	4
50. Make impulsive comments to others	1	2	3	4
51. Likely to do things without considering the consequences for doing them	1	2	3	4
52. Change my plans at the last minute on a whim or last minute impulse	1	2	3	4
53. Fail to consider past relevant events or past personal experiences before responding to situations (I act without thinking)	1	2	3	4
54. Not aware of things I say or do	1	2	3	4
55. Have difficulty being objective about things that affect me	1	2	3	4
56. Find it hard to take other people's perspectives about a problem or situation	1	2	3	4
57. Don't think about or talk things over with myself before doing something	1	2	3	4
58. Trouble following the rules in a situation	1	2	3	4
59. More likely to drive a motor vehicle much faster than others (Excessive speeding)	1	2	3	4
60. Have a low tolerance for frustrating situations	1	2	3	4
61. Cannot inhibit my emotions as well as others	1	2	3	4
62. I don't look ahead and think about what the future outcomes will be before I do something (I don't use my foresight)	1	2	3	4
63. I engage in risk taking activities more than others are likely to do	1	2	3	4

	Never or rarely	Some-times	Often	Very often
64. Likely to take short cuts in my work and not do all I am supposed to do	1	2	3	4
65. Likely to skip out on work early if my work is boring to do	1	2	3	4
66. Do not put as much effort into my work as I should or than others are able to do	1	2	3	4
67. Others tell me I am lazy or unmotivated	1	2	3	4
68. Have to depend on others to help me get my work done	1	2	3	4
69. Things must have an immediate payoff for me or I do not seem to get them done	1	2	3	4
70. Have difficulty resisting the urge to do something fun or more interesting when I am supposed to be working	1	2	3	4
71. Inconsistent in the quality or quantity of my work performance	1	2	3	4
72. Unable to work as well as others without supervision or frequent instruction	1	2	3	4
73. I do not have the willpower or determination that others seem to have	1	2	3	4
74. I am not able to work toward longer term or delayed rewards as well as others	1	2	3	4
75. I cannot resist doing things that produce immediate rewards even if they are not good for me in the long run	1	2	3	4
76. Quick to become angry or become upset	1	2	3	4
77. Overreact emotionally	1	2	3	4
78. Easily excitable	1	2	3	4
79. Unable to inhibit showing strong negative or positive emotions	1	2	3	4
80. Have trouble calming myself down when once I am emotionally upset	1	2	3	4
81. Cannot seem to regain emotional control and become more reasonable once I am emotional	1	2	3	4
82. Cannot seem to distract myself away from whatever is upsetting me emotionally to help me calm me down. I can't refocus my mind to a more positive framework.	1	2	3	4
83. Unable to manage my emotions in order to accomplish my goals successfully or get along well with others	1	2	3	4
84. I remain emotional or upset longer than others	1	2	3	4

	Never or rarely	Some-times	Often	Very often
85. I find it difficult to walk away from emotionally upsetting encounters with others or leave situations in which I have become very emotional	1	2	3	4
86. I cannot rechannel or redirect my emotions into more positive ways or outlets when I get upset	1	2	3	4
87. I am not able to evaluate an emotionally upsetting event more objectively	1	2	3	4
88. I cannot redefine negative events into more positive viewpoints when I feel strong emotions	1	2	3	4

APPENDIX E

DERS

Please indicate how often the following statements apply to you by writing the appropriate number from the scale below on the line beside each item:

1	2	3	4	5
almost never (0-10%)	sometimes (11-35%)	about half the time (36-65%)	most of the time (66-90%)	almost always (91-100%)

_____ 1) I am clear about my feelings.
_____ 2) I pay attention to how I feel.
_____ 3) I experience my emotions as overwhelming and out of control.
_____ 4) I have no idea how I am feeling.
_____ 5) I have difficulty making sense out of my feelings.
_____ 6) I am attentive to my feelings.
_____ 7) I know exactly how I am feeling.
_____ 8) I care about what I am feeling.
_____ 9) I am confused about how I feel.
_____ 10) When I'm upset, I acknowledge my emotions.
_____ 11) When I'm upset, I become angry with myself for feeling that way.
_____ 12) When I'm upset, I become embarrassed for feeling that way.
_____ 13) When I'm upset, I have difficulty getting work done.
_____ 14) When I'm upset, I become out of control.
_____ 15) When I'm upset, I believe that I will remain that way for a long time.
_____ 16) When I'm upset, I believe that I'll end up feeling very depressed.
_____ 17) When I'm upset, I believe that my feelings are valid and important.
_____ 18) When I'm upset, I have difficulty focusing on other things.
_____ 19) When I'm upset, I feel out of control.
_____ 20) When I'm upset, I can still get things done.
_____ 21) When I'm upset, I feel ashamed with myself for feeling that way.
_____ 22) When I'm upset, I know that I can find a way to eventually feel better.
_____ 23) When I'm upset, I feel like I am weak.
_____ 24) When I'm upset, I feel like I can remain in control of my behaviors.
_____ 25) When I'm upset, I feel guilty for feeling that way.
_____ 26) When I'm upset, I have difficulty concentrating.
_____ 27) When I'm upset, I have difficulty controlling my behaviors.
_____ 28) When I'm upset, I believe that there is nothing I can do to make myself feel better.
_____ 29) When I'm upset, I become irritated with myself for feeling that way.
_____ 30) When I'm upset, I start to feel very bad about myself.
_____ 31) When I'm upset, I believe that wallowing in it is all I can do.
_____ 32) When I'm upset, I lose control over my behaviors.
_____ 33) When I'm upset, I have difficulty thinking about anything else.
_____ 34) When I'm upset, I take time to figure out what I'm really feeling.
_____ 35) When I'm upset, it takes me a long time to feel better.
_____ 36) When I'm upset, my emotions feel overwhelming.

APPENDIX F

BFIS-LF: Self Report

Instructions

How much difficulty do you have functioning effectively in each of these major life activities? That is, to what degree do you see yourself as being impaired in each of these life domains? Please circle the number next to each item that best describes your difficulties in functioning DURING THE PAST 6 MONTHS. If that situation does not apply to you (for instance, you don't drive a car, don't have children, don't live with anyone, etc.), please circle the 99 in the last column (under "Does not apply")

Major Life Activities	Not at all		Somewhat		Mild		Moderate		Severe		Does not apply
1. In your home life with your immediate family	0	1	2	3	4	5	6	7	8	9	99
2. In getting chores completed at home and managing your household	0	1	2	3	4	5	6	7	8	9	99
3. In your work or occupation	0	1	2	3	4	5	6	7	8	9	99
4. In your social interactions with strangers and acquaintances	0	1	2	3	4	5	6	7	8	9	99
5. In your relationships with friends	0	1	2	3	4	5	6	7	8	9	99
6. In your activities in the community (church, clubs, social groups, organizations)	0	1	2	3	4	5	6	7	8	9	99
7. In any educational activities (college, night classes, technical training, occupational training).	0	1	2	3	4	5	6	7	8	9	99
8. In your marital, co-living, or dating relationships	0	1	2	3	4	5	6	7	8	9	99
9. In your management of your money, your bills, and your debts	0	1	2	3	4	5	6	7	8	9	99

Major Life Activities	Not at all	Somewhat		Mild		Moderate			Severe		Does not apply
10. In driving a motor vehicle and in your history of citations and accidents	0	1	2	3	4	5	6	7	8	9	99
11. In your sexual activities and sex relations with others	0	1	2	3	4	5	6	7	8	9	99
12. In your organization and management of your daily responsibilities	0	1	2	3	4	5	6	7	8	9	99
13. In caring for yourself daily (dressing, bathing and hygiene, eating, sleeping, etc.)	0	1	2	3	4	5	6	7	8	9	99
14. In maintaining your health (exercise, nutrition, preventive medical and dental care, etc.)	0	1	2	3	4	5	6	7	8	9	99
15. In taking care of and raising your children	0	1	2	3	4	5	6	7	8	9	99

APPENDIX G

RISK-TAKING BEHAVIOUR QUESTIONNAIRE

Please indicate which behaviours you have engaged in over the LAST **SIX** MONTHS.

	N / Y		**HOW OFTEN?**				
DRIVING							
Have you exceeded the speed limit?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you NOT worn a seatbelt in a moving car?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you driven without a license?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you been a passenger with a drunk driver?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you ever driven after drinking?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
DRUGS/ALCOHOL/CIGARETTES							
Have you smoked marijuana?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you used cocaine or crack?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you used heroine or another illegal opiate?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you used inhalants (e.g. “huffing”)	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you used prescription drugs not prescribed by a doctor or that were not prescribed for you?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you consumed alcohol?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you smoked cigarettes?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times
Have you used any other illegal drug?	[1] NO	[2] YES	Never	Once	2-5 times	6-10 times	11 or more times

LAW BREAKING

Have you broken any laws with non-violent behaviour (e.g., shoplifting)?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you broken any laws with violent behaviour (e.g., assault with or without a weapon)?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you ever broken probation or other legal agreement?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you had an interaction with the police that resulted in arrest or detainment?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

FAMILY RULES BROKEN

Have you broken any rules set by your family (e.g.., curfew)?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

SEXUAL BEHAVIOUR

Have you had sexual intercourse? [1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you had oral sex? [1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you had anal sex? [1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you NOT used a condom or any other barrier method when engaged in sexual activity with a partner?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you been paid for sexual activity? [1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Have you had sexual activity with more than one person in a 24-hour period?

[1] NO [2] YES Never Once 2-5 times 6-10 times 11 or more times

Age at first sexual intercourse? _____ years Total life-time number of sexual partners? _____

VITA AUCTORIS

Dragana Ostojic was born in 1987 in Sarajevo, Bosnia and Herzegovina. She graduated from Etobicoke Collegiate Institute in 2005. From there she attended the University of Toronto where she obtained an H.B.Sc. in Human Behavioural Biology in 2010. During her undergraduate study, she completed two semesters at Uppsala University in Sweden. She is currently in the M.A. program in the Clinical Neuropsychology track at the University of Windsor.