Association Between Pubertal Onset and Symptoms of ADHD in Female University Students

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Abstract
Objective: This exploratory study investigated how the timing of female pubertal maturation was associated with the symptoms of ADHD in a non-clinical female undergraduate sample (N = 253). Method: Participants (M<sub>age</sub> = 20.2 ± 1.7 years) completed a set of self-report rating scales examining pubertal onset and ADHD symptoms and related deficits. Results: Logistic regression analysis indicated that early puberty was associated with elevation in symptoms, including difficulties in attention (odds ratio [OR] = 1.270, <i>p</i> = .019), emotion regulation (OR = 1.070, <i>p</i> = .038), and more risky behavior (OR = 1.035, <i>p</i> = .045). That is, increased symptom endorsement was shown to significantly help classify those who reported having an earlier pubertal onset relative to their peers. Conclusion: Findings highlight the potential role of sex hormones during puberty in explaining the gender differences in prevalence rates of ADHD and symptom profiles. (J. of Att. Dis. 2016; 20(9) 782-791)

Keywords
adult ADHD, females, sex hormones

Recent research suggests that ADHD affects approximately 3% to 4% of the adult population worldwide (Fayyad et al., 2007). Nevertheless, data on prevalence rates in adolescence/adulthood are limited and likely echo both underdiagnosis and less clinical attention in this age range (Kalbag & Levin, 2005; Miller, 2012). Across clinical settings, ADHD is reported to be more common in boys than in girls (American Psychiatric Association [APA], 2013); yet, there is some indication that the sex differences in ADHD are eliminated by adulthood (Nussbaum, 2012). Notably, however, most of the research conducted with adults who have ADHD, including those based on university samples, is focused on those who were previously diagnosed and are male (Nussbaum, 2012; Rodriguez & Span, 2008). By extension, the manifestation of ADHD symptoms in females has often been neglected in the literature, as have sex differences in ADHD symptom presentation (Nussbaum, 2012).

According to the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-V; APA, 2013), symptoms of ADHD include inattention and hyperactivity–impulsivity, yet most adult females do not evidence hyperactivity and their symptom levels often present at a later onset and with more subtle clinical symptoms (Taylor & Keltner, 2002). Moreover, the current set of symptoms neglects to mention certain central features of adult ADHD, such as emotional impulsiveness, poor emotional self-regulation, and poor working memory that may be more common in females with ADHD. Importantly, females with ADHD are equally susceptible to life course impairments as males (Lee, Lahey, Owens, & Hinshaw, 2008). Gaps in our current understanding of symptoms of adult ADHD, particularly in women, and controversy regarding what might constitute appropriate diagnostic criteria highlight the need for additional research.

The lack of a clear and consistent pattern of results across studies examining symptoms, behavior, 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, Keshava, & Giedd, 2008). 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). The developing adolescent brain is highly receptive to the effects of gonadal steroid hormones. Circulating steroids (e.g., estradiol and...
progestosterone) 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, Culber, Sisk, & Klump, 2007). This process is highly individualized, influencing virtually all mechanisms involved in the remodeling of the adolescent brain (e.g., dendritic elaboration, synaptic pruning, and axonal sprouting). As such, variation in the age of puberty onset contributes to individual differences in developmental course and behavioral maturation (Sisk & Zehr, 2005). Relatively, differences in the timing of puberty will contribute to the diversity of adult psychological characteristics, behaviors, 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).

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 symptoms of ADHD in females. 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 dopaminergic receptors may lead to a subsequent increase in symptoms (Nussbaum, 2012). Therefore, previous reports of remitting symptoms in ADHD into adolescence and young adulthood may be more reflective of the trajectory of male ADHD symptoms. Lending further support for an association between pubertal onset and ADHD symptoms, increased hormonal fluctuations throughout the phases of the menstrual cycle have been associated with increased symptomatology (Nadeau, Littman, & Quinn, 2006), and the manifestation of many of the known common comorbidities in females with ADHD has been shown to be affected by pubertal timing, such as anxiety disorders (Zehr et al., 2007). 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, and the prefrontal area, particularly involved in executive function, has been chiefly implicated in ADHD (Nussbaum, 2012). Yet, to date, there has been little research on this topic. This exploratory study sought to address the current gaps in our understanding of how female pubertal maturation is related to ADHD symptoms in a non-clinical female sample. Given that the ADHD symptoms reflect the extreme end of a behavioral continuum, it is possible to categorize all individuals somewhere on this spectrum, allowing for the investigation of symptoms of ADHD in a non-clinical sample. This was primarily an exploratory study, but given the noted negative consequences of early puberty onset, such as disordered eating and anxiety (Klump et al., 2012; Zehr et al., 2007), sexual risk taking, substance use, and anti-social behavior (Downing & Bellis, 2009), it was predicted that early maturation relative to peers would be associated with elevated levels of ADHD symptoms and difficulties in emotion regulation.

**Method**

**Participants**

Participants were 253 full- and part-time female undergraduate students recruited through a departmental research pool from a mid-sized university in southwestern Ontario. Participants were excluded if they reported having a history of a traumatic brain injury, lack of fluency in English, and were not between the ages of 18 and 25. Males were not eligible to participate. The mean age of the total sample was 20.19 years (±SD = 1.69). 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. Parental education level was used as a proxy for socioeconomic status (SES), with nearly 70% of participants reporting that a parent completed at least high school (69.9% of mothers and 68.1% of fathers). With regard to disclosure of relevant medical history, 2 participants identified history of head injury accompanied with a loss of consciousness, 7 participants disclosed history of seizures, 6 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. Given that internalizing problems are a common comorbidity of females with ADHD, and diagnosis could not be verified, information from these individuals was not excluded. Finally, it should be noted that the pubertal onset groups, described in detail below, did not significantly differ on any of these demographic variables (p = .096-.895).

**Measures**

**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 modified version of the PDS allows for the assessment of timing of pubertal development in post-pubertal adults. Initially used with an undergraduate sample at a large midwestern university, Sisk and colleagues (2005) reported that the modified measure has adequate psychometric properties. Specifically,
they reported good internal consistency ($\alpha = .80$ in females; $\alpha = .84$ in males) and excellent test–retest reliability ($r = .87$ for females; $r = .83$ 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, but these items were not incorporated in the total PDS-RV score and were purely informational. As was done by Zehr and colleagues (2007), the total PDS-RV score was calculated by summing the scores from the following six items, paralleling the Tanner staging categories: “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. 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. As was done by Zehr and colleagues (2007), quartiles for the summed total score of PDS were used to categorize early (lowest quartile), on-time (middle two quartiles), and late (highest quartile) pubertal onset. It should be noted that there are multiple ways to understand early and late pubertal onset. However, to be consistent with previous literature using the PDS-RV, quartiles for the summed total score on the PDS-RV were used to categorize individuals as having an early or late pubertal onset. The early pubertal onset group ($n = 64$) had a mean total score of 12.21 ($SD = 1.966$, range = 6.00-14.75), with the on-time onset pubertal group ($n = 127$) having a mean score of 17.22 ($SD = 1.314$, range = 15.00-19.18) and the late onset pubertal group ($n = 60$) 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 .81.

**Barkley Adult ADHD Rating Scale–IV (BAARS-IV).** Designed with consideration of DSM-IV (4th ed.; APA, 1994) diagnostic criteria, the BAARS-IV (Barkley, 2011a) is a self-report questionnaire in which the participants were asked to report their current ADHD symptoms. Specifically, with regard to the current symptoms interview, the participants were asked to indicate to what extent each item described their behavior during the past 6 months. The possible response for the questionnaire ranged from “Never/Rarely” (1) to “Very Often” (4). A total ADHD score and symptom count were calculated by adding up the scores for each item answered. From the BAARS-IV current symptoms questionnaire, four subscale totals were calculated corresponding 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. This score is near the middle of the norming sample distribution or slightly above it (51st-75th percentile). The internal consistency for all 27 items was excellent, with an alpha value of .90. Calculation of Cronbach’s alpha for the inattention, hyperactivity, impulsivity, and SCT subscales revealed alpha values of .82, .59, .69, and .87, respectively.

**Barkley Deficits in Executive Functioning Scale (BDEFS).** The BDEFS (Barkley, 2011b) is an 89-item Likert-type rating scale designed to evaluate the variety of behavioral, 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 6 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 of a possible maximum total score of 356 and was near the middle or slightly above the middle of the norming sample distribution. Internal consistency for the total score was evaluated to be excellent, with an alpha value of .97. Relatedly, the Cronbach’s alpha values for each subscale were also evaluated to be satisfactory (Self-Management to Time = .94, Self-Organization/Problem Solving = .94, Self-Restraint = .85, Self-Motivation = .84, and Self-Regulation of Emotion = .92).

**Difficulties in Emotion Regulation Scale (DERS).** The DERS (Gratz & Roemer, 2004) 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, Tull, Baruch, Bornovalova, & Lejuez, 2008). Participants were asked questions reflecting difficulties in four domains of emotion regulation, including (a) awareness and understanding of emotions, (b) acceptance of emotions, (c) the ability to use goal-directed behavior and control impulsive behavior when having negative emotions, and (d) 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) reported 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. This mean score was highly similar to the mean score for the normative sample (77.99 + 20.72). The internal consistency for all 36 items of the DERS was excellent, with an alpha value of .94. Cronbach’s alphas were also calculated for each DERS subscale, revealing the following: nonacceptance of emotional responses subscale (α = .89), difficulties engaging in goal-directed behavior subscale (α = .91), impulse control difficulties (α = .88), lack of emotional awareness (α = .84), limited access to emotion regulation strategies (α = .89), and lack of emotional clarity (α = .82).

The **Risk-Taking Behavior Questionnaire (RTBQ)**. Participants were asked to complete a self-report measure of their engagement in and frequency of specific risky behaviors, including driving, substance experimentation, lawbreaking, family rules compliance, and sexual behavior. This is an unpublished measure used extensively with university students. Preliminary analyses suggest that this measure has adequate psychometric properties. Participants were asked how frequently they engaged in the listed behaviors over the past 6 months, with possible responses ranging from 0 (“Never”) to 4 (“11 or more times in the past six months”). Only the summed total score is interpreted, which 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. Internal consistency, evaluated by calculating Cronbach’s alpha using all 23 items, was adequate with an alpha value of .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 revealing a messy missing data pattern. This suggested that the data were missing completely at random (MCAR). As such, given that there was minimal amount of missing data and evidence that the values were missing at random, Expectation-Maximization (EM) was used to replace the missing values.

A one-way multivariate analysis of variance (MANOVA) 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, because it was speculated that given the related content of the questionnaires the outcome variables would likely be correlated. Before proceeding with the analysis, verification of the statistical assumptions for MANOVA was done revealing that the assumptions were not met, and all subsequent attempts to remedy this problem were unsuccessful. Consequently, 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 (Tabachnick & Fidell, 2007). 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 in 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.

Logistic regression analysis was conducted to allow for a direct comparison between the early pubertal onset group and the on-time pubertal group, with timing of puberty serving as the outcome variable and the subscale and composite totals on the questionnaires serving as the predictors. Although counterintuitive, it allowed us to utilize statistical criteria to include and remove predictors from the equation (Tabachnick & Fidell, 2007). Furthermore, this methodology serves a good purpose in screening or hypothesis generating (Tabachnick & Fidell, 2007), which is appropriate given the exploratory nature of this study. When selecting a stepwise method, the backward method was preferred over the forward method because it allowed for the detection of suppressor effects, and thus was less likely to cause a Type II error (Field, 2009). Finally, with regard to the test statistic to be used in the stepwise method, the likelihood ratio method was used, given that the Wald statistic may produce inaccurate results under certain conditions (Field, 2009).

The potential explanatory variables were entered in groups based on their corresponding questionnaires. Relatedly, 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. For the purposes of comparing early with on-time...
pubertal onset using logistic regression, pubertal onset was operationalized as a dichotomous variable, with those characterized as having on-time pubertal onset labeled as the reference group. This is in line with how the PDS-RV has been used in previously published work. As such, the odds ratio was interpreted as 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.

### Results

**BAARS-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 SCT) were entered in stepwise backward entry logistic regression model. 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, $\chi^2(1) = 3.571, p = .059$. The Hosmer–Lemeshow test produced a non-significant chi-square value, $\chi^2(8) = 2.913, p = .940$, indicating an improvement in the model. None of the variables were identified as making a significant, unique contribution to the prediction of pubertal timing membership.

In contrast to the non-significant finding noted above, when symptom counts for the four subscales were entered in a logistic regression analysis, 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, $\chi^2(1) = 5.619, p = .018$, and the Hosmer–Lemeshow test produced a non-significant chi-square value, $\chi^2(8) = 2.913, p = .940$, indicating an improvement in the model. None of the variables were identified as making a significant, unique contribution to the prediction of pubertal timing membership.

### BDEFS

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 stepwise method. A significant model chi-square statistic, $\chi^2(1) = 5.680, p = .017$, revealed an improvement in model fit with the addition of the self-regulation of emotion subscale total to the model. Furthermore, the Hosmer–Lemeshow test produced a non-significant finding, $\chi^2(8) = 6.581, p = .582$. With the inclusion of the self-regulation of emotion subscale total, the model classified 66.5% of participants correctly. Table 2 includes regression coefficients in the final model, showing that 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%.

### DERS

The DERS yields a total score and six subscale scores: (a) nonacceptance of emotional responses, (b) difficulties engaging in goal-directed behavior, (c) impulse control difficulties, (d) lack of emotional awareness, (e) limited access to emotion regulation strategies, and (f) lack of emotional clarity. The six subscale totals were entered in the stepwise logistic regression analysis using the backward entry method. Examination of model fit revealed a significant model chi-square statistic, $\chi^2(1) = 4.357, p = .037$, indicating an improvement in model fit with the addition of the impulse subscale total to the model. The non-significant Hosmer–Lemeshow test, $\chi^2(7) = 7.867, p = .344$, also suggested an improvement in the model. With the inclusion of the impulse subscale, the model classified 66.5% of participants correctly. As depicted in Table 3, the odds ratio for the impulse subscale is 1.070, indicating that the odds of correctly predicting classification increased by 7.0% with the addition of this variable.

**Table 1. Regression Coefficients for the Final Model Variables: BAARS-IV Current Symptom Counts.**

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>Wald</th>
<th>df</th>
<th>p</th>
<th>Lower Odds ratio</th>
<th>Upper Odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inattention symptom count</td>
<td>0.239 (0.102)</td>
<td>5.492</td>
<td>1</td>
<td>.019</td>
<td>1.040</td>
<td>1.270</td>
</tr>
<tr>
<td>Constant</td>
<td>−0.911 (0.185)</td>
<td>24.309</td>
<td>1</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. $R^2 = .496$ (Hosmer & Lemeshow), .029 (Cox & Snell), and .040 (Nagelkerke). BAARS-IV = Barkley Adult ADHD Rating Scale–IV; CI = confidence interval.
All Total Scores

When the total scores from all four questionnaires were entered in the backward stepwise logistic regression analysis, examination of model fit revealed a significant model chi-square statistic, $\chi^2(1) = 4.040$, $p = .044$, 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, $\chi^2(7) = 8.467$, $p = .293$. Specifically, including the RTBQ total, the overall accuracy of the classification improved to 68.3%.

Table 4 includes regression coefficients in the final model. Examination of the final model revealed that the RTBQ total score makes a significant contribution to the prediction of group membership. The odds of correctly predicting classification increased by 3.5%.

For two of the six questionnaires used in the study, it was possible to calculate symptom counts: BAARS-IV current symptom count and BDEFS-EF (Executive functions) symptom count. These were entered in the stepwise 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, $\chi^2(1) = 4.040$, $p = .044$, 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, $\chi^2(7) = 8.467$, $p = .293$. Specifically, including the RTBQ total, the overall accuracy of the classification improved to 68.3%.

Table 5 includes regression coefficients in the final model. Examination of the final model revealed that the RTBQ total score makes a significant contribution to the prediction of group membership. The odds of correctly predicting classification increased by 3.5%.

Discussion

Overall, the present findings from this exploratory study suggest that early puberty may be associated with more current symptoms of ADHD and greater impairment on a variety of ADHD-related factors, including deficits in attention, difficulties in emotion regulation, and risky behavior. Consistent with existing literature suggesting that females with ADHD present with more inattentive rather than hyperactive/impulsive symptoms (Gaub & Carlson, 1997; Nussbaum, 2012), 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.

Deficits in Attention

The present findings suggest that pubertal onset is associated with current level of inattention symptoms, such as having difficulties in sustaining attention, difficulty following instructions, experiencing frequent forgetfulness in daily activities, and losing things. Specifically, as current inattention symptoms increase, the odds of an individual reporting that they matured earlier than their peers also increase. This finding provides empirical support for the influence of pubertal timing on ongoing attention difficulties, 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 may be misdiagnosed as having other psychopathology (e.g., dysthymia when inattentive symptoms are present alongside
low levels of arousal; Wender, Wolf, & Wasserstein, 2001), and comorbidities for which a role of puberty onset has been suggested, such as major depressive disorder (Martel, Gobrogge, Breedlove, & Nigg, 2008).

**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, the more likely they would be to be classified in the early pubertal onset group. Similarly, reporting more impulse control difficulties 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 & 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). There continues to be a need for further studies examining deficits in emotional regulation in an ADHD sample (Surman et al., 2013).

**Risky Behavior**

In line with previous reports noting the negative consequences of early puberty onset on adolescent risk-taking behavior (Downing & Bellis, 2009), early pubertal onset was associated with greater report of risk-taking behavior 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 behavior, including unhealthy substance use and risky sexual behavior (Downing & Bellis, 2009; Witt, 2007). For instance, Biehl, Natsuaki, and Ge (2007) reported 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- to 15-year-old Canadian girls sampled from the National Longitudinal Survey of Children and Youth revealed no association. Interestingly, Wichstrom (2001) reported that 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 behavior in domains that have been explored to a lesser extent, such as risky driving behavior or rule breaking.

**General Discussion**

Deficits in the prefrontal cortex and/or neural circuits linking the prefrontal cortex to subcortical regions have been implicated in symptoms of ADHD (Biederman et al., 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 et al., 2005; Halperin & Schulz, 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 in 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 female rats do not display this pattern of over-production and elimination of dopamine receptors (Andersen & Teicher, 2000). Andersen and Teicher (2000) suggested that the lack of pre-programmed elimination of dopamine receptors in the striatum in females with ADHD may explain why there is persistence of problems into adulthood. Furthermore, they hypothesized that delayed pruning of dopamine receptors in the prefrontal 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 behavioral impairments in adulthood. Relatedly, it is possible that the observed relationship may be due to the fact that childhood mental health problems, including the diagnosis of ADHD and conduct problems, have been associated with higher BMI scores (Duarte et al., 2010; Holtkamp et al., 2004; Waring & Lapane, 2008). In turn, higher BMI has been linked to the timing of pubertal onset (Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001). Finally, 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, 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 behavioral problems, experiences which may affect current symptom presentation via their direct or indirect impact on the development of the nervous system post-puberty (Mendle, Turkheimer, & Emery, 2007).

The findings from this exploratory study are limited by several factors. First, by using a non-clinical university sample, one is limited in the conclusions that are warranted with regard to the nature of ADHD symptoms 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 adult females (Gaub & Carlson, 1997). Thus, future studies should aim to capture as large of a community sample of adult 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 (Kaplowitz et al., 2001; Wu, Mendola, & Buck, 2002), 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) reported moderate to high agreement between adult self-report and others-report with regard to current symptoms and impairment. Future studies should attempt to incorporate objective pubertal measures and biomarkers reflective of pubertal hormonal processes. 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, and future work in this area would benefit from studies implementing a prospective longitudinal design. Despite these limitations, the study does help address the gap in our understanding of the relationship between pubertal timing and ADHD-related symptoms and impairments later in life. Narrowing 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, the study takes advantage of self-report behavior 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.

The present findings do not provide evidence for a causational relationship between pubertal timing and ADHD symptoms in emerging adulthood. They do, however, 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 on ADHD symptom presentation in a non-clinical sample. 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). Furthermore, 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). 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 on ADHD symptoms.

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