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The Developmental Trajectory of ADHD in Females: Predictors and Associations of Symptom
Change from Childhood to Young Adulthood

by

Fred Loya

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The Developmental Trajectory of ADHD in Females: Predictors and Associations of Symptom Change from Childhood to Young Adulthood

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Abstract

The Developmental Trajectory of ADHD in Females: Predictors and Associations of Symptom Change from Childhood to Young Adulthood

by

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Doctor of Philosophy in Psychology

University of California, Berkeley

Professor Stephen P. Hinshaw, Chair

This dissertation presents an examination of the developmental course of symptoms of hyperactivity-impulsivity (HI) and inattention from childhood (*Mdn* age = 8.6 years) through young adulthood (*Mdn* age = 20.0 years) in an ethnically diverse sample of females diagnosed with childhood ADHD ($n = 140$) and a matched comparison group ($n = 88$). Latent growth curve models of caregiver-reported symptoms indicate that, over time, probands experienced a marked decline in all ADHD symptom domains (total, HI, and inattentive) yet continued to show significantly elevated symptom levels in young adulthood relative to the comparison sample (4 to 11 times their mean levels). Probands also experienced more maladaptive outcomes in adulthood, including internalizing symptomatology, functional impairments, and tobacco use. ADHD symptom severity in childhood and rate of symptom change over time each independently predicted these outcomes, with inattentive symptoms serving as more robust predictors of maladjustment than HI symptoms. Indeed, changes in inattentive symptoms across development exerted nearly double the effect of HI symptom change on internalizing symptoms and impairment in adulthood. Moreover, baseline inattentive symptoms and their rate of change were predictive of tobacco use, whereas HI symptoms were not. In addition, adult symptoms of ADHD were predicted by child psychopathology, paternal distress, and parental psychopathology, controlling for baseline ADHD status. These findings prospectively show that ADHD symptoms persist into young adulthood in women, and can be predicted by child and parent psychosocial and psychopathological variables. Overall, females with a childhood diagnosis of ADHD continue to show clinically significant impairments in crucial life domains into young adulthood. Findings underscore the relative importance of inattentive symptoms for females and stress the need to identify high-risk cases early in development.

Introduction

Although attention-deficit/hyperactivity disorder (ADHD) has traditionally been viewed as a psychiatric condition primarily affecting males, it is increasingly recognized to be associated with significant morbidity and functional impairment in samples of girls during both childhood and adolescence (Arnold, 1996; Gershon, 2002; Hinshaw, 2002; Hinshaw, Owens, Sami, Fargeon, 2006). Yet prospective studies following girls with ADHD into young adulthood are relatively rare (for exceptions, reviewed below, see Babinski et al., 2011; Biederman et al., 2010). Indeed, nearly all of what is known about the developmental trajectory of ADHD emanates from predominantly or exclusively male samples (e.g., Barkley, Murphy, & Fischer, 2008; Rasmussen & Gillberg, 2000; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Weiss, Hechtman, Millroy, & Perlman, 1985). Such prospective follow-up studies consistently document that childhood ADHD is associated with maladaptive outcomes in adulthood, including psychiatric comorbidity (Biederman, Monuteaux, Mick, Spencer, Wilens, Silva et al., 2006; Rasmussen & Gillberg, 2000), poor academic and job performance (Barkley, Fischer, Smallish, & Fletcher, 2002), risky sexual behaviors (Flory, Molina, Pelham, Gnagy, & Smith, 2006), and substance use (Barkley, Fischer, Smallish, & Fletcher, 2004). Given the public health implications of these negative developmental outcomes as well as girls' generally high risk for developing internalizing problems during adolescence (Hinshaw, 2009), a better understanding of the impact of childhood ADHD on functioning and adjustment for women in young adulthood (and beyond) is sorely needed.

In childhood, girls with ADHD display marked dysfunction relative to community controls (see Biederman et al., 1999; Hinshaw, 2002). Biederman and colleagues (1999) reported on a sample of girls, aged 6-17 years, with ($n = 140$) and without ($n = 122$) childhood ADHD, finding elevated rates of anxiety, mood, and conduct disorder, as well as gross functional impairments within multiple domains among ADHD participants. A follow-up of this sample into mid-adolescence ($M = 16.4$ years) yielded similar outcomes (Biederman, Monuteaux, Mick, Spencer, Wilens, Klein et al., 2006): relative to controls, probands were at increased risk for both lifetime and adolescent psychopathology, particularly major depressive disorder ($OR = 36.2$) and oppositional defiant disorder ($OR = 31.2$). Using an ethnically and socioeconomically diverse sample, Hinshaw and colleagues found that in childhood, girls with ADHD ($n = 140$) experienced more internalizing and externalizing pathologies, performed worse academically, were less socially accepted, and had significant decrements in executive functioning relative to their non-ADHD peers ($n = 88$) (Hinshaw, 2002; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002). At adolescent follow-up ($M = 14.2$ years), probands continued to be characterized by higher rates of externalizing pathologies, worse academic achievement, poorer peer relationships and social skills, worse executive functions, more functional impairment, and fewer instances of positive adjustment than comparisons (Hinshaw et al., 2006; Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Owens, Hinshaw, Lee, & Lahey, 2009). These differences were of medium to large effect and were maintained even after potential confounding variables (e.g., current medication status, childhood comorbidities, demographic variables, IQ) were strictly controlled, signifying that childhood ADHD status exerts a direct or "specific" negative effect on adolescent outcomes not attributable to other known risk factors (Hinshaw et al., 2006).

Only two prospective, longitudinal studies have followed girls with ADHD into young adulthood: the Pittsburgh Adolescent Longitudinal Study (PALS) by Molina and colleagues (e.g., Flory, Molina, et al., 2006) and the aforementioned work of Biederman and colleagues

(2010). Analyzing the female subsample of PALS participants ($n = 34$ ADHD and $n = 24$ non-ADHD comparisons), Babinski et al. (2011) found that young adult women with ADHD ($M = 20.0$ years) performed worse academically and vocationally, had more interpersonal conflicts with family and friends, and had lower self-esteem than comparison women – effects that were, by and large, evident only via parental report (see Barkley et al., 2002). However, the authors failed to find differences in other developmental domains, including self-reported substance use, perhaps reflecting the low statistical power afforded by their relatively small sample. Biederman and colleagues (2010) reassessed their cohort of ADHD ($n = 140$) and comparison ($n = 122$) girls in young adulthood ($M = 22.0$ years), finding that young adult women with childhood or adolescent ADHD had significantly higher lifetime prevalence rates across six composite measures of psychopathology (i.e., Mood, Anxiety, Antisocial, Developmental, Substance Dependence, and Eating Disorders). All measures remained significantly elevated after controlling for baseline rates of these disorders. Still, key questions remain regarding the developmental course of ADHD in girls, particularly related to changes in specific symptom clusters (i.e., hyperactivity/impulsivity [HI] and inattention) over time and how symptom change may impact important domains of functioning for girls as they progress into adulthood.

In addition to these prospective accounts documenting the effects of childhood ADHD on adult outcomes, a number of studies have shown that ADHD persists into adulthood in a substantial number of cases (Barkley et al., 2002; Babinski et al., 2010; Rasmussen & Gillberg, 2002; Faraone, Biederman, & Mick, 2006). Although reported estimates of ADHD-persistence vary widely based upon the diagnostic criteria employed, it is clear that between two-thirds and three-quarters of children with ADHD continue to exhibit clinically meaningful symptoms into adulthood, even if they don't surpass official *DSM* thresholds (Barkley et al., 2002; Biederman, Petty, Clarke, Lomedico, & Faraone, 2011; also see Faraone et al., 2006). Yet, these studies often fail to provide crucial information regarding specific risk factors that may account for why some cases of ADHD persist into adulthood while others remit over time.

Most studies (e.g., Biederman et al., 1996; Fisher, Barkley, Fletcher, & Smallish, 1993; Keown, 2011; Lahey et al., 1994; Mick et al., 2011; Sciberras, Ukoumunne, & Efron, 2011) addressing ADHD-persistence have been limited to childhood and adolescent outcomes and/or have included predominately male participants. Nonetheless, these studies have consistently found that severity of ADHD in childhood, and childhood psychiatric comorbidities and adversity, the latter including parental psychiatric illness and family discord, have predicted the persistence of ADHD into adolescent years. For instance, Biederman and colleagues (1996) reported on an exclusively male sample and found that baseline ADHD symptom severity, a family history of ADHD, family conflict, and child externalizing pathologies (conduct disorder and oppositional defiant disorder) each independently predicted ADHD-persistence into adolescence. Consistent with these findings, Hart et al (1995) found that boys continuing to meet criteria for ADHD at four-year follow-up were more hyperactive-impulsive and more likely to have conduct disorder at baseline. Utilizing a population-based sample ($N = 3,474$; 49% female), Sciberras et al. (2011) found that children with maternal postnatal depression were twice as likely to have ADHD in childhood than children whose mothers were not depressed.

Whereas some research has examined predictors of ADHD-persistence into adolescence, there is paucity of information available about (a) predictors of ADHD-persistence into adulthood and (b) predictors of ADHD-persistence for females. This situation needs to be redressed, given that the disorder may be more persistent for women relative to men (Hinshaw et al., 2006; Monuteaux, Mick, Faraone, and Biederman, 2010). In addition, adolescent girls are at

heightened risk for internalizing pathologies (Hinshaw, 2009), which may confer a unique risk-factor for the persistence of ADHD beyond adolescence in this population. For instance, preliminary evidence based upon an exclusively female sample suggests that both externalizing and internalizing pathologies (anxiety/depression) are predictive of ADHD-persistence into adolescence (Mick et al., 2011), although previous studies with males have not found predictive associations with depressive symptoms (Biederman et al., 1996). The unique predictive associations between internalizing problems and adolescent-persistent ADHD in girls underscore the unique influence of internalizing problems for girls relative to their male counterparts.

Extant findings are mixed about the role that childhood psychopathology plays with respect to later ADHD. Biederman et al (2011) examined childhood predictors in their sample of boys, finding that maternal psychopathology, severity of childhood ADHD, and childhood psychopathology each predicted persistence into adulthood. An important finding in this analysis was that baseline externalizing psychopathology (ODD and conduct disorder) was the only variable that differentiated boys who continued to exhibit symptoms into adulthood compared to those whose symptoms remitted. However, Kessler and colleagues (2005), utilizing a well-characterized epidemiological sample ($N = 3197$; 41.1% female), found that childhood ADHD symptom severity was the only significant predictor of ADHD-persistence into adulthood. These discrepant findings may reflect the retrospective design utilized by Kessler et al (2005); previous research has established that underreporting of symptoms is characteristic of retrospective studies (see Hardt & Rutter, 2004).

Available information suggests that ADHD symptom severity and comorbidities are important risk factors for the persistence of ADHD among females into at least adolescence and into adulthood for males. Clearly, more work is needed in order to gain a better understanding of modifiable risk factors affecting the developmental course of ADHD in women. In addition, the studies reviewed here have all assessed predictors of ADHD-related persistence evaluated as a dichotomy (i.e., present vs. absent) – often utilizing widely discrepant operational definitions of persistence – rather than via changes in symptom levels measured as continua. This methodological difference is important as previous research has established that functional impairments persist for children with ADHD who fail to meet diagnostic criteria in later years (Biederman et al., 2010; Mick et al., 2011), even when persistence is defined as *clinically significant* symptom presence not meeting *DSM* standards. Thus, even the liberal definitions of persistence utilized in these studies may fail to capture levels of ADHD symptoms that are still associated with maladjustment.

In this dissertation, I utilize 10-year follow-up data on girls originally diagnosed with ADHD in childhood, plus matched comparison girls (Hinshaw, 2002), to assess for group differences in key domains of functioning in young adulthood and child- and parent-level variables that predict symptom change across development. This is the largest sample of preadolescent-ascertained girls with ADHD in existence. First, through the use of latent growth curve models (LGMs), I examine the developmental trajectory of caregiver-reported ADHD symptom change from childhood through young adulthood (see Barkley et al., 2002, for issues regarding self-report bias among young adults with ADHD). I address which symptom clusters (i.e., HI vs. inattentive, measured by childhood severity as well as change across development) independently predict adult outcomes. I focus on these ADHD symptom domains as continua because categorical subtype classifications may be unstable, fluctuating markedly across development (e.g., Lahey, et al., 2004). Additionally, modeling symptoms as continua may allow for the detection of subtle associations that otherwise could go undetected when ADHD is

defined as absent vs. present. Exploring the impact of inattentive symptoms may also be particularly relevant, given their potential importance for females (American Psychiatric Association [APA], 2000; Hinshaw & Blachman, 2005). Second, I specify developmental outcomes as latent constructs, allowing them to be modeled in a broad and inclusive manner, assessed via multiple measures (e.g., symptom counts, clinical correlates) and, where possible, multiple informants (probands, study staff, caregivers). Finally, I focus on child- and parent-level predictors of the trajectory of ADHD symptoms across development. Specifically, I focus on variables established by previous research as being related to ADHD persistence, namely familiarity of ADHD, maternal depression, parental stress, and child psychopathology.

I hypothesize the following: First, based on a recent study by Monuteaux, Mick, Faraone, and Biederman (2010), who found that rate of symptom change did not differ between male and female participants, I predict that symptoms of HI and inattention will abate as a function of age, with HI symptoms declining more sharply (see Faraone, Biederman, & Mick, 2006; Hart, Lahey, Loeber, Applegate, & Frick, 1995). Second, I hypothesize that girls with a childhood ADHD diagnosis will continue to experience marked impairment across the following developmental outcomes in adulthood: internalizing symptomatology, functional impairment, risky sexual behaviors, and substance use. I selected these outcomes because of their clinical significance and established relevance to both male and female ADHD samples (e.g., Biederman, Monuteaux, Mick, Spencer, Wilens, Silva et al., 2006; Hinshaw et al., 2006). Third, I predict that group differences in these impairment domains will be predicted by initial symptom severity and rate of symptom change over time. However, because of the paucity of research on the relation of symptom clusters to functioning, I make no predictions regarding the relative predictive abilities of HI versus inattentive symptoms on young adult outcomes. Regarding predictors of ADHD change, I predict that child psychopathology will be associated with greater numbers of symptoms in adulthood and less symptom change across time. In addition, I predict that maternal ADHD and depressive symptoms, as well as parental stress, will be associated with a slower decline in symptoms across development and heightened symptom levels in adulthood.

Method

Participants

Recruitment strategies and sampling procedures have been reported previously (see Hinshaw, 2002; Hinshaw et al., 2006) and are summarized here. A multi-gated procedure was used to recruit a sample of girls with ADHD and age- and ethnicity-matched comparisons (baseline age range: 6-12 years), primarily from medical settings, pediatric practices, school referrals, and community advertisements. Interested families were initially screened by phone and were administered parent and teacher rating scales of their daughters' ADHD symptoms. Potential participants were provisionally placed in the ADHD group if their scores surpassed sex-specific thresholds (e.g., presence of 5 of 9 symptoms on the Swanson, Nolan, and Pelham scale [SNAP-IV]; [Swanson, 1992]). Parents were then invited for a face-to-face diagnostic interview (i.e., the Diagnostic Interview Schedule for Children – Parent version [4th ed., DISC-IV]; [Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000]). ADHD status was defined as meeting full Diagnostic and Statistical Manual – 4th, Ed. (*DSM-IV*; APA, 2000) criteria for ADHD, derived from DISC-IV scores. To ensure the representativeness of the sample, common psychiatric comorbidities were permitted. Exclusion criteria were mental retardation, lack of English spoken in the home, evidence of psychosis or overt neurological dysfunction, or medical issues preventing participation in summer camps. A comparison sample, group-matched on age

and ethnicity, could not have met diagnostic criteria for ADHD based upon either the DISC-IV or SNAP-IV. The sample was ethnically and socioeconomically diverse (see Hinshaw, 2002).

Participants took part in one of three separate summer enrichment day camps (1997, 1998, 1999) and were assessed on a wide range of symptom-based, behavioral, relational, neuropsychological, and observational measures (Hinshaw, 2002). They were then invited to participate in a 5-year follow-up ($n = 209$, retention rate 92%; Hinshaw et al., 2006), where data were again collected across multiple domains. The retained sample ($M = 14$ years) was highly reflective of the total sample; participants lost to attrition differed on only 2 of 31 demographic, symptom, and diagnostic variables assessed at baseline (see Hinshaw et al., 2006). For the present 10-year follow-up, 216 of the original 228 subjects (95 %) participated, reflecting considerable efforts to track and locate the sample, including use of social media. Age at follow-up ranged from 17 to 25 years ($M = 19$; $SD = 1.7$). The 10-year follow-up sample did not differ from the small number lost to attrition on 19 of 23 baseline characteristics, although they were less symptomatic and had higher socioeconomic status (Hinshaw et al., 2011). I analyzed participants' caregiver-reported symptom data across all three assessment periods, but outcomes in young adulthood reflect functioning at the 10-year follow-up period only.

Procedures

Highly trained bachelor-level research assistants or graduate students in clinical psychology conducted all follow-up assessments. Evaluations of young adults and their primary caregiver (usually mother) were conducted across two half-day assessments, involving structured diagnostic interviews and self-report measures (as well as neuropsychological tests, not considered herein). The majority of assessments were conducted on campus; phone interviews or home visits were conducted for participants unable to travel to this location. Study assessors were unaware of participants' baseline diagnostic status. Although many participants were currently taking stimulants (~22%) and/or other psychotropic medications (~15%), participants and their caregivers were instructed to rate ADHD symptoms during periods in which stimulant medications were not taken. Finally, to promote participant comfort and greater self-report accuracy, particularly within highly-sensitive domains (e.g., number of sexual partners), female staff conducted all young adult interviews. Participants aged 18 or over provided informed consent; for younger probands (all of whom were 17; $n = 44$), we obtained their written assent, as well as written parental consent. The University's Institutional Review Board approved all study procedures.

Measures

ADHD Status and Symptoms

Diagnostic Interview Schedule for Children-4th edition (DISC-IV) (Shaffer et al., 2000). The DISC-IV is a highly-structured, well-validated diagnostic interview widely used in research with child and adolescent psychiatric populations. The DISC-IV yields total symptom counts and categorical diagnoses for major *DSM-IV* disorders based on symptom duration and degree of impairment. DISC-IV *DSM-IV* diagnoses have shown adequate one-year test-retest reliability across clinical and community samples and have been shown to be concordant with clinician-based diagnoses derived in research settings (Shaffer et al., 2000). The DISC-IV was administered to caregivers at Wave 1 to ascertain childhood ADHD status. DISC-IV dimensional symptom counts were *not* used in the present LGMs.

Swanson, Nolan, and Pelham Rating Scale – 4th Edition (SNAP – IV) (Swanson, 1992). We used the 39-item version of the SNAP-IV, which includes the 18 *DSM-IV* symptoms of ADHD (9 symptoms of Hyperactivity/Impulsivity and 9 symptoms of Inattention). Caregivers rated each symptom on a 4-point scale, ranging from 0 (*not at all present*) to 3 (*very much present*). Ratings within each symptom domain were averaged. Caregivers provided SNAP-IV ratings at each assessment wave, which served as the continuous measure of ADHD symptoms used to model all LGMs. The SNAP-IV has been used as the primary measure of ADHD symptoms in previous research (e.g., MTA Cooperative Group, 1999). Bussing and colleagues (2008) reported moderate to excellent internal consistency estimates ($\alpha = 0.79 - 0.90$) for parent-rated SNAP-IVs ($n = 1,613$). Parent-rated SNAP-IV scales are also significantly elevated amongst children qualifying for a DISC-IV ADHD diagnosis (Bussing et al., 2008).

Latent Outcome Variables

Internalizing Symptomatology. Adult Self-Report (ASR) (Achenbach, 2009). The ASR is the adult, self-report version of the extensively used Child Behavior Checklist (Achenbach, 1991), incorporating many items used previously in Achenbach's Child Behavior Checklist forms. The ASR has well-established psychometric properties. We utilized the ASR's *DSM*-oriented subscales of Depressive Problems and Anxiety Problems as indicators of participants' internalizing symptoms. Items were rated on a 0-2 metric; *T*-scores were utilized in this analysis.

Rosenberg Self-esteem Scale (RSES) (Rosenberg, 1965). The RSES is a ten-item measure of global self-esteem, with positively- and negatively-worded statements about the self. Participants rated statements on a 1 (*strongly disagree*) to 4 (*strongly agree*) scale; scores were averaged after reverse scoring half the items. The RSES is among the most widely used and validated research measure assessing self-esteem; it has excellent estimates of internal consistency, test-retest reliability, and convergent validity (Blascovich & Tomaka, 1993).

Functional Impairment

Columbia Impairment Scale (CIS) (Bird, 1999). The CIS is widely used to assess functional impairment across home, school/vocational, peer, and leisure domains. Caregivers rated 13-items from 0 (*No problem*) to 4 (*Very bad problem*), and item scores were averaged. The CIS has acceptable estimates of internal consistency ($\alpha = .89$), test-retest reliability ($r = 0.68$), and convergent validity with other impairment measures (Bird, 1999), and has been used as a primary outcome measure in previous ADHD research (e.g., Hinshaw et al., 2006).

Adult Self-Report (ASR) (Achenbach, 2009). We utilized the ASR subscale of Total Problems as an indicator of the degree of functional impairment experienced by participants (see above for more detail). Items were rated on a 0-2 metric and *T*-scores were used in all analyses.

Global Assessment of Functioning (GAF) (APA, 2000). Two independent staff (i.e., the proband and caregiver interviewers) rated the young adults' overall level of functioning with the *DSM-IV* (APA, 2000) GAF scale. These staff members then met to discuss their respective interview impressions and GAF ratings, and a single consensus GAF score was then generated.

Risky Sexual Behavior

We asked participants three questions about their sexual practices as indicators of risky sexual behavior. First, participants rated from 1 (*11 years or younger*) to 8 (*18 years or older*) the age they first had sexual intercourse; participants who reported that they had not yet had any sexual contact were coded as 9. No significant difference between the ADHD and comparison

sample was found for having any sexual contact, $\chi^2 [1, N = 198] = 0.48, p = 0.49$. Second, participants rated from 1 (*Never*) to 4 (*Always*) how often their partner used condoms during sex; participants who had not yet engaged in sexual contact were coded as 5. Finally, young adults reported their total number of sexual partners across their lifetime.

Substance Use

Substance Use Questionnaire (SUQ) (Molina & Pelham, 2003). The SUQ is a semi-structured interview that was adapted from previous substance use and health questionnaires (see Molina & Pelham, 2003). The SUQ includes questions pertaining to any use of tobacco, alcohol, marijuana, and other illicit substances over the lifetime, as well as the frequency and quantity of use over the past six months. The SUQ has been used previously with ADHD samples (e.g., Hinshaw et al., 2006). For our purposes, we analyzed six-month frequency estimates for tobacco, alcohol, marijuana, and other non-marijuana substances.

Predictors of Symptom Change

The first three measures below reflect functioning in participants' caregivers; the last two are caregiver reports of participants' symptomatology.

Conners' Adult ADHD Rating Scale (CAARS) (Conners et al., 1999). This measure was used to ascertain levels of ADHD-related symptomatology in caregivers of the participants. The CAARS self-report short-form consists of 26-items, including the 18 *DSM* symptoms of ADHD. Items were rated from 0 (*not at all*) to 3 (*very much present*). For this analysis, I combined the subscales of inattention/memory problems, hyperactivity/restlessness, and impulsivity/emotional lability to form an overall ADHD score. As such, subscale scores were first converted into standard units (i.e., *z*-scores) and were then averaged. All primary caregivers completed these items at baseline. The CAARS has well-established psychometric properties (Conners et al., 1999).

Beck Depression Inventory - II (BDI-II) (Beck & Steer, 1987). The BDI-II consists of 21-items in which each item consists of four different statements about the severity of different symptoms of depression. Scores across all items are summed such that higher scores reflect higher levels of depression. The BDI-II is one of the most widely utilized and validated measures of depressive symptomatology. The internal consistency of the measure derived from various clinical samples is excellent ($\alpha=0.86$; Beck & Steer, 1987). Primary caregivers completed the BDI-II at baseline with respect to their depressive symptoms over the preceding two weeks.

Parenting Stress Index (PSI) (Abidin, 1995). The PSI is a 36-item self-report measure of parenting stress, with items being rated on a 1-5 metric. The PSI has three subscales, each consisting of 12 items. For this analysis, I utilized the Parenting Distress subscale, which reflects the parental distress resulting from a combination of personal factors and the demands of parenting. Note that on other PSI subscales, some items ask parents to rate child problem behavior, confounding stress in the parent with problematic child behavior; Parenting Distress does not. The PSI has good psychometric properties (Abidin, 1995). Primary caregivers were administered the PSI at baseline.

Child Behavior Checklist (CBCL) (Achenbach, 1991). The CBCL is a widely used parental report of child psychopathology. In this analysis, I utilized the broadband Externalizing and Internalizing dimensions, as well as the narrowband aggressive and anxious/depressed behaviors. The CBCL has excellent internal consistency, test-retest reliability, and convergent

validity with other measures of child psychopathology (Achenbach, 1991). Caregivers completed the CBCL at baseline; *T*-scores were utilized in all analyses.

Swanson, Nolan, and Pelham Rating Scale – 4th Edition (SNAP – IV) (Swanson, 1992). I utilized 8-items from the SNAP as a measure of oppositional defiant disorder (ODD). Items were rated on a 0-4 scale; items were summed and averaged. Caregivers reported on their daughter's ODD symptoms at baseline.

Data Analytic Strategy

The trajectory of ADHD symptoms (total, HI, and inattentive) was evaluated with separate LGMs utilizing Amos structural equation modeling software (v. 18; Arbuckle, 2009). I reorganized the data to allow symptom change to be modeled on approximate age rather than assessment wave (see Bollen & Curran, 2006, chapter 3). I first divided participants' ages across all three assessment periods into quartiles, approximating developmental periods (in months) of childhood (79 – 128 mos., *Mdn* = 104), early adolescence (129 – 165 mos., *Mdn* = 145), adolescence (166 – 217 mos., *Mdn* = 192), and early adulthood (218 – 300 mos., *Mdn* = 240). I then assigned participants to respective groups based upon their age at each assessment wave. Thus, a participant with complete data would have a maximum of three observations, with the fourth observation estimated via maximum-likelihood procedures. This analytic strategy is considered an improvement over earlier techniques (e.g., controlling age at baseline in the prediction of slope and intercept) when wave of assessment and age are confounded (Curran & Bollen, 2006).

Parental reports of SNAP-IV ADHD symptoms were the observed indicators in modeling all growth curves. I did not specify the trajectory of the growth curve *a priori*; factor loadings were freely estimated from the data. This approach models rate of change empirically, allowing it to take non-linear forms. Moreover, estimated factor loadings reflect the proportion of change in symptom levels that occurred between baseline and each subsequent developmental period. I included the ADHD status of participants ascertained at the first assessment period (see Hinshaw, 2002) as a predictor of slope and intercept factors. Tests of these associations serve as an omnibus appraisal of the multiplicative interaction between initial ADHD status and time in the prediction of ADHD symptoms (see Curran, Bauer, & Willoughby, 2004). Thus, any significant associations indicate that mean symptom levels in childhood (intercept) and rate of symptom change (slope) differed for ADHD and comparison participants. Following a significant omnibus test, I examined simple slopes to pinpoint the nature of the moderated effect.

To test the predictive effects of initial ADHD symptom severity and symptom trajectory on outcomes, I first constructed separate latent constructs for each developmental domain. In these models, baseline ADHD status served as a predictor of random slope and intercept factors; these factors, in turn, were used as separate predictors of each (latent) developmental outcome (see Figure 1). I constructed separate models for total, inattentive, and HI symptoms.

Finally, to examine predictors of symptom change across development, I constructed separate models for each predictor. In these models, the intercept was intentionally set to reflect mean symptom levels in young adulthood. Thus, a significant relationship between predictor variables and the intercept indicates that variables measured at baseline are predictive of symptom levels 10-years post-study entry. I included baseline ADHD status as a predictor of slope and intercept, allowing it to covary with predictor variables. As in the other models, associations between a predictor variable and the slope factor represent an interaction effect between the predictor and time in the prediction of ADHD symptoms across time; baseline

ADHD status was controlled in these interaction analyses (See Figure 2). Model fit was evaluated with the model chi-square statistic and three fit indices commonly reported in the SEM literature: the comparative fit index (CFI; values > 0.90), the non-normed fit index (NNFI; values > 0.90), and the root mean square error of approximation (RMSEA; values < 0.05 with 90% CI containing 0) (Hu & Bentler, 1999; Kline, 2005). I appraised significance of model parameters using conventional standards (i.e., $\alpha = 0.05$).

Results

Missing Data and Preliminary Analyses

Missing data emanated from three sources: (a) subject attrition, (b) the organizational structure of the data (see Data Analytic Strategy), and (c) incomplete data on outcome variables. Regarding subject attrition, data from 209 subjects were available at Wave 2 and 216 at Wave 3. Incomplete data for particular outcome measures were more substantial, ranging from 8% for the self-reported frequency of tobacco use ($n = 18$) to 32% for staff-rated GAF scores ($n = 69$). To handle all missing data I utilized full maximum likelihood estimation procedures, an approach with many desirable properties (e.g., yielding unbiased and efficient population estimators). It is viewed as the preferred means of handling missing data with LGMs (Bollen & Curran, 2006). Thus, I utilized data from all 228 participants in all growth curve analyses.

However, I first report preliminary findings of mean differences for young adult women with and without a childhood ADHD diagnosis across all study variables utilizing only the observed data (see Table 1). First, caregivers reported that probands experienced more total, inattentive, and HI symptoms relative to comparison girls in young adulthood (all $t_s > 8.60$, $p_s < 0.001$). Second, women with a childhood ADHD diagnosis were more functionally impaired (all indicators significantly differed), experienced more internalizing symptoms (all indicators significantly differed), and used tobacco more frequently than women without childhood ADHD (all $t_s > |2.00|$, all $p_s < 0.05$). Effect sizes for these differences ranged from $d = 0.30$ for self-esteem to $d = 1.91$ for inattentive symptoms; effect sizes for the non-significant comparisons ranged from $d = -0.18$ for self-reported alcohol use (i.e., comparison girls reported *more* alcohol use) to $d = 0.13$ for the use of non-marijuana illicit substances. Although these latter, small-effect differences did not attain statistical significance, they were in the hypothesized direction (except for alcohol use), such that the ADHD sample showed more impairment.

Growth Curve Analyses of ADHD Symptoms

Total symptoms. The baseline LGM of caregiver report of total ADHD symptoms, as predicted from childhood ADHD status, showed an excellent fit to the data, $\chi^2(5, N = 228) = 4.26$, $p = 0.51$, CFI = 1.00, NNFI = 1.00, RMSEA = 0.00, CI_{0.90} {0.00, 0.09}. The mean slope factor was significant and negative, indicating that total symptoms decreased over time. The freely-estimated factor loadings of the latent slope factor on the repeated total symptom measures were $b_1 = 0.45$, $p < 0.001$, and $b_2 = 0.88$, $p < 0.001$. Thus, the overwhelming majority (88%) of change in total ADHD symptoms occurred by the time participants reached adolescence. Childhood diagnostic status¹ was predictive of initial symptom severity (estimate = 1.74, $p < 0.001$; $\beta = 0.95$) and rate of symptom change (estimate = -0.70, $p < 0.001$; $\beta = -0.63$),

¹ Childhood ADHD status was coded such that positive associations between ADHD status and latent intercept and slope factors reflect increased symptom severity in childhood and more positive rates of symptom change over time, respectively, for participants with childhood ADHD.

the latter reflecting a moderating effect between ADHD status and time in the prediction of the repeated symptom measure (see Figure 2a). An examination of the simple slopes revealed that probands exhibited a steeper rate of total symptom decline ($b = -0.81$, $p < 0.001$) than did comparison girls ($b = -0.11$, *ns*). To gauge the magnitude of this moderated effect, I supplemented these findings with an estimate of the effect size of symptom change between childhood and adulthood for each diagnostic group. Whereas girls with a childhood diagnosis of ADHD experienced a large decrease in total symptoms over time ($d = -1.29$), symptoms for girls without a childhood diagnosis decreased at a slower rate ($d = -0.36$). Although girls with a childhood diagnosis experienced significant (and large) reductions in total symptoms, they still exhibited nearly five times more symptoms in adulthood than girls without a childhood ADHD diagnosis, $M_{ADHD} = 1.30$, $M_{COMP} = 0.26$, $t(226) = 14.68$, $p < 0.001$, $d = 2.15$; see Figure 2d.

Inattentive symptoms. This baseline LGM also fit the data well, $\chi^2(5, N = 228) = 8.01$, $p = 0.16$, CFI = 0.996, NNFI = 0.987, RMSEA = 0.05, CI_{0.90} {0.00, 0.11}. The latent slope was significant and negative, with significant decreases in symptoms of inattention occurring across all developmental periods. Approximately 41% of the total reduction in inattentive symptoms occurred between childhood and early adolescence ($b_1 = 0.41$, $p < 0.01$), and an additional 32% between early adolescence and adolescence ($b_2 = 0.73$, $p < 0.001$). Childhood ADHD status was significantly related to initial symptom severity and exerted a significant moderating effect on rate of symptom change; girls with a childhood diagnosis displayed more symptoms of inattention in early childhood (estimate = 1.93, $p < 0.001$; $\beta = 0.93$) and experienced greater reductions in inattentive symptoms over time (estimate = -0.66, $p < 0.001$; $\beta = -0.45$; see Figure 2b) than comparison girls. An analysis of the simple slopes showed that inattention symptoms decreased significantly only for probands ($b = -0.65$, $p < 0.001$). Symptoms of inattention decreased by approximately 28% over time for girls with a childhood ADHD diagnosis ($d = -0.84$), but remained relatively stable for comparison girls ($d = 0.03$). Furthermore, probands displayed nearly four times the inattentive symptoms in adulthood as did girls without childhood ADHD, $M_{ADHD} = 1.70$, $M_{COMP} = 0.44$, $t(226) = 14.80$, $p < 0.001$, $d = 2.12$. Approximately 49% ($n = 68$) of probands' and 1.1% ($n = 1$) of comparison girls' adult symptom levels were suggestive of the presence of the inattentive subtype of ADHD according to *DSM-IV* criteria. See Figure 2e for distributions of inattentive symptom scores for proband and comparison women.

HI symptoms. This baseline LGM fit the data exceptionally well, $\chi^2(5, N = 228) = 4.20$, $p = 0.52$, CFI = 1.00, NNFI = 1.00, RMSEA = 0.00, CI_{0.90} {0.00, 0.08}. HI symptoms decreased sharply over time, with approximately 52% of symptom change occurring by early adolescence ($b_1 = 0.52$, $p < 0.001$) and an additional 44% occurring by adolescence ($b_2 = 0.96$, $p < 0.001$). Girls with childhood ADHD showed more severe HI symptoms in childhood (estimate = 1.59, $p < 0.001$; $\beta = 0.87$) and exhibited steeper rates of decline in symptoms over time than comparison girls (estimate = -0.79, $p < 0.001$; $\beta = -0.62$; see Figure 2c). However, simple slope analyses showed that HI symptoms decreased significantly for both the ADHD ($b = -0.98$, $p < 0.001$) and comparison groups ($b = -0.20$, $p < 0.05$); reductions in symptoms were more pronounced for probands ($d = -1.40$) than for girls without a childhood ADHD diagnosis ($d = -0.75$). Finally, young adult probands were characterized by approximately nine times the mean HI symptoms in adulthood than girls without a childhood ADHD diagnosis, $M_{ADHD} = 0.88$, $M_{COMP} = 0.10$, $t(226) = 10.67$, $p < 0.001$, $d = 1.48$. Nearly a quarter of the original ADHD sample ($n = 32$) had significantly elevated HI symptoms in adulthood, per *DSM-IV* criteria, whereas no comparison participants' symptom levels were reflective of the HI subtype. Distributions of probands' and comparisons' HI symptoms in adulthood are displayed in Figure 2f.

Predictive Effects of Symptom Severity and Rate of Symptom Change on Young Adult Outcomes

Next, I examined the predictive associations of latent intercept and slope factors within each symptom domain with respect to four young adult outcomes. Of the resulting 12 models, only one evidenced a marginal fit to the data: the Inattentive LGM predicting functional impairment. Thus, estimates from this particular model should be interpreted with caution. The remaining models fit the data exceptionally well, with all manifest indicators loading significantly onto their respective latent constructs (all z s > 3.46, all p s < 0.001). Additionally, childhood ADHD status was a significant predictor of symptom severity in childhood and rate of symptom change over time for all models (see detailed results above). Model-fit statistics and standardized path coefficients relating intercept and slope factors to developmental outcomes are shown in Table 2.

*Total symptoms: baseline severity*². For total ADHD symptoms, LGMs revealed that childhood symptom severity was positively and significantly associated with internalizing symptoms (estimate = 2.02, p < 0.001; β = 0.85), functional impairment (estimate = 0.94, p < 0.001; β = 1.11), and substance use (estimate = 0.32, p < 0.05; β = 0.23). However, initial symptom severity was only marginally predictive of risky sexual behaviors (estimate = 0.16, p = 0.09; β = 0.21). These estimates differed widely in their strength of association: baseline total symptoms were strongly predictive of functional impairment, but weakly so for substance use.

Total symptoms: rate of change. Positive associations were found between rate of total ADHD symptom change and internalizing symptoms (estimate = 1.94, p < 0.01; β = 0.53) and functional impairment (estimate = 0.92, p < 0.001; β = 0.71). A marginally significant association was found between rate of total symptom change and substance use (estimate = 0.58, p < 0.06; β = 0.26), but no relation was found between rate of symptom change and risky sexual behaviors (estimate = 0.19, p = 0.26; β = 0.15). These positive associations between (the negative) latent slope and developmental outcomes indicate that a *slower* decrease of ADHD symptoms over time predicted greater young adult maladjustment.

Inattentive and HI symptoms: Baseline severity. Severity of inattentive and HI symptoms in childhood were each predictive of internalizing symptoms (Inattentive estimate = 1.65, p < 0.001; β = 0.78; HI estimate = 1.87, p < 0.001; β = 0.74) and functional impairment (Inattentive estimate = 0.77, p < 0.01; β = 1.02; HI estimate = 0.92, p < 0.001; β = 1.00) by young adulthood. However, whereas initial levels of inattentive symptoms were associated with later substance use (estimate = 0.28, p < 0.05; β = 0.22) and were marginally associated with risky sexual behaviors (estimate = 0.15, p < 0.06; β = 0.21), HI symptom severity in childhood was not predictive of either of these two outcomes (substance use estimate = 0.24, p = 0.16, β = 0.18; risky sexual behavior estimate = 0.09, p = 0.33; β = 0.12). The severity of childhood symptoms of inattention and HI were equivalently predictive of affective difficulties and functional impairment, but childhood inattentive symptoms were more robust predictors of risky sexual behaviors and substance use than were HI symptoms.

² I interpreted significant pathways from the intercept of total symptoms to adult outcome to represent significant differences in outcome based upon initial ADHD status. Diagnostic status was determined based upon baseline symptom severity, and as such, the intercept factor and the childhood ADHD status variable represent a tautology. Indeed, separate models (not reported herein) that include a pathway from childhood ADHD status (instead of from the intercept) to adult outcomes yielded nearly identical model-fit statistics and standardized estimates. I focused on total symptoms because they include both HI and inattentive symptom estimates and are thereby elevated for all probands at baseline.

Inattentive and HI symptoms: Rate of change. The rates of inattentive and HI symptom change were each predictive of internalizing problems (Inattentive estimate = 1.51, $p < 0.01$; $\beta = 0.54$; HI estimate = 1.10, $p < 0.05$; $\beta = 0.29$) and functional impairment (Inattentive estimate = 0.75, $p < 0.001$; $\beta = 0.73$; HI estimate = 0.60, $p < 0.001$; $\beta = 0.42$). The standardized estimates for predictive effects of inattentive symptom change on both these outcomes were larger (nearly double) than those for HI symptom change. However, only inattentive symptom change was predictive of substance use (estimate = 0.54, $p < 0.05$; $\beta = 0.31$). There was no association between either inattentive or HI symptom change and risky sexual behaviors (Inattentive estimate = 0.18, $p = 0.20$; $\beta = 0.18$; HI estimate = 0.04, $p = 0.80$; $\beta = 0.04$).

Predictors of Adult ADHD Symptoms and Rate of Symptom Change

In the final set of analyses, I examined parent and child predictors of (a) ADHD symptoms in adulthood and (b) rate of symptom change across development. All analyses controlled for baseline ADHD status, and predictor and baseline status was allowed to co-vary in each model. All resulting models evidenced an adequate fit to the data. Estimates of model parameters and fit statistics are presented in Table 3.

Total Symptoms in Young Adulthood. The only parent variable measured at baseline that was predictive of ADHD symptoms in adulthood, controlling for baseline ADHD status, was maternal distress (estimate = 0.016, $p < 0.001$; $\beta = 0.18$). Maternal self-reported ADHD (estimate = 0.010, *ns*; $\beta = 0.04$) and depression (estimate = 0.011, *ns*; $\beta = 0.09$) symptoms were unrelated to offspring's adult symptom levels. However, all child psychopathology variables were predictive of symptoms of ADHD in adulthood. Baseline CBCL externalizing (estimate = 0.016, $p < 0.001$; $\beta = 0.30$) and aggressive behaviors (estimate = 0.025, $p < 0.001$; $\beta = 0.29$), as well as ODD symptoms (estimate = 0.025, $p < 0.001$; $\beta = 0.25$) were each predictive of increased ADHD symptoms in adulthood, controlling for baseline ADHD status. In addition, CBCL internalizing (estimate = 0.010, $p < 0.001$; $\beta = 0.18$) and anxiety/depression (estimate = 0.11, $p < 0.001$; $\beta = 0.16$) were predictive of greater numbers of ADHD symptoms in adulthood.

Rate of Total Symptom Change. Controlling for baseline ADHD status, no maternal variable was predictive of ADHD symptom change across development (see Table 3). However, CBCL externalizing problems (estimate = -0.011, $p < 0.05$; $\beta = -0.25$) and aggressive behaviors (estimate = -0.11, $p < 0.05$; $\beta = -0.22$) and ODD symptoms (estimate = -0.018, $p < 0.05$; $\beta = -0.22$) were all negatively related to the rate of symptom change. These findings indicate that ADHD total symptoms decreased more quickly across development for children with *higher* rates of externalizing and aggressive behaviors and ODD symptoms in childhood. CBCL internalizing and anxiety/depressive problems were unrelated to symptom change (see table 3).

Inattentive and HI Symptoms in Young Adulthood. Inattentive symptom severity in young adulthood, controlling for baseline ADHD status, was predicted by maternal depression (estimate = 0.023, $p < 0.05$; $\beta = 0.14$), maternal distress (estimate = 0.021, $p < 0.01$; $\beta = 0.20$), CBCL externalizing (estimate = 0.015, $p < 0.01$; $\beta = 0.22$), internalizing (estimate = 0.016, $p < 0.01$; $\beta = 0.22$), aggressive (estimate = 0.017, $p < 0.001$; $\beta = 0.23$), and anxiety/depressive problems (estimate = 0.16, $p < 0.01$; $\beta = 0.18$), as well as ODD symptoms (estimate = 0.015, $p < 0.01$; $\beta = 0.23$). Maternal ADHD symptoms were the only baseline measure not predictive of inattentive symptoms in adulthood. HI symptoms in adulthood, controlling for baseline ADHD status, were predicted by maternal distress (estimate = 0.011, $p < 0.05$; $\beta = 0.14$) and CBCL externalizing (estimate = 0.018, $p < 0.001$; $\beta = 0.26$) and aggressive problems (estimate = 0.020,

$p < 0.001$; $\beta = 0.35$) and ODD symptoms (estimate = 0.023, $p < 0.01$; $\beta = 0.25$) (see Table 3). Maternal ADHD and depressive symptoms, as well as all internalizing problems (CBCL internalizing and anxiety/depression) were unrelated to HI symptoms in adulthood. A review of effect size estimates indicated comparable effects of all predictor variables on inattentive and HI symptoms in adulthood.

Rate of Inattentive and HI symptom change. The only variable that affected the rate of inattentive symptom change across development, controlling for baseline ADHD status, was maternal depression (estimate = 0.22, $p < 0.07$; $\beta = 0.16$). That is, girls whose mothers experienced more depression at baseline evidenced a less steep reduction in inattentive symptoms across development (see interaction effect in Figure 4). Regarding HI symptom change, no maternal variables (ADHD and depressive symptoms and parental distress) predicted change across development. However, CBCL externalizing (estimate = -0.020, $p < 0.001$; $\beta = -0.43$) and aggressive behaviors (estimate = -0.021, $p < 0.001$; $\beta = 0.41$) and ODD symptoms (estimate = -0.032, $p < 0.001$; $\beta = -0.36$) all predicted differential rates of HI symptom change across development. Specifically, participants experiencing higher levels of these variables at baseline all experienced *steeper* rates of HI symptom decline across development, controlling for baseline ADHD status. As depicted in Figure 4, participants with more externalizing pathologies also had substantially more HI symptoms at baseline – a difference that remained significant, albeit attenuated, in young adulthood (see results above). Thus, the faster rate of HI symptom decline appears linked to the extreme differences in symptoms at baseline and does not reflect a *protective* effect of externalizing comorbidities on symptom change. All indicators of internalizing problems in childhood were unrelated to HI symptom change.

Discussion

In this dissertation study, I assessed a sample of girls with ADHD and matched comparison girls, ascertained carefully in childhood and followed prospectively into adolescence and young adulthood, to determine (a) the developmental course of ADHD symptoms into young adulthood (total, inattentive, and HI); (b) predictive associations between symptom severity in childhood (i.e., childhood diagnostic status) and rate of symptom change across development with respect to functional outcomes particularly relevant to young adult women; and (c) parent and child predictors of ADHD symptoms in adulthood and rate of symptom change across development. The sample was ethnically and socioeconomically diverse; the overall retention rate by young adulthood in this longitudinal investigation was exceptional.

First, as hypothesized—and consistent with past meta-analytic research involving primarily male participants (Faraone, Biederman, & Mick, 2006)—I found an age-dependent decline across both inattentive and HI symptom domains for women with childhood ADHD. Relative to inattentive symptoms, HI symptoms declined more precipitously, with a greater reduction in symptoms occurring earlier in development (see Biederman, Mick, & Faraone, 2000). Whereas 96% of the total reduction in HI symptoms occurred by the time the sample reached adolescence (*Mdn* age = 16 years), only 73% of total inattentive change transpired by this developmental stage. The greater overall decline in HI symptoms (compared to inattentive symptoms) is convergent with results derived from both child clinical (Hart et al., 1995) and epidemiologically-derived adult samples (Kessler et al., 2010). In addition, a significant reduction in HI symptoms occurred for both ADHD and comparison participants, reflecting normative developmental declines in hyperactivity (see Hart et al., 1995). However, a significant

reduction in inattentive symptoms was observed only among probands. These findings represent the first time symptom clusters of ADHD have been modeled as continua in females into young adulthood utilizing latent growth curve models.

Critically, despite a substantial abatement of symptoms over time, probands continued to be characterized by four to 11 times the mean level of ADHD symptoms in young adulthood, relative to comparisons. Mean differences were extremely large for both inattentive ($d = 2.12$) and HI ($d = 1.48$) symptom levels. However, despite these pronounced differences in adulthood, only a minority of probands' symptom levels surpassed criteria designed to identify significantly impairing problems of inattention ($n = 68$; $\sim 49\%$) and hyperactivity/impulsivity ($n = 32$; $\sim 23\%$), at least by *DSM* standards (see also Hinshaw et al., 2011). The discrepancy between (a) heightened symptom levels and (b) the relatively small proportion of the proband sample that exceeded established cut-off criteria raises questions about the sensitivity of current *DSM* criteria to accurately identify probable cases of adult ADHD. This observation is bolstered by examination of the distributions of HI and inattentive symptoms in adulthood for probands and comparison participants (see Figure 2E – F), which reveals that these two groups continue to be distinct, with only a small percentage of overlap occurring in the tails of their distributions.

Estimates of the persistence of ADHD into adulthood vary widely based upon definitions of persistence employed across studies (Barkley et al., 2002; Biederman, et al., 2000; Faraone et al., 2006; see also McGough & Barkley, 2004). For instance, Barkley and colleagues (2002) have argued that the application of a fixed '6 of 9' symptom cutoff across all age groups is inappropriate, as it is not sensitive to the natural attenuation of ADHD symptoms over time that characterizes normal development. They suggest that these static diagnostic thresholds be replaced by a developmentally-referenced criterion of +2 SDs (i.e., $> \sim 98^{\text{th}}$ percentile) above the normal adult mean for any given age group (Barkley et al., 2002). These findings support this developmental rationale with respect to HI symptoms but question its applicability to inattentive symptoms. Caregiver-reported HI symptom levels for comparison girls were near zero, suggesting that the presence of even minimal HI symptoms in adulthood may constitute psychopathology. However, comparison participants' symptom levels of inattention remained virtually unchanged over time, meaning that established *DSM* inattentive subtype criteria may be at least somewhat developmentally appropriate for adults when cases are defined as statistical deviations from the normative range.

Still, these findings do not address the face validity of the current *DSM* symptoms for adults; it is unlikely that symptoms originally identified as reflective of childhood manifestations of the disorder best characterize its expression in later years (see Barkley et al., 2008 and McGough & Barkley, 2004 for discussion). Furthermore, this study did not consider the symptom count criterion that optimally captures meaningful instances of maladjustment. Biederman and colleagues (2000) showed that the functional impairments associated with ADHD remit at a slower rate than its symptoms. Other investigators have suggested that only four symptoms in adulthood are needed to distinguish between persons with and without impairment (Heiligenstein, Conyers, Berns, Miller, & Smith, 1998; Kooij, Buitelaar, van den Oord, Furer, Rijnders, & Hodiament, 2005). Clearly, much work remains to arrive at a better conceptualization of adult ADHD (McGough & Barkley, 2004; McGough & McCracken, 2006).

Of importance, results showed that young adult women with a childhood ADHD diagnosis, relative to comparison females, continue to show statistically significant and clinically meaningful impairments across important life domains, including more symptoms of internalizing pathologies (depression & anxiety constellations), decrements in day-to-day

functioning, and increased tobacco use (the only ‘type’ of substance use yielding a significant difference for the manifest variables included in these analyses). These findings are partially concordant with earlier prospective reports (Babinski et al., 2011; Biederman et al., 2010) documenting increased lifetime and 1-year risk for psychiatric illness, poorer self-esteem, and worse overall job and academic functioning amongst adult women with childhood ADHD. They also corroborate epidemiological findings of the increased risk of adults with ADHD for mood, anxiety, and substance use disorders and the heightened functional, work, and social impairments they experience (Kessler et al., 2006). Overall, these findings underscore the profound negative ramifications girls with childhood ADHD experience as they age (for data on additional domains of impairment, see Hinshaw et al., 2011).

The largest predictive associations were related to the latent construct of functional impairment, signifying that childhood ADHD results in considerable difficulties across important life domains in adulthood. Crucially, per Biederman et al. (2000), the increased dysfunction characteristic of probands occurred in the context of significant rates of symptom decline and syndrome remission. The clinical implications of this observation are noteworthy, suggesting that an increased emphasis be placed on current adult functioning (versus symptom counts per se) when considering ADHD in adulthood, particularly when considering previously undiagnosed cases.

Some differences emerged with regard to the predictive associations of inattentive and HI symptoms in childhood—and their rates of change across development—on adult functioning and psychopathology. First, comparisons of effect sizes indicated that there were no meaningful differences between the ability for childhood inattentive and HI symptoms in isolation or combined to predict affective symptoms or functional impairment in adulthood. These observations mirror earlier reports on this sample (Hinshaw, 2002; Hinshaw et al., 2006). However, the effects of *rate of change* for inattentive symptoms on both internalizing symptoms and impairment were larger than for HI symptoms, indicating that the remission of inattentive symptoms over time had more predictive power related to positive adjustment than did decreases in HI symptoms. Somewhat counter-intuitively, only inattentive symptoms in childhood—and the rate these symptoms changed over time—were predictive of the latent construct of substance use in young adulthood. These findings corroborate previous research indicating the importance of inattentive symptoms with respect to later substance use disorders (Glass & Flory, in press; Molina & Pelham, 2003).

What might account for the unique influence of inattentive symptoms on later maladjustment? One possibility is co-occurring deficits in executive functioning (EF). In some research inattentive symptoms are uniquely predictive of EF deficits (Chhabildas, Pennington, & Willcutt, 2001). Such EF deficits, in turn, are associated with a host of negative developmental outcomes, including poor academic achievement, social functioning, and impairment (e.g., Miller & Hinshaw, 2010; see also Barkley, 1997). Indeed, Thorell (2007) showed that executive (dys)function partially mediates the relationship between symptoms of inattention and early academic skills, providing support for a model of ADHD development containing a distinct cognitive–executive function pathway. Extending these findings into additional domains of maladjustment, particularly those including affective and/or motivational components, represents an important area for future research. Implications for intervention and prevention strategies are also noteworthy: preliminary evidence suggests that even preschool-aged children can be trained to improve visual- and spatial-working memory, which may transfer into improvements in attention (Thorrell, Lindqvist, Nutley, Bohlin, & Klingberg, 2009).

Contrary to my predictions and to past findings (e.g., Flory et al., 2006), I did not find strong statistical evidence that probands engaged in higher rates of risky sexual behaviors in adulthood than did comparison participants. Marginally significant associations were found between baseline total ($p < 0.09$) and inattentive symptoms ($p < 0.06$), although baseline HI symptoms and the rates of change of either symptom cluster were not related to this outcome. A possible explanation is that these data were obtained via self-report and thus subject to social desirability biases. For instance, the ubiquity of the double-standard regarding gender and sexuality and sexual permissiveness (see Crawford & Popp, 2003) may have motivated the exclusively female sample to underreport certain behaviors (e.g., number of sexual partners). Methodological improvements including the use of objective measures (e.g., hospital records regarding STDs, pregnancies) and multiple-informants are worth pursuing. Additionally, I focused on only a select few indicators of risky sexual behaviors; additional domains (e.g., number of pregnancies, STD rates, violent sexual encounters, sexual contact under the influence) should be considered.

Third, I found several parent and child predictors of ADHD symptoms in young adulthood. Consistent with past findings regarding the persistence of ADHD into adolescence in young adulthood amongst males (Biederman et al., 1996; Biederman et al., 2011), I found that parental psychopathology (depression), parental distress, and child psychopathology, including anxiety/depression, aggression, and ODD symptoms, were each independently predictive of increased ADHD symptoms in young adulthood, after controlling for baseline ADHD status. These findings extend previous research that found that behavioral problems in girls were associated with both worse psychosocial adjustment and academic achievement (Lee & Hinshaw, 2006) and the persistence of ADHD into adolescence (Mick et al., 2010). Importantly, these predictive associations survived strict statistical control of baseline ADHD status, signifying direct, specific effects of comorbidities on symptoms of ADHD in adulthood that function independently of core baseline symptoms (see Kessler et al., 2005). However, contrary to study hypotheses and past findings with boys (Biederman et al., 1996), familiarity of ADHD (at least as measured herein with symptom counts on the CAARS) was unrelated to ADHD symptoms into adulthood.

The predictive significance of child externalizing psychopathology on adult ADHD symptoms for girls corroborates previous findings derived from exclusively male samples. Hart and colleagues (1995) demonstrated that ADHD persistence at four-year follow-up was predicted by comorbid conduct problems at baseline. Similarly, previous work has shown that early aggressive behavior is a potent predictor of ADHD persistence into later years (Biederman et al., 1996; Taylor et al., 1991; Molina et al., 2008). This study replicated these findings and extended them to include internalizing problems as predictors of ADHD into adulthood for females. In a prospective sample, Mick and colleagues (2010) found that CBCL behavioral problems (both externalizing and internalizing problems) differentiated girls whose ADHD status persisted into adolescents from girls whose ADHD had remitted. The findings reported herein extend these results into adulthood (also see Biederman et al., 2011, for similar findings regarding ADHD-persistence into adulthood for males). In addition, these prospective findings are convergent with retrospective accounts of lifetime estimates of psychiatric comorbidities in adults with ADHD (Kessler et al., 2005).

In addition to child psychopathology, these results indicate that exposure to maternal psychopathology (depression) and parental stress were predictors of ADHD symptoms in adulthood, although maternal ADHD symptoms were not. This latter finding was surprising as it

is discrepant from previous research (Biederman et al., 1996; Faraone et al., 2000) that has shown ADHD persistence can be predicted by familiarity of ADHD. A possible explanation of this discrepancy is that previous research did not control for ADHD symptoms at baseline (as in this study), raising the possibility that these previously documented associations are mediated by childhood ADHD status. Thus, family ADHD may confer risk for adult-persistent ADHD via more severe cases of childhood ADHD. Regardless, these findings do replicate results from previous investigations (Biederman et al., 1996; Biederman et al., 2011), underscoring the importance of additional familial factors, both genetic and psychosocial, in predicting ADHD symptoms across development.

The mechanism(s) responsible for the effects of maternal depression during childhood on later adult symptoms cannot be ascertained from this study. One such possibility, however, is that depression impairs parenting skills and contributes to increased family discord and/or negative parent-child interactions (Goodman & Gotlib, 1999). In this study, maternal stress during childhood was associated with later adult symptoms, lending partial support for this hypothesis. Future research examining the mediating role of parental resources and skills on the relation between maternal depression and later ADHD symptoms may help address this possibility. In addition, maternal depression could confer direct risk via a genetic predisposition to psychopathology more broadly, or through genetic effects on the social environment (see Kendler, 2001).

A distinguishing characteristic of this study from past research is that baseline predictors of adult ADHD symptoms were examined across each ADHD symptom cluster. Whereas baseline parental distress and child externalizing pathologies were found to be equally predictive of both inattentive and HI symptoms in young adulthood, maternal depression and baseline child internalizing psychopathology were uniquely predictive of inattentive symptoms in young adulthood. These results highlight the importance of recognizing and treating comorbidities in early preventive and intervention efforts regarding children with ADHD. The significance of maternal depression and childhood internalizing pathologies is particularly noteworthy, given the present findings as well as past results (e.g., Molina & Pelham, 2003) revealing that inattentive symptoms confer greater risk for later maladjustment than do HI symptoms. Thus, it is important that internalizing problems during childhood not be dismissed as demoralization secondary to ADHD in at-risk females (see Faraone & Biederman, 1997).

Few significant relationships were found for predictors of rate of symptom change across development. Externalizing pathologies were associated with greater HI symptom reduction across time; however, this effect should not be interpreted as a protective effect as externalizing problems were also associated with increased symptom levels in adulthood. Study participants with comorbid externalizing pathologies evidenced differences in HI symptom levels in childhood that far exceeded their differences in adulthood. Thus, the more precipitous decline in ADHD symptoms for this group may well represent regression to the mean. A marginal association was found between maternal depression and rate of inattentive symptom change across time, such that children whose mothers experienced more symptoms of depression evidenced a slower reduction in symptoms across time. Thus, maternal depression exerts not only a direct effect on symptoms in adulthood but also on the rate symptoms change across time. As previously noted, future research should consider the mechanisms underlying the link between maternal depression and later ADHD.

Several methodological limitations need to be considered. First, this sample was recruited from pediatric and community referrals and therefore cannot be considered representative of the

larger US population. However, the inclusion of socioeconomically and ethnically diverse participants increases generalizability. Second, the modeling of ADHD symptoms was obtained via caregiver report on a rating scale. Although the modeling of symptoms as continua allowed for the detection of effects that may otherwise have gone unnoticed, modeling ADHD symptoms categorically (i.e., as absent vs. present) may facilitate addressing the crucial issue of the persistence of ADHD into adulthood for females. Moreover, this analysis did not consider predictors of persistence from the perspective of which participants with childhood ADHD continued to have ADHD as adults, but rather focused on mean symptom levels across study participants in adulthood. Future research should examine this relationship utilizing categorical outcomes. Third, a sizable portion of our sample was either currently taking stimulant medications (~22%); even more had used them at some point since adolescence (see Hinshaw et al., 2011). We cannot therefore discuss the “natural” developmental course of ADHD, uninfluenced by medication. However, a completely unmedicated sample would not be generalizable, particularly given ever-increasing medication rates for youth with ADHD across the U.S. (Visser et al., 2010). Fourth, I had several instances of missing data that were handled by maximum-likelihood estimation procedures. Yet results of preliminary analyses utilizing only cases with complete data yielded an identical pattern of findings, with comparable effect sizes, increasing confidence in these findings. Finally, I did not include any statistical controls that might be predictive of proband-comparison differences (e.g., childhood comorbidities, medication use). I did not include these variables because of the desire to preserve an adequate variable-to-case ratio required of structural equation models (Kline, 2005). Employing alternative analytic strategies that do not place these restrictions on the data will allow these questions to be addressed. For data on adult impairment in the sample with stringent statistical control of baseline confounders as well as ongoing medication use, see Hinshaw et al. (2011).

Overall, girls with childhood ADHD experience continued maladjustment as adults, including higher levels of ADHD and internalizing symptoms, functional impairments, and tobacco use than comparison participants. Inattentive symptomatology was especially salient for these outcomes, as opposed to the more visible and disruptive cluster of HI symptoms. These findings extend previous work, which has demonstrated that inattentive symptoms, but not HI symptoms, were predictive of functional outcomes at the five-year adolescent follow-up (Lee & Hinshaw, 2006, in this case regarding academic functioning). Early inattention is also a potent predictor of subsequent academic impairments (see Hinshaw, 1992). In addition, adult symptoms of ADHD were predicted by comorbidities in childhood, maternal psychopathology, and parental stress. These findings should help alert clinicians of the crucial importance that these modifiable risk factors play in shaping the course of ADHD across development when developing intervention strategies and predicting patient progress. Additionally, they underscore the importance of treatments targeting inattentive symptoms early in development. A number of promising psychosocial treatments have recently been developed that have shown to be effective in addressing symptoms of inattention and associated problem areas in children with ADHD (e.g., Pfiffner et al., 2007). It is imperative that researchers continue to innovate novel treatment approaches that target symptoms of inattention in order to mitigate future maladjustment.

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Tables

Table 1. Descriptive statistics and comparisons of outcome variables by childhood ADHD status

Variable	ADHD	Comparison	<i>t</i> (df)	<i>D</i>
	M (SD)	M (SD)		
ADHD Symptoms				
Total	1.32 (0.72)	0.25 (0.30)	11.95 (173)***	1.86
Inattention	1.74 (0.83)	0.39 (0.49)	12.33 (173)***	1.91
Hyperactivity/Impulsivity	0.89 (0.75)	0.10 (0.19)	8.65 (173)***	1.34
Internalizing Symptoms				
ASR DSM-Depression	60.28 (9.09)	52.25 (3.99)	6.92 (173)***	1.08
ASR DSM-Anxiety	56.73 (8.10)	52.49 (4.16)	4.01 (173)***	0.62
Self-esteem	30.54 (6.41)	32.42 (6.30)	-2.08 (204)*	-0.30
Functional Impairment				
Staff-rated GAF	65.33 (11.94)	79.95 (10.36)	-7.95 (157)***	-1.30
ASR Total Problems	59.32 (10.47)	43.49 (10.36)	9.88 (173)***	1.54
CIS	1.42 (0.84)	0.48 (0.52)	8.35 (176)***	1.30
Risky Sexual Behaviors				
Age of First Intercourse	6.67 (1.96)	6.93 (1.77)	-0.95 (195)	-0.14
Frequency of Condom Use	3.09 (1.42)	3.29 (1.37)	-0.99 (195)	-0.14
Total Sexual Partners	4.02 (4.75)	3.79 (4.40)	0.34 (194)	0.07
Substance Use				
Tobacco	1.65 (2.34)	0.84 (1.56)	2.81 (208)**	0.40
Alcohol	3.26 (2.67)	3.72 (2.36)	-1.28 (207)	-0.18
Marijuana	2.51 (3.52)	2.53 (3.08)	-0.04 (203)	0.00
Other	0.16 (0.44)	0.11 (0.30)	0.96 (208)	0.13

Note. Sample size differs for each comparison due to missing data.

GAF = Global Assessment of Functioning; ASR = Adult Self-report; CIS = Columbia Impairment Scale; DSM = Diagnostic and Statistical Manual

* $p < 0.05$, ** $p < 0.01$ *** $p < 0.001$.

Table 2. Predictive effects of ADHD symptom severity in childhood and rate of symptom change on maladaptive outcomes in young adulthood for girls with ($n = 140$) and without ($n = 88$) childhood ADHD.

Latent Outcome	Predictors					
	Total Symptoms		Inattentive Symptoms		HI Symptoms	
	Intercept	Slope	Intercept	Slope	Intercept	Slope
1. Internalizing Symptoms	2.02*** <i>0.51</i>	1.94** <i>0.59</i>	1.65*** <i>0.42</i>	1.51** <i>0.47</i>	1.87*** <i>0.50</i>	1.10* <i>0.49</i>
2. Functional Impairment	0.94*** <i>0.06</i>	0.92*** <i>0.71</i>	0.77*** <i>0.06</i>	0.75*** <i>0.10</i>	0.92*** <i>0.07</i>	0.60*** <i>0.11</i>
3. Risky Sexual Behavior	0.55 [†] <i>0.33</i>	0.70 <i>0.61</i>	0.51 [†] <i>0.27</i>	0.63 <i>0.50</i>	0.31 <i>0.33</i>	0.16 <i>0.53</i>
4. Substance Use	0.32* <i>0.16</i>	0.58 [†] <i>0.31</i>	0.28* <i>0.14</i>	0.54* <i>0.25</i>	0.24 <i>0.17</i>	0.32 <i>0.28</i>

Note. Childhood ADHD status was included as a predictor of latent intercept and slope factors. Values in plain font are unstandardized path coefficients; values in italics are standard errors.

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

¹ Total fit: $X^2(18, N=228) = 17.38$, $p=0.50$; $CFI = 1.000$; $NNFI = 1.000$; $RMSEA = 0.000$, {0.000, 0.057}.
Inattentive fit: $X^2(18, N=228) = 16.42$, $p=0.56$; $CFI = 1.000$; $NNFI = 1.000$; $RMSEA = 0.000$, {0.000, 0.052}.
HI fit: $X^2(18, N=228) = 21.47$, $p=0.26$; $CFI = 0.995$; $NNFI = 0.989$; $RMSEA = 0.029$, {0.000, 0.069}.
Indicators (3) include: Adult Self-report DSM-Depression and DSM-Anxiety scales; Rosenberg Self-Esteem Scale (reverse-scored).

² Total fit: $X^2(18, N=228) = 27.94$, $p=0.06$; $CFI = 0.992$; $NNFI = 0.984$; $RMSEA = 0.049$, {0.000, 0.083}.
Inattentive fit: $X^2(18, N=228) = 31.44$, $p=0.03$; $CFI = 0.989$; $NNFI = 0.977$; $RMSEA = 0.057$, {0.020, 0.090}.
HI fit: $X^2(18, N=228) = 24.42$, $p=0.14$; $CFI = 0.993$; $NNFI = 0.987$; $RMSEA = 0.040$, {0.000, 0.076}.
Indicators (3) include: Staff rated GAF (reverse-scored); Adult Self-report Total Problems; Columbia Impairment Scale.

³ Total fit: $X^2(18, N=228) = 19.41$, $p=0.37$; $CFI = 0.998$; $NNFI = 0.996$; $RMSEA = 0.019$, {0.000, 0.063}.
Inattentive fit: $X^2(18, N=228) = 28.00$, $p=0.06$; $CFI = 0.987$; $NNFI = 0.974$; $RMSEA = 0.049$, {0.000, 0.083}.
HI fit: $X^2(18, N=228) = 12.94$, $p=0.80$; $CFI = 1.00$; $NNFI = 1.00$; $RMSEA = 0.000$, {0.000, 0.039}.
Indicators (3) include: Age of losing virginity; Frequency of condom use; Total sexual partners

⁴ Total fit: $X^2(25, N=228) = 27.93$, $p=0.31$; $CFI = 0.996$; $NNFI = 0.993$; $RMSEA = 0.023$, {0.000, 0.060}.
Inattentive fit: $X^2(25, N=228) = 35.27$, $p=0.08$; $CFI = 0.987$; $NNFI = 0.976$; $RMSEA = 0.043$, {0.000, 0.073}.
HI fit: $X^2(25, N=228) = 28.02$, $p=0.31$; $CFI = 0.995$; $NNFI = 0.991$; $RMSEA = 0.023$, {0.000, 0.060}.
Indicators (4) include: Self-reported frequency of tobacco, alcohol, marijuana, and other drug use.

Table 3. Predictive effects of child- and mother-level variables on ADHD symptom severity in young adulthood and rate of symptom change across development for girls with ($n = 140$) and without ($n = 88$) childhood ADHD.

Predictors	Total		Inattentive		HI	
	Intercept	Slope	Intercept	Slope	Intercept	Slope
Maternal Variables						
1. ADHD Symptoms	0.10 <i>0.17</i>	-0.28 <i>0.20</i>	0.20 <i>0.22</i>	-0.22 <i>0.26</i>	0.02 <i>0.17</i>	-0.32 <i>0.02</i>
2. Depression Symptoms	0.11 <i>0.08</i>	0.04 <i>0.09</i>	0.23* <i>0.10</i>	0.22 [†] <i>0.12</i>	0.04 <i>0.08</i>	-0.10 <i>0.01</i>
3. PSI: Distress	0.16** <i>0.05</i>	0.03 <i>0.06</i>	0.21** <i>0.06</i>	0.13 <i>0.08</i>	0.11* <i>0.05</i>	-0.07 <i>0.06</i>
Child Variables						
Externalizing Problems						
4. CBCL Externalizing	0.16*** <i>0.04</i>	-0.11* <i>0.05</i>	0.15** <i>0.06</i>	-0.03 <i>0.07</i>	0.18*** <i>0.04</i>	-0.20*** <i>0.05</i>
5. CBCL Aggressive	0.18*** <i>0.04</i>	-0.11* <i>0.05</i>	0.17*** <i>0.06</i>	0.00 <i>0.07</i>	0.20*** <i>0.04</i>	-0.21*** <i>0.05</i>
6. ODD Symptoms	0.25*** <i>0.07</i>	-0.18* <i>0.09</i>	0.29** <i>0.10</i>	-0.02 <i>0.12</i>	0.23** <i>0.07</i>	-0.32*** <i>0.09</i>
Internalizing Problems						
7. CBCL Internalizing	0.10** <i>0.04</i>	-0.05 <i>0.05</i>	0.16** <i>0.05</i>	-0.02 <i>0.06</i>	0.05 <i>0.04</i>	-0.07 <i>0.05</i>
8. CBCL Anxious/Depression	0.11* <i>0.05</i>	-0.05 <i>0.05</i>	0.16** <i>0.06</i>	-0.001 <i>0.07</i>	0.07 <i>0.05</i>	-0.09 <i>0.06</i>

Note. Childhood ADHD status was included as a predictor of latent intercept and slope factors. Values in plain font are unstandardized path coefficients; values in italics are standard errors. All values are 10^{-1} .

PSI = Parenting Stress Index; CBCL = Child Behavior Checklist; ODD = Oppositional Defiant Disorder; DBR = Daily Behavior Ratings; CDI = Child Depression Inventory

[†] $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

¹ Total fit: $X^2(7, N=228) = 7.00$, $p=0.43$; $CFI = 1.000$; $NNFI = 1.000$; $RMSEA = 0.000$, {0.000, 0.082}.

Inattentive fit: $X^2(7, N=228) = 9.17$, $p=0.25$; $CFI = .997$; $NNFI = 0.991$; $RMSEA = 0.037$, {0.000, 0.095}.

HI fit: $X^2(7, N=228) = 6.85$, $p=0.44$; $CFI = 1.000$; $NNFI = 1.000$; $RMSEA = 0.000$, {0.000, 0.081}.

² Total fit: $X^2(7, N=228) = 9.43$, $p=0.22$; $CFI = 0.997$; $NNFI = 0.990$; $RMSEA = 0.039$, {0.000, 0.096}.

Inattentive fit: $X^2(7, N=228) = 11.52$, $p=0.12$; $CFI = .993$; $NNFI = 0.980$; $RMSEA = 0.053$, {0.000, 0.106}.

HI fit: $X^2(7, N=228) = 8.29$, $p=0.31$; $CFI = 0.997$; $NNFI = 0.992$; $RMSEA = 0.028$, {0.000, 0.090}

³ Total fit: $X^2(7, N=228) = 5.45$, $p=0.61$; $CFI = 1.000$; $NNFI = 1.000$; $RMSEA = 0.000$, {0.000, 0.070}.

Inattentive fit: $X^2(7, N=228) = 9.21$, $p=0.24$; $CFI = 0.997$; $NNFI = 0.991$; $RMSEA = 0.037$, {0.000, 0.095}.

HI fit: $X^2(7, N=228) = 7.47$, $p=0.38$; $CFI = 0.999$; $NNFI = 0.997$; $RMSEA = 0.017$, {0.000, 0.085}.

⁴ Total fit: $X^2(7, N=228) = 9.30$, $p=0.23$; $CFI = 0.998$; $NNFI = 0.993$; $RMSEA = 0.038$, {0.000, 0.095}.

Inattentive fit: $X^2(7, N=228) = 12.66$, $p=0.08$; $CFI = 0.993$; $NNFI = 0.980$; $RMSEA = 0.059$, {0.000, 0.111}.

HI fit: $X^2(7, N=228) = 7.49$, $p=0.38$; $CFI = 0.999$; $NNFI = 0.998$; $RMSEA = 0.018$, {0.000, 0.085}.

Table 3 (continued). Predictive effects of child- and mother-level variables on ADHD symptom severity in young adulthood and rate of symptom change across development for girls with ($n = 140$) and without ($n = 88$) childhood ADHD.

⁵ Total fit: $X^2(7, N=228) = 8.00$, $p=0.33$; $CFI = 0.999$; $NNFI = 0.997$; $RMSEA = 0.025$, {0.000, 0.088}.
 Inattentive fit: $X^2(7, N=228) = 10.57$, $p=0.16$; $CFI = 0.996$; $NNFI = 0.987$; $RMSEA = 0.047$, {0.000, 0.102}.
 HI fit: $X^2(7, N=228) = 9.02$, $p=0.25$; $CFI = 0.997$; $NNFI = 0.992$; $RMSEA = 0.036$, {0.000, 0.094}.

⁶ Total fit: $X^2(7, N=228) = 5.29$, $p=0.62$; $CFI = 1.000$; $NNFI = 1.005$; $RMSEA = 0.000$, {0.000, 0.068}.
 Inattentive fit: $X^2(7, N=228) = 8.90$, $p=0.26$; $CFI = 0.998$; $NNFI = 0.993$; $RMSEA = 0.035$, {0.000, 0.093}.
 HI fit: $X^2(7, N=228) = 4.41$, $p=0.73$; $CFI = 1.000$; $NNFI = 1.016$; $RMSEA = 0.000$, {0.000, 0.060}.

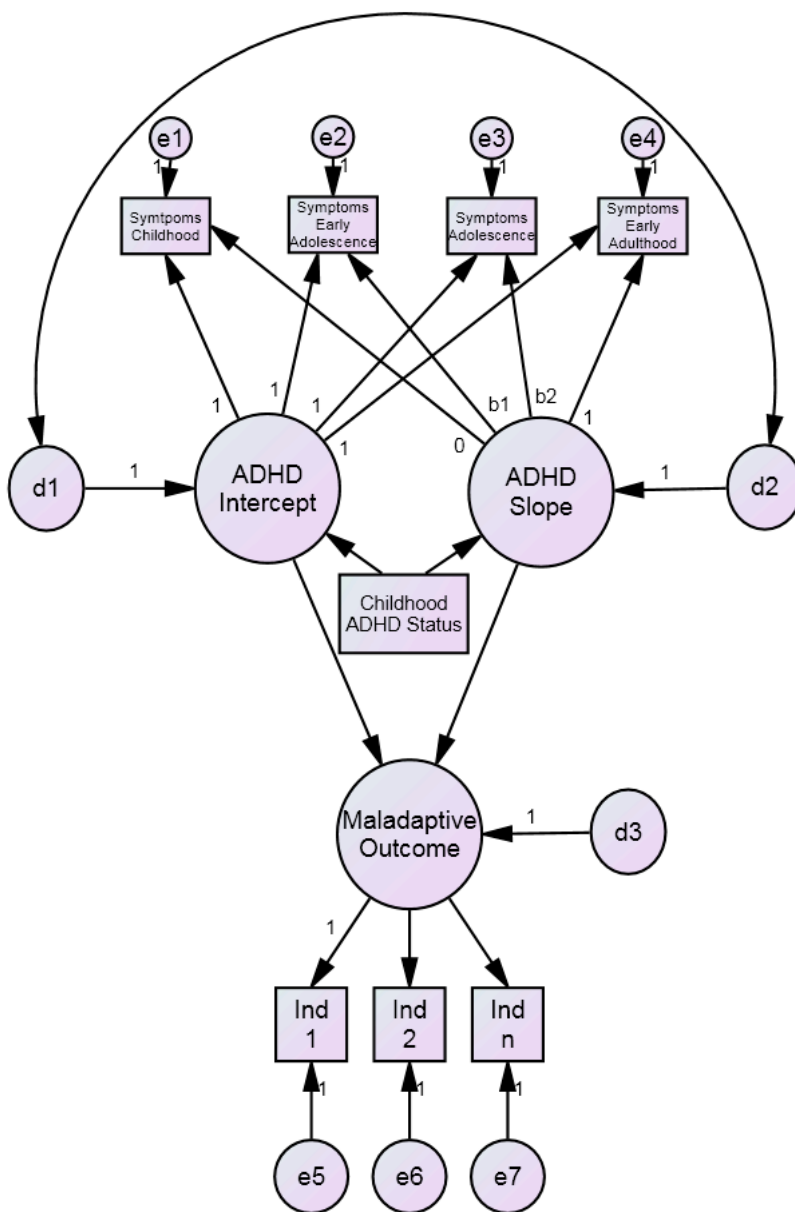
⁷ Total fit: $X^2(7, N=228) = 4.51$, $p=0.72$; $CFI = 1.000$; $NNFI = 1.009$; $RMSEA = 0.000$, {0.000, 0.061}.
 Inattentive fit: $X^2(7, N=228) = 8.38$, $p=0.30$; $CFI = 0.998$; $NNFI = 0.995$; $RMSEA = 0.029$, {0.000, 0.090}.
 HI fit: $X^2(7, N=228) = 4.41$, $p=0.73$; $CFI = 1.000$; $NNFI = 1.016$; $RMSEA = 0.000$, {0.000, 0.060}.

⁸ Total fit: $X^2(7, N=228) = 6.44$, $p=0.49$; $CFI = 1.000$; $NNFI = 1.003$; $RMSEA = 0.000$, {0.000, 0.078}.
 Inattentive fit: $X^2(7, N=228) = 8.33$, $p=0.30$; $CFI = 0.998$; $NNFI = 0.995$; $RMSEA = 0.029$, {0.000, 0.090}.
 HI fit: $X^2(7, N=228) = 4.83$, $p=0.68$; $CFI = 1.000$; $NNFI = 1.008$; $RMSEA = 0.000$, {0.000, 0.064}.

⁹ Total fit: $X^2(7, N=228) = 8.50$, $p=0.29$; $CFI = 0.998$; $NNFI = 0.994$; $RMSEA = 0.031$, {0.000, 0.091}.
 Inattentive fit: $X^2(7, N=228) = 11.70$, $p=0.11$; $CFI = 0.993$; $NNFI = 0.980$; $RMSEA = 0.054$, {0.000, 0.107}.
 HI fit: $X^2(7, N=228) = 7.53$, $p=0.38$; $CFI = 0.999$; $NNFI = 0.998$; $RMSEA = 0.018$, {0.000, 0.085}.

Figures

Figure 1.



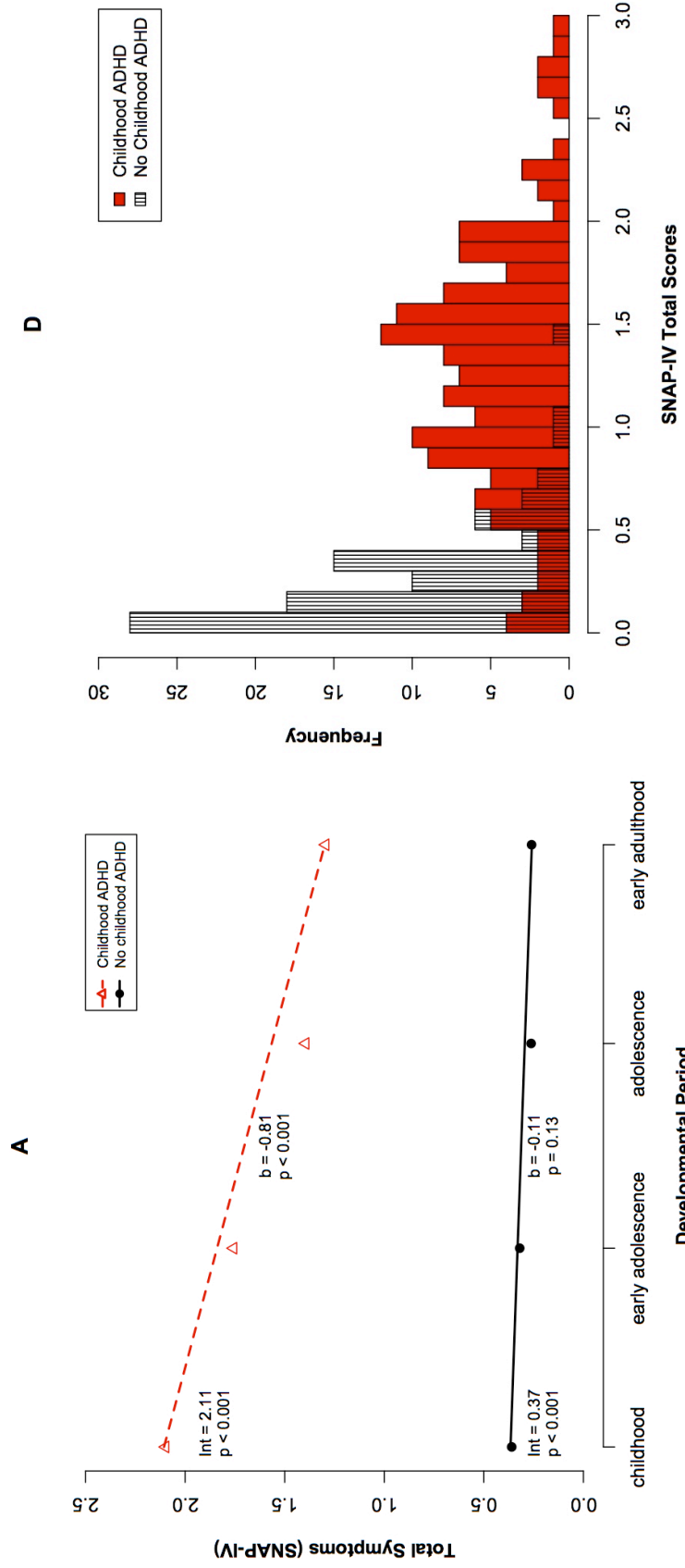


Figure 2.

Figure 2 (continued).

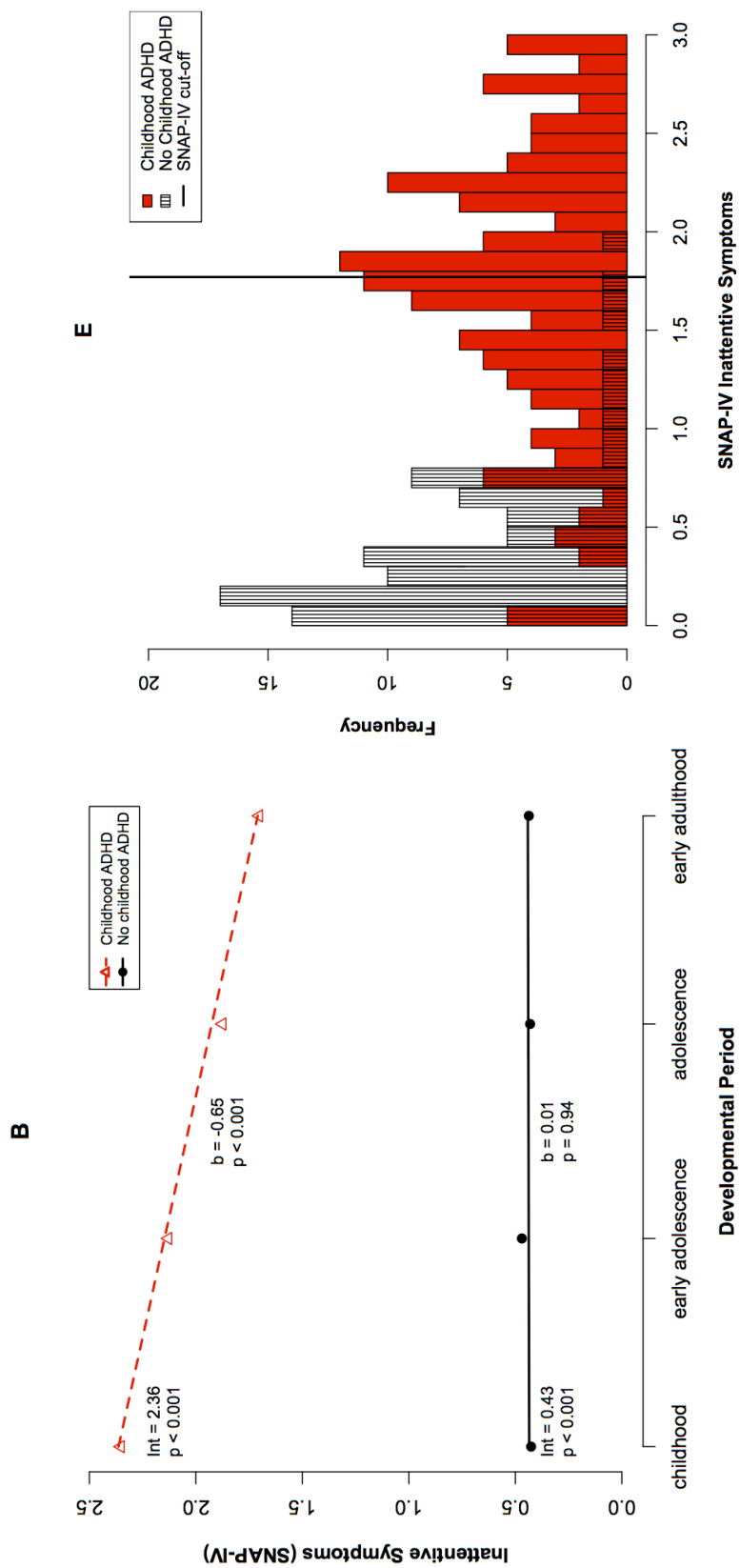


Figure 2 (continued)

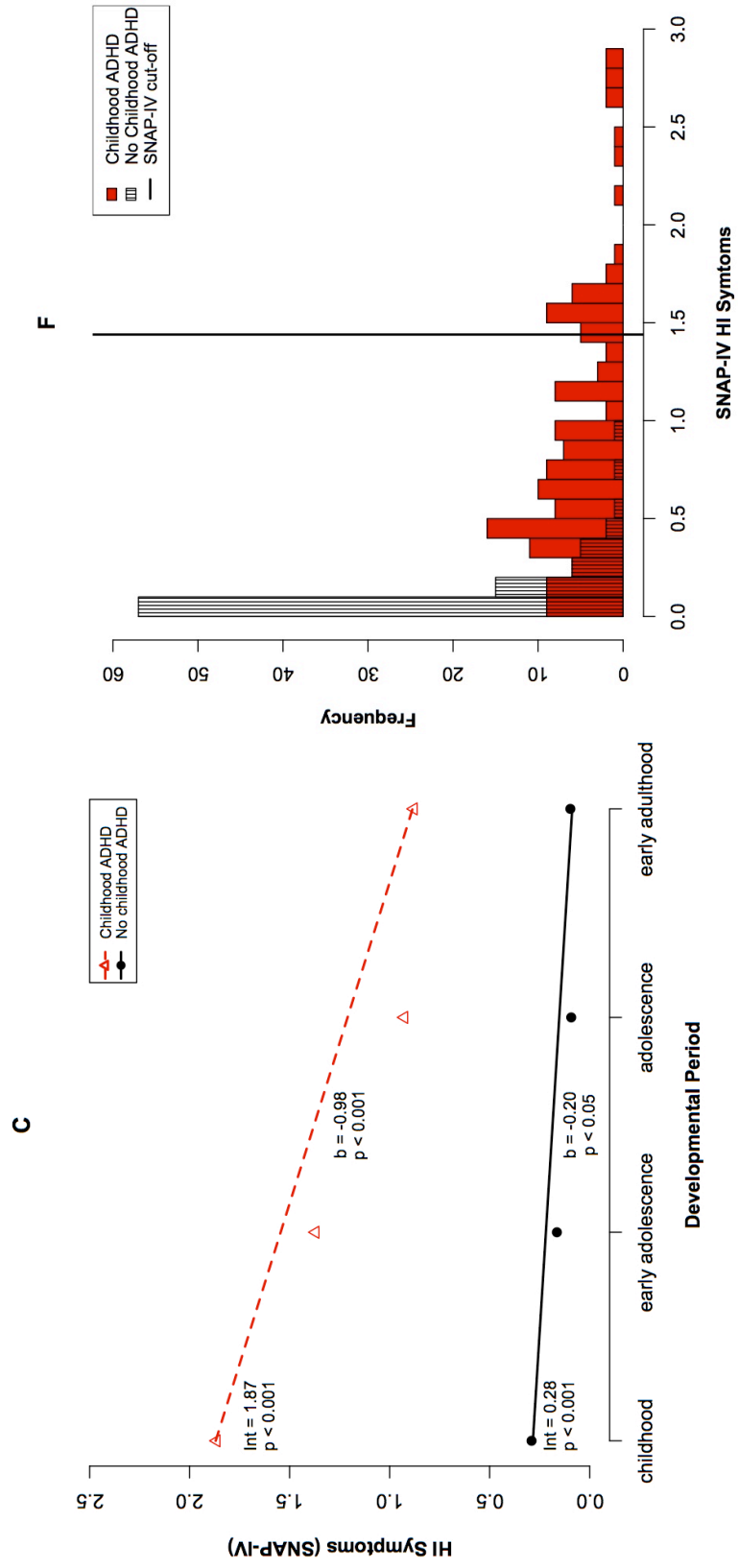


Figure 3.

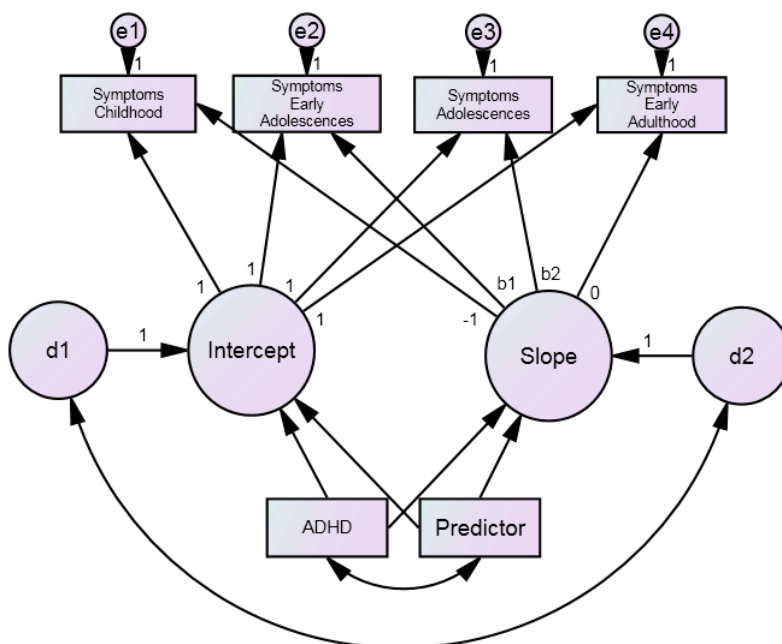


Figure 4.

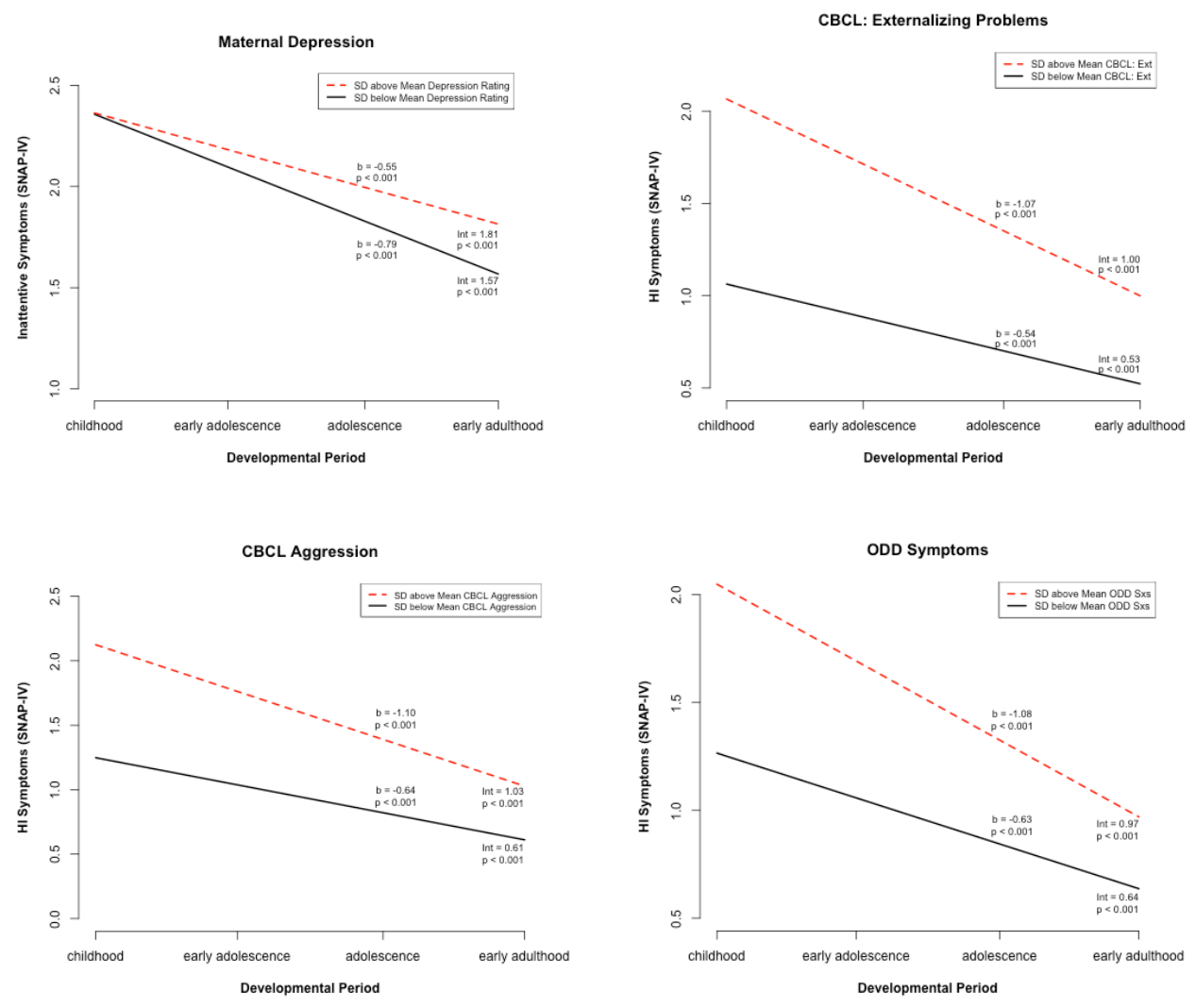


Figure Captions

Figure 1. An overview of latent growth curve models relating caregiver report of symptoms of inattention, hyperactivity/impulsivity, and their combination to maladaptive outcomes in girls with ($n = 140$) and without ($n = 88$) a childhood ADHD diagnosis. ADHD slope coefficients b_1 and b_2 reflect freely estimated parameters. Manifest indicators of the latent outcome variables differ for each model and are depicted here as $Ind_1, Ind_2, \dots, Ind_n$. Factor loadings set to 1 were done so for purposes of model identification.
e = error term; d = disturbance term.

Figure 2. (A-C). The developmental trajectory of (A) total, (B) inattentive, and (C) HI symptoms for girls with ($n = 140$) and without ($n = 88$) a childhood ADHD diagnosis. (D-F). Histograms depicting proband's ($n = 140$) and comparison's ($n = 88$) (D) total, (E) inattentive, and (F) HI symptoms in young adulthood. Panels D and E include cutoff criteria for the SNAP-IV.

Figure 3. A schematic representation of predictors of latent growth curve models relating caregiver report of symptoms of inattention, hyperactivity/impulsivity, and their combination in girls with ($n = 140$) and without ($n = 88$) childhood ADHD. ADHD slope coefficients b_1 and b_2 reflect freely estimated parameters. Note that the intercept in these models is set to represent mean ADHD symptom levels in young adulthood. Factor loadings set to 1 were done so for purposes of model identification.
e = error term; d = disturbance term.

Figure 4. Interactive effects of select predictor variables and time on the changes in ADHD symptoms over time. Upper Left: Mean inattentive symptoms in young adulthood and rate of symptom change across development for children above and below the mean for maternal depression. Upper Right: Mean HI symptoms in young adulthood and rate of symptom change across development for children above and below the mean for Child Behavior Checklist: Externalizing problems. Lower Left: Mean HI symptoms in young adulthood and rate of symptom change across development for children above and below the mean for Child Behavior Checklist: Aggressive Problems. Lower Right: Mean HI symptoms in young adulthood and rate of symptom change across development for children above and below the mean for symptoms of oppositional defiant disorder. Baseline ADHD status was controlled in all analyses.