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Parenting Stress as a Mediator between Childhood ADHD and Early Adult Female Outcomes

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Abstract

Objective—To examine the mediating role of parenting stress (both parental distress and stress due to dysfunctional interactions in the mother-daughter relationship [PSDI]) in the link between childhood attention-deficit/hyperactivity disorder (ADHD) status and several important young-adult outcomes.

Methods—The diverse sample comprised 140 girls with ADHD and 88 age- and ethnicitymatched comparisons, evaluated at ages 6-12 years and followed prospectively for 5 years (mean age = 14.2) and 10 years (mean age = 19.6).

Results—(a) The PSDI experienced by a mother during her daughter's adolescence mediated the link between her daughter's childhood ADHD status and adult externalizing and internalizing symptoms. (b) PSDI also mediated the link between ADHD status and young adult non-suicidal self-injury and had an indirect effect in the relation between childhood ADHD and young-adult depressive symptoms. (c) The mediating role of PSDI with respect to internalizing symptoms and depressive symptoms remained in place even when covarying adolescent internalizing/depressive symptoms.

Conclusion—Parenting stress, particularly related to maternal perceptions of dysfunctional interactions with adolescent daughters, serves as a key mediator in the association between childhood ADHD status and important domains of young-adult functioning. Minimizing parenting stress and dysfunctional mother-daughter interactions during adolescence might reduce the risk of adverse adult outcomes for girls with ADHD.

Keywords

attention-deficit/hyperactivity disorder; parenting; parent-child relationship; stress

Numerous studies have shown that a majority of children diagnosed with attention-deficit/ hyperactivity disorder (ADHD) continue to experience detrimental impairments in adulthood and are prone to more adverse outcomes than their peers. In particular, these individuals experience lower educational attainment (e.g., complete fewer years of education with lower

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grade point averages), have poorer work performance, and show more social problems than other young adults (Barkley, Fischer, Smallish, and Fletcher, 2006; Biederman, et al., 2008; see reviews in Barkley, 2015; Hinshaw & Scheffler, 2014). Furthermore, children diagnosed with ADHD also have an elevated risk of developing other psychiatric disorders—including antisocial, anxiety, mood, and substance use disorders—by early adulthood (Biederman et al., 2006; Meinzer et al., 2013; Charach, Yeung, Troy, & Lillie, 2011). However, little is known about how parenting-related stress experienced by the parents of these children during adolescence may influence these outcomes. Our objective is to investigate the mediating role of parenting stress—particularly the stress experienced by the parents of girls with ADHD during adolescence—in the development of adverse young adult outcomes.

Parenting and Related Stress

It has been well-established that parenting is part of a bidirectional and dynamic process in which both the parent and child influence each other (Pettit & Arsiwalla, 2008; see also Bell, 1968, for a classic formulation). In some parent-child dyads, this bidirectional process can perpetuate increasingly negative, transactional interactions, which can lead to the development of problem behaviors. In particular, Patterson's (2002) coercion model postulates that unskilled parents, in an attempt to control a defiant and oppositional child, may inadvertently reinforce problem behavior by increasing their initial aversive reaction to the child's behavior and then eventually relenting when the child's behavior worsens. Ultimately, the parent learns to withdraw in order to avoid these aversive interactions. This pattern of coercion negatively influences the parent-child relationship and increases the child's risk for the development of deviant behaviors, particularly aggression (Patterson, 2002).

Abidin's model focuses on parental stress in the attempt to explain why some parents are prone to engage in dysfunctional parenting (Abidin, 1992). Parental stress can be described as the negative feelings that a parent has toward the self or toward his or her children, which result from a mismatch between perceived resources and the demands of his or her parenting role (Morgan, Robinson, & Aldridge, 2002; Deater-Deckard, 1998). Abidin posits that the amount of parenting stress experienced by a parent is a function of both parental characteristics (e.g., their sense of competence; the quality of their relationship with their child) and the child's characteristics (e.g., adaptability; demandingness). In combination with life stressors (e.g., loss; divorce), these mutually interactive characteristics can lead to increased risk for dysfunctional parenting (Abidin, 1995; Morgan et al., 2002).

Pelham et al. (1997) examined the effects of child behavior on parental stress. Parents were asked to interact with child confederates who either displayed typical behavior or were trained to display the symptoms of a behavioral disorder. Those parents who interacted with a child mimicking externalizing behavior reported more distress, greater feelings of role inadequacy, and more anxiety, depression, and hostility than those parents interacting with control confederates. Crucially, when this group of parents was subsequently given unlimited access to alcohol while waiting to engage in a second interaction with the same child, they consumed more alcohol than the control parents. Thus, the child's problematic

behavior not only influenced the level of distress felt by the parent but also subsequent parental coping strategies.

Stress in Families with Children with ADHD

Parents of children with ADHD may be particularly prone to parenting stress because, as a function of their children's symptoms, they may experience greater parenting demands than other parents. For example, DuPaul, McGoey, Eckert, and VanBrankle (2001) found that, when asked to perform a task by their parents, children with ADHD exhibited twice the level of noncompliance and had levels of inappropriate behaviors that were equivalent to five times the levels displayed by control children. Their parents concurrently reported higher levels of stress than parents of children without ADHD. The association between a child's ADHD symptoms and parental stress is likely to be mediated by the child's difficulty with self-regulation (Graziano, McNamara, Geffken, & Reid, 2011). That is, parents of children with ADHD may have to exert considerable effort to correct their children's behaviors and to compensate for their children's difficulties with behavioral and emotion self-regulation, which may, in turn, lead them to experience parenting stress. As a result, these parents have trouble utilizing appropriate coping strategies when faced with stressful situations (DuPaul et al., 2001).

Parents of children with ADHD are also prone to dysfunctional interactions with their children and, thus, may be susceptible to the stress associated with these interactions. In particular, such parents tend to display more negative behaviors toward their children than other parents and respond to problematic behaviors with higher levels of reprimands and corrective actions (DuPaul et al, 2001; Modesto-Lowe, Danforth, & Brooks, 2008). The direction of effects is substantially related to the child's problem behavior. For example, Barkley, Karlsson, Pollard, and Murphy (1985) showed that the receipt of stimulant medication by the child with ADHD reduced parental control and direction. This linked to the child's greater compliance and attention.

Past research has also linked negative parental behaviors to reciprocal increases in problematic behavior in children with ADHD. In particular, negative maternal behaviors during parent-child interactions have been associated with stealing and noncompliance during classroom and playground activities in boys with ADHD, even taking into account the boys' levels of noncompliance and negativity during the parent-child interactions (Anderson, Hinshaw, & Simmel, 1994). Aggressive discipline methods and poor parental coping strategies have also been associated with increases in hyperactive symptoms in children with ADHD (Woodward, Taylor, & Dowdney, 1998). Such effects are even found in adoptive families of children with ADHD symptoms (see Harold et al., 2013), which control for gene-environment correlation, revealing that psychosocial as well as genetic processes are at work. In short, dysfunction within families of children with ADHD may be perpetuated by a bidirectional process in which adverse actions from both the parent and the child influence each other, fueling additional stress in the relationship as well as parental perceptions of negativity in relevant interactions.

The stress fueled by dysfunctional parent-child interactions may be particularly salient as children transition to adolescence. During this transition, adolescents begin to spend less time with their parents and more time with peers, and they begin to make normative bids for more autonomy (Eccles, 1999). This tendency is likely to increase tension in the parent-child relationship. Specifically, parents report increases in the affective intensity of parent-child conflict during adolescence (Laursen, Coy, & Collins, 1998). Accordingly, parents report parenting a 14-year-old child as being more stressful than parenting a 10-year-old child. In particular, they report increases in the stress associated with dysfunctional parent-child interactions as their child enters teenage years (Putnick, Bornstein, Hendricks, Painter, Suwalsky & Collins, 2010). Families of adolescents with ADHD—particularly those with comorbid oppositional defiant disorder—have been shown to display more conflict than families of comparison adolescents (Barkley, Fischer, Edelbrock, & Smallish, 1991). Thus, these families are prone to experience higher levels of stress associated with such dysfunctional interactions than other families.

Managing parental stress is important because such stress is associated with poor outcomes in children. For example, parenting stress has been shown to be a predictor of behavioral problems and externalizing issues in young children (Benzies, Harrison, & Magill-Evans, 2004; Heller, Baker, Henker, & Hinshaw, 1996). Parental stress related to dysfunctional interactions in the parent-child relationship has been linked to later offspring internalizing symptoms during adolescence (Costa, Weems, Pellerin, & Dalton, 2006). Yet few studies have examined the associations between stress experienced by parents and longer-term child outcomes, extending into young adulthood. This empirical gap is particularly wide for families with children diagnosed with ADHD, particularly for girls with this condition. Because females normatively begin to experience increases in internalizing behaviors as they transition to adolescence (Nolen-Hoeksema & Girgus, 1994), they may be more likely than their male peers to develop internalizing symptoms in response to the stress experienced by their parents during this period. Hence, our focus herein is on the role of parental stress specifically during adolescence, a particularly challenging period for the parent-child relationship—on key youngadult problems domains in their female offspring.

As noted above, children with ADHD are more prone to adverse adult outcomes than their peers. In addition to poorer education, social, and occupational outcomes, recent research has suggested that they have increased risk for self-harm behaviors in early adulthood. Specifically, young women with a childhood diagnosis of ADHD (particularly when marked by impulsivity) reported far more non-suicidal self-injury (NSSI) and suicide attempts than comparison women (Swanson, Owens, & Hinshaw, 2014). Thus, we investigate the role of parental stress in such elevated risk for NSSI and suicide, as well as for outcomes of depression, internalizing symptoms more generally, and externalizing symptoms.

Hypotheses

We investigate two aspects of parental stress: parental distress and stress-inducing dysfunctional parent-child interactions (PSDI). Parental distress refers to the personal stress that a parent has over his or her parenting role, whereas PSDI addresses a parent's perception of dysfunction in the parent-child relationship that contributes to his or her

feelings of parental stress. Because parents of children with ADHD tend to experience greater parenting demands than other parents, we predict that they will report more parental distress and PSDI than parents of our comparison sample. We also specifically examine how these aspects of stress mediate the pathways between childhood ADHD status and six young-adult criterion measures (i.e., self-reported depressive symptoms; self- and parent-reported externalizing and internalizing symptoms; NSSI severity and frequency; and suicide attempts). Accordingly, for all six outcomes, we expect that both indicators of parental stress (i.e., distress and PSDI) will mediate the links between childhood ADHD status and their young-adult outcomes.

Method

Overview

The current data were drawn from a longitudinal study of elementary-school-aged girls with and without ADHD. Initial data were collected during three summer enrichment programs that took place from 1997 to 1999. Each summer, a new cohort of girls with ADHD participated in a five-week program that offered a combination of classroom, art, drama, and playground activities, along with a group-matched comparison sample of girls without ADHD. Specifically, comparison girls were recruited to be similar, in terms of age and ethnicity, to the ADHD sample. Parents and teachers completed questionnaires as part of the screening process; girls and their families then went through a thorough assessment battery pertaining to ADHD status as well as comorbidities, impairments, and academic, social, and cognitive functioning. All evaluations were conducted during a period in which girls with prior medication histories were not receiving stimulant medication. See Hinshaw (2002) for additional details.

The families were invited to participate in 5-year and 10-year follow-up studies after their initial participation. Participants completed a thorough evaluation, spanning two half-days at our lab/clinic (occasionally, telephone interviews or home visits were performed). Hinshaw, Owens, Sami, & Fargeon (2006) and Hinshaw et al. (2012) provide full information about the follow-up.

Participants—The original sample consisted of 140 girls with ADHD (mean age = 9.7) and 88 comparison girls (mean age = 9.4) at baseline (Wave 1). The sample was ethnically diverse (53% White, 27% African American, 11% Latina, and 9% Asian American). The ADHD sample was recruited through medical settings (e.g., health maintenance organizations), mental health centers, pediatric practices, and local school districts. Advertisements were also placed in local newspapers and parenting newsletters. Comparison girls were recruited through school districts and local community centers and through advertisements in the local newspapers and parenting newsletters. Eligible families were sent packets about the program and then screened for ADHD status. ADHD diagnosis was made on the basis of initial ratings from parents and teachers, with initial screening criteria set somewhat low to avoid prematurely excluding potentially eligible girls. Yet for final eligibility in the ADHD group, the girl had to meet full criteria for ADHD (either Combined or Inattentive type) with respect to diagnostic interview criteria (i.e., at least six impairing

symptoms of inattention for ADHD-Inattentive or at least six inattention and six hyperactivity-impulsivity symptoms for ADHD-Combined). For eligibility in the comparison group, a diagnosis of ADHD needed to be absent (see Hinshaw, 2002).

Of the original 228 families, 209 (92%) participated in the five-year follow-up study (designated as Wave 2; mean age = 14.2) and 216 (95%) participated in the 10-year follow-up study (designated as Wave 3: mean age = 19.6). See Hinshaw et al. (2006) and Hinshaw et al. 2012) for more information. The parental stress measures below were administered at the Wave 2 follow-up; the primary caregiver served as the informant. In 94% of the cases, this was the child's mother (1% were fathers, 1% were stepmothers, 3% were grandmothers, and 1% were other relatives). In order to focus on the parental stress measure was completed by the mother (120 girls with ADHD and 81 comparison girls) in our analysis.

Measures

Parental Stress during adolescence

Parenting distress and parental stress regarding dysfunctional interactions

(PSDI): These constructs were assessed via the Parenting Stress Index-Short Form (PSI-SF), a self-report measure assessing the personal stress experienced by parents in their parenting role (Abidin, 1995). Mothers rated each item on a 5-point scale (5 = strongly disagree, 1 = strongly agree). The subscale tapping parental distress consists of 12 items (e.g., "I feel that I cannot handle things") that measure the stress a parent feels in her/his parental role. The Parental-Child Dysfunctional Interaction subscale, which taps PSDI, consists of 12 items (e.g., "My child does not like me or want to be close") that focus on parents' perception of their interactions with their child and their perception of how reinforcing their child is toward them (Abidin, 1995). The items were coded so that higher scores represent higher levels of stress-related dysfunctional interactions. The test-retest reliability and internal consistency of the PSI-SF have been well established, with the reliability of the subscales ranging from .68 to .85 and the internal consistency ranging from .80 to .87 (Abidin & Brunner, 1995). Previous research has also established its validity with respect to other indicators of parental stress and family dysfunction (Haskett, Ahern, Ward, & Allaire, 2006). In our sample, the internal consistency (Cronbach's alpha) of the parental distress scale and the dysfunctional interaction scale (measuring PSDI) at Wave 2 were .87 and .89 respectively.

Young-adult outcomes

Externalizing and internalizing behaviors: Composites of scores using data from both the Adult Behavior Checklist (ABCL) and the Adult Self Report (ASR) were created to measure externalizing and internalizing behaviors. The ABCL and ASR are 126-item measures that parallel each other. ABCL is completed by a caregiver, whereas the ASR is completed by the young adult. Both informants are asked to rate items assessing the young adult's behavior using a 3-point scale (0 = never happened; 2 = happened with great frequency or great intensity). These measures have extensively established internal consistency, test-retest reliability, and validity. Cronbach's alphas for the internalizing and the externalizing scales have been reported at as .80 and .92, respectively on the ABCL, with parallel figures being .

89 and .91 for the ASR (Achenbach & Rescorla, 2003). The raw scores on these scales are converted to T-scores using age and gender norms. Composites were then created by taking the mean of these scores. In our study, the parent-reported ABCL was correlated with the ASR at r(170) = .44, p < .001 for internalizing behaviors and r(170) = .49, p < .001 for externalizing behaviors.

Youth-reported depression: The young women's self-reported depressive symptoms were measured via the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). This measure comprises 21 items and is used to assess the presence of depressive symptoms including negative mood, interpersonal problems, and negative self-esteem. Participants are asked to rate the presence of each symptom during the past two weeks on a 4 point scale (i.e. "0" indicates an absence of a symptom; "3" indicates the presence of an extreme form of a symptom). Both the test-retest reliability and internal consistency of the CDI have been well established: internal consistency = .92 in outpatient populations and .93 in college age students; test–retest reliability averages .93 (Beck et al., 1996). In our sample, the internal consistency of this measure was .93.

Nonsuicidal Self-injury (NSSI): NSSI was assessed using a modified version of the Self-Injury Questionnaire (SIQ) for which participants were asked if they had deliberately injured themselves in the past year using six different methods (Claes, Vandereycken, & Vertommen, 2001). If a method was endorsed, participants were asked how frequently they engaged in this method on a 6-point scale (1 = only once; 6= a couple of times a day). A sum of the frequency of scores across the six methods was taken and used in the present analyses. This measure was validated in a sample of individuals with eating disorders and was found to have an internal consistency of $.62^1$ (see Vanderlinden & Vandereychen, 1997; Claes & Vandereycken, 2007). A similar internal consistency of .60 was found in our sample. A 4-point score assessing the severity of NSSI endorsed was also created. This score was informed by a lethality continuum of potential tissue damage postulated by Skeggs (2005) and confirmed by factor analysis. It was created based on the highest level of severity endorsed on the SIO with a score of "0" representing non-endorsement of any NSSI behaviors,"1" representing low severity of NSSI methods, "2" representing mild-moderate severity of NSSI methods, and "3" representing the endorsement of the highest level of severity (see Swanson et al., 2014).

Number of suicide attempts: The Barkley Suicide Questionnaire (Barkley & Murphy, 2006) and the Family Information Packet (FIP) were used to assess suicide attempts. The Barkley Suicide Questionnaire is a self-report questionnaire in which participants were asked three yes/no questions: "*have you ever attempted suicide*", "*have you ever considered suicide*", "*have you ever been hospitalized for an attempt?*" The FIP, which was completed by the parents, was a comprehensive, year-by-year grid requesting information about significant events in the young adult's life, including suicide attempts. Information from both measures was used to create a dichotomous self-injury variable indicating whether or not a participant had made a suicide attempt. In the single case for which an attempt was

¹The internal consistency for this measure is not extremely high, because this score reflects the sum of six methods endorsed. Participants tend to only endorse one method (e.g. "cutting") and not others (e.g. "burning)".

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endorsed on the FIP but not on the Barkley Suicide Questionnaire, the individual was counted as positive for a suicide attempt.

Covariates: Several important background variables, scored at Wave 1, were included as covariates in the mediation analyses: (a) age, (b) maternal education, and (c) parent-reported family income. We also performed a second set of analyses with the young women's adolescent externalizing, internalizing, and depressive symptoms as covariates, in order to assess the mediating role of parental stress in the link between ADHD and the development of internalizing, externalizing, and depressive symptoms, beyond levels of those same outcomes measured during adolescence. That is, for each adult criterion variable, we added the respective adolescent variable as a covariate in addition to age, maternal education, and family income. The girls' adolescent internalizing and externalizing symptoms were reported by the mothers in Wave 2 using the Child Behavior Checklist (CBCL), which is a 113-item measure that parallels the ABCL (Achenbach, 1991). The girls' adolescent depressive symptoms were self-reported using the short form of the Children's Depression Inventory (Kovacs, 1992).

In previous findings, we have found that externalizing symptoms mediated the link between childhood ADHD status and young adult NSSI, whereas internalizing symptoms mediated the link between childhood ADHD status and young adult suicide attempts (Swanson et al., 2014). Thus, for NSSI frequency and severity we used adolescent externalizing symptoms as the additional covariate; for suicide attempt count we used internalizing symptoms.

Statistical Analyses

Statistical analyses were performed using SPSS Version 22. T-tests were conducted to assess diagnostic group differences with respect to covariates, putative mediator variables, and young-adult criterion measures. We then performed sophisticated mediator analyses with the bootstrapping procedure of Preacher and Hayes (2008) to examine the mediating role of each of the two parental stress measures in the link between childhood ADHD status and each criterion variable. In this procedure, 10,000 random samples are drawn from the original data with replacement. A point estimate of the mediated effect is determined for each sample, with 95% bias-corrected confidence intervals determined from the distribution of these estimates. Intervals are considered statistically significant if they do not contain 0. Least-squares regressions were performed to ensure that predictor-mediator and mediatoroutcome pathways were in hypothesized directions. We only performed multi-meditator models for those criterion measures that were significantly correlated with both mediator variables. In those models, Wave 1 ADHD status was the predictor, Wave 2 parental distress and PSDI were tested together as potential mediators, and each Wave 3 outcome variable (i.e., internalizing symptoms, self-reported depression symptoms, externalizing symptoms, NSSI severity, NSSI frequency, and suicide attempt count) served as the criterion measure. Figure 1 provides a graphical representation of the pathways and effects explored. We performed the meditational analyses both with and without use of the adolescent psychopathology covariates (see Covariates, above).

Results

Table 1 presents scores for the ADHD and comparison samples with respect to demographic, mediating, and behavioral variables. The diagnostic groups were statistically indistinguishable with respect to age, family income, and maternal education. However, a chisquare test did reveal a significant difference in the ethnic composition of the groups, $\chi^2(4)$, N = 201 = 9.30, p <.05, with a higher percentage of Asian American girls in the comparison group. Furthermore, the young women who were diagnosed with ADHD in childhood had far worse scores on all adult behavioral outcomes, and their mothers also had significantly higher Wave 2 parental distress and PSDI scores than the mothers of comparison girls.

Table 2 presents intercorrelations between the parental stress and behavioral variables. Neither of the parenting stress variables was significantly correlated with suicide attempt count. Thus, mediational analyses were not performed for this criterion measure. Parental distress was also not significantly correlated with depressive symptoms, but PSDI was. Thus, for the meditational analyses including depressive symptoms, only PSDI was explored as a possible mediator.

Mediational Analyses

The results for the mediational analyses are presented in Tables 3, 4, 5, and 6.

ADHD-Internalizing linkage—When controlling for age, maternal education, and family income, there was a significant and positive relation between Wave 1 ADHD status and Wave 3 internalizing symptoms, b = 7.11, standard error [SE] = 1.64, t(182) = 4.34, p < .001 (i.e., c path). PSDI was a significant partial mediator of this relation, indirect effect [IE] = 2.92, SE = 1.01, CI₉₅ = 1.35–5.38. Parental distress was not a significant partial mediator, IE = -.34, SE = .54, CI₉₅ = -1.61–.57.

The relation between Wave 1 ADHD status and Wave 3 internalizing symptoms remained significant when covarying Wave 2 internalizing symptoms, b = 3.86, SE = 1.73, t(182) = 2.23, p < .05. In these analyses, PSDI also served a significant full mediator, IE = 1.13, SE = 1.13, CI₉₅ = .13–2.84, but parental distress was not a significant mediator, IE = -.15, SE = . 26, CI₉₅ = -1.09–.14.

ADHD-Depression linkage—When controlling for age, maternal education, and family income, the relation between Wave 1 ADHD status and Wave 3 depressive symptoms was not significant: b = 2.51, SE = 1.58, t(180) = 1.58, p > .05. However, despite insignificant total effects for the relation between ADHD and depressive symptoms, a significant indirect effect was found for PSDI, IE = 1.39, SE = .58, CI₉₅ = .46–2.77.²

Parallel results were found when Wave 2 depressive symptoms were added as a covariate. The relation between Wave 1 ADHD status and Wave 3 depressive symptoms remained non-

²Some investigators argue that significant total effects are not required to test the indirect effects, suggesting quantification of the indirect the effects instead of inferring them from the significance testing of the direct and total effects (see Hayes, 2009 for a more detailed explanation).

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significant: b = 1.18, SE = 1.56, t(179) = .76, p > .05. Significant indirect effects were found for PSDI, IE = 1.00, SE = .52, CI₉₅ = .18–2.23.

ADHD-Externalizing linkage—When controlling for age, maternal education, and family income, there was a significant and positive relation between Wave 1 ADHD status and Wave 3 externalizing symptoms, b = 10.18, SE = 1.45, t(182) = 7.35, p <.001. Parental distress was not a significant mediator, IE = -.25, SE = .48, CI₉₅ = -1.25-.71. However, PSDI was a significant partial mediator, IE = 1.96, SE = .75, CI₉₅ = .80–3.80.

The relation between Wave 1 ADHD status and Wave 3 externalizing symptoms remained when covarying Wave 2 externalizing symptoms, b = 4.68, SE = 1.62, t(181) = 2.90, p < .01. However, neither parental distress, IE = .10, SE = .11, CI₉₅ = -.16-.95, nor PSDI, IE = -.05, SE = .26, CI₉₅ = -.85-.31 remained as significant mediators with such stringent control.

ADHD-NSSI frequency linkage—When controlling for age, maternal education, and family income, the relation between Wave 1 ADHD status and Wave 3 NSSI frequency was significant: b = 2.07, SE = .55, t(168) = 3.74, p < .001. Neither parental distress served, IE = . 31, SE = .25, CI₉₅ = -.05–97; nor PSDI, IE = .10 SE = .27, CI₉₅ = -.39–.70 served as significant mediators.

The relation between ADHD status and NSSI frequency was retained when Wave 2 externalizing symptoms was added as a covariate, b = 1.41, SE = .68, t(167) = 2.08, p < .05. However, neither parental distress, IE = -.05, SE = .11, CI₉₅ = -.42-.09, nor PSDI, IE = -. 01, SE = .08, CI₉₅ = -.23-.10, was a significant mediator with such stringent control.

ADHD-NSSI severity linkage—When controlling for age, maternal education, and family income, the relation between Wave 1 ADHD status and Wave 3 NSSI severity was significant: b = .64, SE = .15, t(169) = 4.36 p < .001. Parental distress did not serve as a mediator, IE = .01, SE = .06, CI₉₅ = -.06–.18. However, PSDI, IE = .12, SE = .07, CI₉₅ = . 01–.30, did serve as a partial mediator.

When Wave 2 externalizing was added as a covariate, the relation between ADHD status and Wave 3 NSSI severity remained significant, b = .52, SE = .16, t(168) = 3.21, p > .05. Neither parental distress, IE = .01, SE = .02, CI₉₅ = -.01-.09, nor PSDI, IE = .04, SE = .04, CI₉₅ = -.01-.17, remained as significant mediators here.

Discussion

As predicted, the mothers of young women diagnosed with childhood ADHD reported experiencing more parental distress and PSDI during their daughter's adolescence than did comparison mothers. Furthermore, and crucially, PSDI served as a mediator between childhood ADHD status and several criterion measures (i.e., externalizing and internalizing outcomes as well as NSSI severity), even though parental distress did not. PSDI continued to serve as a mediator between ADHD and internalizing symptoms and had an indirect effect in the relation between ADHD and depressive symptoms, even with stringent means of statistical control (i.e., including an adolescent version of the criterion variables as a covariate). The strong suggestion is that PSDI plays a role (independent of parental distress)

in the pathway from childhood ADHD toward young-adult internalizing and depressive symptoms.

Our findings are consistent with previous research reporting that children diagnosed with ADHD—particularly girls—are more prone to adverse adult outcomes than their peers (e.g. Barkley et al., 2006; Biederman et al., 2006, Biederman et al., 2008). As noted earlier, mothers of children with ADHD are likely to experience greater parental demands because of their child's symptoms and difficulties with self-regulation; this increases their own vulnerability to parental stress (Granziano et al., 2011; Du Paul et al., 2001). Our results suggest that the parental stress associated with dysfunction in the parent-child relationship plays a key role in the development of adverse outcomes.

Abidin's stress model (1992) suggests that certain child factors (in this case, the child's ADHD status) and parent factors (in this case, the parent's perception of her interactions with her child) contribute to the amount of stress a parent feels, which, in turn, affects her parenting ability. Maladaptive parenting practices have been shown to exacerbate the symptoms of ADHD, which can lead to adverse outcomes in children (see Johnston & Mash, 2001). Thus, the link we found between PSDI and adverse outcomes is likely to be related to the tendency for stressful interactions in the parent-child relationship to trigger less-than-optimal parenting practices (e.g., ineffective monitoring, inconsistent discipline, withdrawal of parental support).

In addition, other forms of reciprocal, transactional processes are also likely to be at play. For example, adolescent-related ADHD behavior patterns may lead youth to seek deviant peers, who also reinforce tendencies toward internalization or externalization (Marshal, Molina, & Pelham, 2003). Further research should be done to investigate the mechanisms by which other child characteristics and parental stress interact to increase the risk for adverse outcomes.

Because neither indicators of parenting stress was associated with suicide attempt count, there are doubtless other factors that may mediate the relation between early ADHD symptomatology and suicide attempts. For example, maternal depression has been associated with increased risk for depression and suicidal behaviors in children with ADHD (Chronis-Tuscano et al., 2010). Past research has also suggested that the high rate of suicidal behaviors in individuals with ADHD may be related to the presence of comorbid conditions, such as anxiety disorder, major depressive disorder, and substance abuse disorders (Balazs, Miklosi, Kereszteny, Dallos, & Gadroros, 2014). Furthermore, in Swanson et al. (2014), we found that an objective measure of response inhibition/impulsivity, as well as both externalizing and internalizing symptoms during adolescence, fell on the pathway to NSSI and suicide attempts. Thus, additional research should be done to explore other mediators in the link between ADHD and suicide attempts.

It is also important to explore why PSDI, but not parental distress, mediated the link between ADHD and internalizing and depressive symptoms. As noted earlier, parental distress focuses on the aspect of parental stress that is associated with parents' perception of their parenting role, while PSDI focuses on parents' perception of their parent-child

interactions that may induce parental stress. Because of the dynamic nature of parent-child interactions, a child's ADHD symptoms are likely to have more of a direct role on dysfunction and associated stress in the parent-child relationship than on a parent's more general perception of her parenting role. Furthermore, previous research has suggested that there is an increase in the affective intensity of parent-child conflict in adolescence (Laursen, Coy, & Collins, 1998), along with an increase in the parental stress associated with such conflict (Putnick et al., 2010). Thus, PSDI may be salient during this time period and, as a result, may increase the chance that an adolescent may develop resultant internalizing and depressive symptoms—even over and above her adolescent levels of these symptoms.

Our use of an all-female sample might also account for why PSDI continues to mediate ADHD and internalizing/depressive symptoms but not externalizing symptoms when covarying adolescent behavior patterns, as increases in depressive and internalizing symptoms are normative for girls during the transition to adolescence (Nolen-Hoeksema & Girgus, 1994). Future studies should explore whether the same persistent mediational role of PSDI in the relation between ADHD and internalizing symptoms can be generalized to males with ADHD.

In addition to a lack of generalizability to males with ADHD, there are additional limitations in our investigation that should be taken into consideration when interpreting our results. First of all, we did not explore the role of other child characteristics (i.e., personality traits) in the development of their parents' stress. Given the aforementioned bidirectional nature of parenting stress and child behaviors, a future study investigating the role of a child's early temperament and/or personality characteristics—in addition to their ADHD symptoms— could enlighten our understanding of how parental stress develops and influences later outcomes. In addition, future research should also explore how other parent characteristics and broader family variables might affect the associations found. For example, a parent's own ADHD status and depressive symptomology are strong predictors of their parental stress (Theule, Wiener, Rogers, & Marton, 2011; Theule, Wiener, Tannock, & Jenkins, 2013.)

We also were not able to include the girls' own account of their experience of their parents' stress. Such an investigation would also help us understand the mechanisms by which parental stress influences later outcomes. Furthermore, the primary reporters for parental stress in our study were mothers. Because of low participation rates and a number of single-parent families, we were not able to include the father's report of parental stress, which might also be associated with key young-adult behavior patterns in females with ADHD.

Because the mothers' reports were used (in addition to teacher reports) to determine the girls' childhood diagnoses, to construct the girls' young-adult externalizing and internalizing composites, and to assess parental stress, our investigation may be subject to shared method variance. Although we tried to mitigate this issue by including input from other sources, future investigations could be improved by including more objective measures of externalizing and internalizing symptoms.

In addition, the adjusted R² values for the models exploring the mediating role of PSDI on depressive symptoms were low, meaning that only a small percentage of variance of this key outcome was attributable to PSDI. Furthermore, the statistical methods used in our analyses allowed us to explore only one criterion variable at a time. Because some of our criterion variables were highly correlated (e.g., girls' internalizing symptoms and depressive symptoms), future research exploring these criteria as composites may be in order. Finally, because we did not covary baseline internalizing and externalizing symptoms, we are not able to postulate that the relations observed are specific to girls' childhood ADHD symptoms and not their associated early internalizing/externalizing behaviors. However, our investigation is still relevant to children with ADHD, as these behavior patterns are common comorbidities (Jensen et al., 2001).

Despite these limitations, we have demonstrated the importance of dysfunctional family interactions and the stress experienced by parents of adolescent girls with ADHD in young adult development of crucial comorbidities and impairments. Interventions targeting dysfunctional parent-child interactions may help to reduce girls' risk for internalizing, externalizing, and depressive symptoms as young adults. Such interventions would doubtless need to incorporate multimodal efforts, including enhancement of parental competence, treatments tailored to the particular challenges of adolescence for youth with ADHD, and broader provision of supports to such families.

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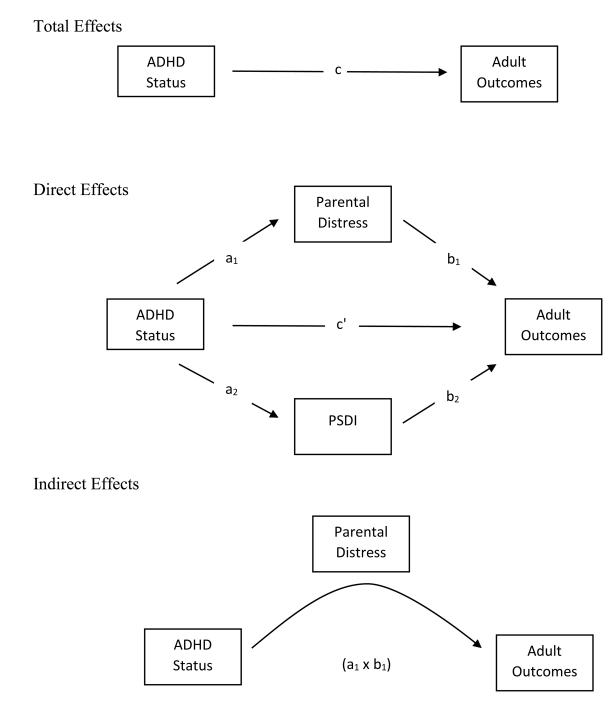


Figure 1.

Graphical Representation of Mediation Model. The total effect is composed of the direct effect (c') and indirect effects (the product of a and b). (See Preacher and Hayes, 2008 for more information)

Demographics, Mediators, and Outcome Variables

	ADHD ^a	Da	Compa	Comparison ^b	T-test	Conen's
	Μ	SD	М	SD	t (df)	q
Demographic Variables (Wave 1)						
Age (months)	115.95	20.66	111.84	19.30	-1.42 (199)	.21
Ethnicity (%)						
Caucasian	60.8		46.9			
African American	22.5		27.2			
Hispanic	11.7		11.1			
Asian American	4.2		14.8			
Total Annual Family Income $^{\mathcal{C}}$	6.47	2.59	6.81	2.37	.94 (197)	14
Maternal Education ^d	4.76	.88	4.98	.95	1.66 (199)	24
Mediators (Wave 2)						
Parental Distress	2.24	.80	1.88	.55	-3.69 (192)**	.52
PSDI	2.25	.80	1.68	.67	-5.35 (192)**	LT.
Adolescent Covariates (Wave2)						
Internalizing Behaviors	56.50	10.81	45.57	10.94	-6.92 (194) ^{**}	1.01
Externalizing Behaviors	61.67	10.87	45.80	9.92	$-10.38\left(194 ight)^{**}$	1.53
Self-reported BDI	7.53	5.87	5.01	5.28	$-3.10(198)^{**}$.45
Outcome Variables (Wave 3)						
Internalizing Behaviors	56.83	11.29	49.04	10.57	$-4.84(190)^{**}$.71
Externalizing Behaviors	59.43	10.25	48.64	8.56	-7.69 (190)**	1.14
Self-reported BDI	11.37	10.18	8.32	10.72	-1.99 $(187)^{*}$.29
NSSI Frequency	3.01	4.47	.93	1.83	-4.28 (176)**	.61
NSSI Severity	.93	1.07	.32	.66	-4.70 (177)**	69.
Suicide Attempt Count e	19 (17.1%)	.1%)	4 (5.1%)	1%)		

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 $a_{n=120}^{a}$

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 $b_{n=81}^{b}$

 $\mathcal{C}_{\text{For total annual family income, }1<\$10,000; 9>\$75,000.$

 $d_{\rm For}$ maternal education, 1 = less than 8th grade; 6 = advanced or professional degree

 e For suicide attempt count, $\chi^{2}(1,N=199)=6.30,\,p<05$

* p < .05,

** p < .01.

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Table 2

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Summary of Correlations for Mediators and Outcomes

Measures	1	7	3	4	S	9	7
1. W2 Parental Distress	,		,				
2. W2 PSDI	** 69.	ı			1	ı.	
3. Externalizing Behaviors	.30**	.43 **	,	,	,	,	
4.Internalizing Behaviors	.30 **	.47 **	.75 **	ı	ı		
5. Self-Reported BDI	60.	.22 ^{**}	.57 **	.65 **	ī		
6. NSSI Frequency	.23 **	.21 ^{**}	.33 **	.33 **	.22 **	,	
7. NSSI Severity	.28 **	.30 **	.38**	.36 ^{**}	.23 **	.80 **	
8. Suicide Attempt Count	.13	.11	.22 *	.33 **	.18**	.46 **	.50**
* p < .05							
** p <.01.							

•	covariates
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5	Summary

bSE b SInternalizing $35 * *$ 11 -96 $1.$ Parental Distress $35 * *$ 11 -96 $1.$ PSDI $50 * *$ 11 $5.85 * *$ $1.$ Parental Distress $35 * *$ 11 $5.85 * *$ $1.$ Parental Distress $35 * *$ 11 $2.89 * *$ $1.$ Parental Distress $35 * *$ 11 $3.94 * *$ $1.$ PSDI $50 * *$ 11 $3.94 * *$ $1.$ PSDI $52 * *$ 11 $2.69 *$ $1.$ Notes: The independent variable (N) is ADHD Status		effects (a x b)	x b)	CI for Indirect Effects	ndirect cts	effects (c')	(c,)	effects (c)	(c)	for Model
Internalizing Parental Distress 35 ** .1196 PSDI .50 ** .11 5.85 [*] Externalizing .11 5.85 [*] Parental Distress .35 ** .1171 PSDI .50 ** .11 3.94 [*] Self-reported BDI .52 ** .11 2.69 PSDI .52 ** .11 2.69 Notes: The independent variable (<i>IV</i>) is ADHD	SE	q	SE	Lower	Upper	q	SE	q	SE	
Parental Distress .35 ** .11 96 PSDI .50 ** .11 5.85 * Externalizing .11 5.85 * Externalizing .35 ** .11 71 Parental Distress .35 ** .11 71 PSDI .50 ** .11 3.94 * PSDI .50 ** .11 3.94 * Self-reported BDI .52 ** .11 2.69 PSDI .52 ** .11 2.69 Notes: The independent variable (<i>IV</i>) is ADHD .50 ** .11 2.69										
PSDI .50** .11 5.85* Externalizing .50** .11 5.94* Parental Distress .35** .11 3.94* PSDI .50** .11 3.94* Self-reported BDI .52** .11 2.69 PSDI .52** .11 2.69 Notes: The independent variable (<i>IV</i>) is ADHD .50 .50	06 1.43	34	.54	-1.61	.57	******		*		ę
Externalizing Parental Distress .35 ^{**} .1171 PSDI .50 ^{**} .11 3.94 ^a Self-reported BDI .52 ^{**} .11 2.69 PSDI .52 ^{**} .11 2.69 Notes: The independent variable (<i>IV</i>) is ADHD	** 1.39	2.92 ^a	1.01	1.35	5.38	4.53	1.02	7.11	1.04	77.
Parental Distress.35 **.1171PSDI.50 **.113.94*Self-reported BDI.52 **.112.69PSDI.52 **.112.69Notes: The independent variable (<i>IV</i>) is ADHD										
PSDI.50**.113.94*Self-reported BDIPSDI.52**.11.52**.11.52**.11.52**.11.52**.11.51.52**.11.52**.11.52**.11.52**.11.52**.11.52**.11.52**.54**.55	1 1.31	25	.48	-1.25	.71	* * 		*****		t
Self-reported BDI PSDI52 ^{**} 11 2.69 Notes: The independent variable (<i>IV</i>) is ADHD	Ļ** 1.27	1.96^{a}	75	.80	3.80	8.97	1.48	10.68	1.45	17:
PSDI52 **11 2.69 Notes: The independent variable (<i>JV</i>) is ADHD										
Notes: The independent variable (IV) is ADHD	9* 1.05	1.39 ^a	.58	.46	2.77	1.12	1.65	2.51	1.58	.03
	O Status									
^{<i>a</i>} Significant point estimate ($p < 0.05$).										
* p < .05,										
** n < 01										

Summary of mediation results with age, maternal education, and family income as covariates

Dependent variable (DV) and Mediators (M)	Effects of <i>IV</i> on <i>M</i> (a)	of <i>IV</i> (a)	Effects of M on DV (b)	v (b)	Indirect effects (a x b)	rect (a x b)	Bootstrap 95% CI for Indirect Effects	tstrap 95% for Indirect Effects	Direct effects (c')	(c ³)	Total effects (c)		Adj. R ² for Model
	q	SE	q	SE	ą	SE	Lower	Lower Upper	q	SE	q	SE	
NSSI (frequency)													
Parental Distress	.37 **	.11	.83	.51	.31	.25	05	76.	****	02	* * 	22	00
PSDI	.48	II.	.22	.50	.10	.27	39	.70	1.60	oc:	2.07	сс .	00.
NSSI (severity)													
Parental Distress	.37**	.11	II.	.13	.01	.06	06	.18	**		**		<u>-</u>
PSDI	.47 **	Ħ.	.25*	.13	.12 ^a	.07	.01	.30	48	ci.	.64	<u>ci</u>	ct.
Notes: The independent variable (IV) is ADHD Status	t variable ((<i>IV</i>) is .	ADHD S	tatus									
^{<i>a</i>} Significant point estimate ($p < 0.05$).	late $(p < 0)$.05).											
* p < .05,													

** p < .01.

Summary of mediation results with age, maternal education, family income, and adolescent variables as covariates

Mediators (<i>M</i>)	Effects of <i>IV</i> on <i>M</i> (a)	of <i>IV</i> (a)	Effects of M on DV (b)	of M (b)	Indirect effects (a x b)	rect (a x b)	Bootstr CI for] Eff	Bootstrap 95% CI for Indirect Effects	Direct effects (c')	ect s (c')	Total effects (c)	al s (c)	Adj. R² for Model
	q	SE	q	SE	q	SE	Lower	Upper	q	SE	ą	SE	
Internalizing b													
Parental Distress	60.	.11	-1.67 1.43	1.43	15	.26	-1.09	.14	000	-	*		l
PSDI	.11*	.11	5.05 **	1.40	1.13^{a}	1.13	.13	2.84	2.88	1.69	3.86	1.73	:22
Externalizing c													
Parental Distress	07	.12	-1.46	1.23	.10	II.	16	.95	***		****		00
PSDI	03	.12	1.76	1.26	05	.26	85	.31	4.63	1.02	4.68	1.02	٥ <i></i> с.
Self-reported BDI ^d													
IDSA	.46	.11	2.16^*	1.03	1.00^{a}	.52	.18	2.23	.18	1.61	1.18	1.56	.07
Notes: The independent variable (IV) is ADHD Status	t variable ((<i>IV</i>) is <i>i</i>	ADHD Sta	tus									
^{<i>a</i>} Significant point estimate ($p < 0.05$)	ıate (p < 0.	.05)											
^b Wave 2 Internalizing added as covariate,	added as co	ovariate	ć										
c Wave 2 Externalizing added as covariate,	added as c	ovariate	ຄົ										
$d_{\rm Wave~2~BDI}$ scores added as covariate	lded as cov	'ariate											
* p <.05,													
** p <.01													

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Summary of mediation results with age, maternal education, family income, and adolescent variables as covariates (continued)

b SE b SE b SE Lower Upper b SE SE SE SE SE SE SE SE SE<	(DV) and Mediators (M)	Effect: on <i>h</i>	Effects of <i>IV</i> on <i>M</i> (a)	Effects of N on DV (b)	Effects of M on DV (b)	Indirect effects (a x b)	ect (a x b)	Bootstr CI for] Eff	Bootstrap 95% CI for Indirect Effects	Direct effects (c')	s (c')	Total effects (c)	al s(c)	Adj. R ² for Model
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$		<i>q</i>	SE	<i>q</i>	SE	9	SE	Lower	Upper	9	SE	q	SE	
ss 07 $.12$ $.76$ $.52$ 05 $.11$ 42 $.09$ 07 $.12$ $.13$ $.53$ 01 $.08$ 23 $.10$ 1.47^{*} $.68$ 1.41^{*} $.68$ ss $.08$ $.11$ $.11$ $.14$ $.01$ $.02$ 01 $.09$ $.47^{**}$ $.16$ $.52^{**}$ $.16$	NSSI (frequency) b													
07 .12 .13 .5301 .0823 .10 1.47 .00 1.41 .00 ss .08 .11 .11 .14 .01 .0201 .09 $.47^{**}$.16 $.52^{**}$.16	Parental Distress	07	.12	.76	.52	05	11.	42	60.	* [07	*		5
ss .08 .11 .11 .14 .01 .0201 .09 .17 .12 .25 .13 .04 .0401 .17 .47*** .16 .52** .16	IQS4	07	.12	.13	.53	01	.08	23	.10	1.47	00.	1.41		/0.
tal Distress .08 .11 .11 .14 .01 .0201 .09 .47 ** .16 .52 ** .16 .52 ** .16	NSSI (severity) ^C													
.17 .12 .25 .13 .04 .0401 .17 .47 .10 .52 .10	Parental Distress	.08	.11	.11	.14	.01	.02	01	60.	*	2	*		5
	PSDI	.17	.12	.25	.13	.04	.04	01	.17	.47	0 T .	.52		c1.
	ignificant point estin	nate (p < l	0.05),											
a Significant point estimate (p < 0.05),	Vave 2 Externalizing	added as	covariat	e,										
tgnificant point estimate (p < 0.05), ∕ave 2 Externalizing added as covariate,	Vave 2 Externalizing	added as	covariat	e										
^a Significant point estimate (p < 0.05), ^b Wave 2 Externalizing added as covariate, ^c Wave 2 Externalizing added as covariate	* p < .05													
^a Significant point estimate (p < 0.05). ^b Wave 2 Externalizing added as covariate, ^c Wave 2 Externalizing added as covariate [*] p < .05	**													