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Association of Parental ADHD and Depression With Externalizing and Internalizing Dimensions of Child Psychopathology

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Abstract

Objective: To study the independent association of parental depression and ADHD on three dimensions of child psychopathology among 178 children aged 5 to 10 years.

Method: Self-reported measures of parental depression and ADHD as well as rating scales and structure diagnostic interviews of child internalizing, ADHD, and externalizing problems were obtained.

Results: Structural equation modeling indicated that parental ADHD was positively associated with a broad child problems factor after a second-order factor of child problems best accounted for the high intercorrelations among the internalizing, ADHD, and externalizing child psychopathology factors. Parental depression did not significantly predict the second-order child problems factor, but it specifically predicted the child internalizing factor.

Conclusion: These results suggest that parental ADHD may be a nonspecific risk factor for child psychopathology broadly, whereas parental depression may function as a specific risk factor for child internalizing problems.

Keywords

Attention-deficit hyperactivity disorder; parental ADHD; parental depression

Parent psychopathology is a reliable predictor of diverse forms of child psychopathology. There is consistent evidence that children of psychiatrically ill parents are more likely to exhibit emotional, behavioral, cognitive, and social difficulties than offspring of healthy parents (Leverton, 2003; Mordoch & Hall, 2002). Parental ADHD, anxiety, bipolar, and substance disorders are frequently associated with negative child outcomes (Del Bello & Geller, 2001; Drummond & Fitzpatrick, 2000; Nigg & Hinshaw, 1998), but parental

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Note

depression, in particular, has emerged as a primary risk factor for child psychopathology (Downey & Coyne, 1990).

The association of maternal depression with child psycho-pathology has been reported in naturalistic studies (Beardslee, Bemporad, Keller, & Klerman, 1983; Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; Shanahan, Copeland, Costello, & Angold, 2008; Trapolini, McMahon, & Ungerer, 2007; Weissman et al., 1984, 2006), carefully controlled intervention studies (Grimbos & Granic, 2009; Shaw, Connell, Dishion, Wilson, & Gardner, 2009), and across developmental periods (i.e., toddlers and school-aged children; Cummings & Davies, 1994; Downey & Coyle, 1990). Although failures to replicate the role of parental depression in child psychopathology have also been reported (Griesler, Hu, Schaffran, & Kandel, 2008), overall, there is considerable research implicating parental depression as a plausible risk factor for child psychopathology, including evidence that it satisfies established criteria for a causal risk factor (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

In addition to the consistent association of parental depression with child depression (Hammen, Shih, & Brennan, 2004), there is evidence that its association may also extend to other child mood disorders and externalizing behavior problems. Offspring of mothers with chronic depression were rated by parents and teachers as having greater internalizing and externalizing problems from the Child Behavior Checklist (CBCL), than children of sometimes or never-depressed mothers (Trapolini et al., 2007). Similarly, depressed mothers were significantly more likely than nondepressed mothers to have adolescent offspring with depression, oppositional defiant disorder (ODD), conduct disorder (CD), and ADHD (Tully, Iacono, & McGue, 2008). However, null associations between parental depression and child ADHD and ODD (Kashdan et al., 2004) suggest that the effects of parental depression on different aspects of child psychopathology have yet to be clearly determined.

The precise association of parental depression with child psychopathology is complicated by its frequent comorbidity with other disorders. That is, the burden of comorbidity may partially account for the relationship between parent and child psychopathology (Loeber, Hipwell, Battista, Sembower, & Stouthamer-Loeber, 2009). For example, adult depression and ADHD are often comorbid (Kessler et al., 2006), and parents of children with ADHD have higher rates of depression and ADHD than parents of children without ADHD (Chronis et al., 2003; Nigg & Hinshaw, 1998). Thus, previous studies of parent psychopathology in studies of children with ADHD may have incorrectly specified the contribution of parental depression unless parental ADHD was adequately considered. Overall, the contribution of specific parental disorders with specific dimensions of offspring psychopathology has not been rigorously examined. Pfiffner and colleagues (1999) found specific associations between parental and child internalizing disorders and parental and child externalizing disorders, but associations did not extend across externalizing and internalizing dimensions (Pfiffner et al., 1999). More recently, Ellis and Nigg (2009) reported that maternal ADHD predicted child ODD and CD.

Given the high comorbidity of adult depression and ADHD (Kessler et al., 2006), and the need to specify the precise association between different aspects of parent and offspring psychopathology, the goal of this study was to test the independent contribution of parental

depression and parental ADHD to child internalizing problems (e.g., anxiety, depression, and somatic complaints), ADHD symptoms, and externalizing problems (e.g., aggressive behavior and ODD symptoms). We hypothesized that both parental depression and ADHD would positively and independently predict off-spring internalizing, externalizing, and ADHD problems.

Method

Participants

A total of 178 children with (n = 91) and without (n = 87) ADHD, aged 5 to 10 (72% male), were recruited through advertisements at local elementary schools, mailings to pediatric offices, presentations to self-help groups, and referrals from clinical service providers. Recruitment material described the study as an investigation of genetic and environmental influences on attention problems and hyper-activity in school-age children. Specifically, we requested that interested parents of children with and without attention problems/hyperactivity contact the study to determine eligibility. ADHD probands and controls were approximately matched on age (7.24 and 7.63 years) and sex (77% vs. 67% male), respectively. Although the overall design was case control, data were pooled across groups and analyzed dimensionally for this specific study. In return for participation, families received US$50 and a written report summarizing results of the assessment of the child's cognitive ability, academic achievement, and the intensive diagnostic ascertainment for ADHD and related problems. The sample was not epidemiological, but our recruitment yielded substantial racial–ethnic diversity that reflected metropolitan Los Angeles (54.5% parents identified their children as mixed, non-White, or Other). To improve the external validity of ADHD probands, common comorbidities (e.g., ODD, anxiety/depression) were included. Similarly, to avoid recruiting a sample of improbably high-functioning control youth, comparison children were allowed to participate if they met criteria for any disorder other than ADHD. For all children, exclusion criteria consisted of low IQ (<70), pervasive developmental disorder, seizure disorder, or any neurological disorder that prevented full participation in the study. Participants were required to live with at least one biological parent at least half the time and parent and child were each required to be fluent in English.

Procedure

Families who called our research laboratory were screened for eligibility and informed about study procedures. Eligible families were invited to an in-person assessment and received rating scales through the mail. We requested that parental ratings be based on the child's unmedicated behavior. These procedures have been used in similar ADHD samples, including the Multimodal Treatment Study of ADHD (Hinshaw et al., 1997). After obtaining consent, parents were interviewed using the computerized Diagnostic Interview Schedule for Children–fourth edition (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) and completed rating scales and additional interviews about their parenting and stressful life experiences. Assessments were conducted by intensively trained clinical psychology graduate students and BA-level staff. All study procedures were approved by the UCLA Institutional Review Board.

Measures

Beck Depression Inventory–II (BDI-II).—One parent, typically the mother (86% mother report) completed this widely used measure of depression. The BDI-II (Beck, Steer, & Brown, 1996) consists of 21 items, with answer prompts for each question ranging from 0 to 3, with higher numbers indicating greater severity. The responses to the individual items were summed to create a dimensional measure of parental depression. In this sample, the BDI-II had excellent internal consistency, with a Cronbach's alpha of .99.

The Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist.—This screening instrument obtained information on ADHD symptoms in adults. For each item of the 18 items, participants were asked to choose among four response options. In this sample, the ASRS (Kessler et al., 2005) had excellent internal consistency, with a Cronbach's alpha of .94. The sum of the items was used as our measure of adult ADHD symptoms. Previous work has demonstrated excellent convergent validity and adequate test–retest reliability (Kessler et al., 2007).

DISC-IV.—We administered the computerized DISC-IV (Shaffer et al., 2000) to each parent to ascertain *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) diagnostic criteria for a number of childhood disorders. This fully structured interview probes symptom levels, duration/persistence, age of onset, and functional impairment. Symptom counts in the present study were based on the 18 *DSM-IV* ADHD and the 8 *DSM-IV* ODD symptoms.

Disruptive Behavior Disorder Rating Scale (DBD).—Parents rated *DSM-IV* DBD symptoms with response options ranging from 0 (*not at all*) to 3 (*very much*). In this sample, the parent DBD (Pelham et al., 1992) had a Cronbach's alpha of .96. The 18 symptoms that comprise *DSM-IV* ADHD and the 8 symptoms that comprise *DSM-IV* ODD were summed to create dimensional measures of ADHD and ODD.

Child Behavior Checklist 6–18 (CBCL).—The 113-item rating scale completed by the parent yielded measures of child psychopathology. Responses were scored on a 3-point scale, from 0 (*not true*) to 2 (*very true or often true*). We utilized the total score from the following narrow-band factors: withdrawn/depressed, anxious/depressed, somatic complaints, attention problems, and aggressive behavior. The CBCL (Achenbach & Rescorla, 2001) was normed on a large sample of children aged 6 to 18. It possesses excellent test–retest and interrater reliability as well as adequate to excellent internal consistency (Achenbach & Rescorla, 2001).

Data Analytic Procedures

Initial data compilation and analysis was completed using PASW (18.0) software. To combat skew, a square root transformation was performed on all variables with the exception of the three child ADHD variables (ADHD symptoms from the DISC, ADHD symptoms from the DBD, and attention problems from the CBCL). Despite the overall case-control design of the study (i.e., ADHD pro-bands and control children), which allowed for greater variability in central constructs, we analyzed data dimensionally. Our general data analytic approach

consisted of structural equation modeling (SEM), which provides the opportunity to assess relationships among constructs that are corrected for biases attributable to random error and construct-irrelevant variance (Bollen, 1989). As an alternative to multiple regression, SEM tests complex models with several predictor and outcome variables, as opposed to separate "minitests" of model components (Tomarken & Waller, 2005). We used SEM to simultaneously compare parental depression and parental ADHD and their unique association with dimensions of child psychopathology. Specifically, we fit SEM models using EQS 6.1 (Multivariate Software Inc., 1998) and accommodated missing data using the expectation-maximization (EM) algorithm to maximize available data points.[1] No variable was missing in more than 5% of the cases. Goodness of fit of the models was assessed with the maximum-likelihood χ^2 statistic, the comparative fit index (CFI), and the root mean squared error of approximation (RMSEA; Bentler, 2006; Bentler & Dudgeon, 1996; Hu & Bentler, 1999). The CFI ranges from 0 to 1 and reflects the improvement in fit of a hypothesized model over a model of complete independence among the measured variables (Bentler, 2006). Values approaching .95 or greater are desirable for the CFI. The RMSEA is a measure of fit per degrees of freedom, controlling for sample size. RMSEA values of less than .06 indicate a relatively good fit (Hu & Bentler, 1999). We obtained suggestions for model modification from the LaGrange Multiplier (LM) test (Bentler, 2006).

Results

The scores on the BDI-II ranged from 0 to 22 but averaged in the nonclinical range of depression ($M = 7.25$, $SD = 6.24$). Adult ADHD scores ranged from 0 to 68 ($M = 25.55$, $SD = 13.79$). As expected, a significant positive association was found between parental depression and ADHD scores (Table 1), thus necessitating tests of their independent contribution.

The means, standard deviations, and ranges of all child psychopathology variables are available in Table 2. Given that the sample was derived from a case-control study of ADHD, ADHD scores from the CBCL attention problems, DBD, and DISC showed considerable variability. Child psychopathology measures within the same domain were highly correlated. Notably, there were a significant number of positive associations across different dimensions of child psychopathology (i.e., ADHD and externalizing variables, see Table 1).

The structural model (Figure 1) was constructed in a manner consistent with the study's hypotheses on the relationship between parent psychopathology and child psychopathology. As such, it contains three factors representing internalizing, externalizing, and ADHD symptom clusters, all of which were hypothesized to be associated with both parental depression and parental ADHD symptoms and set to covary. In addition, parental scores on depression and ADHD symptoms were set to covary. Estimation was done using normal theory maximum likelihood. Because of significant deviations from normality in variable distribution indicated by Mardia's standardized multivariate kurtosis measure ($g_{2,P} = 9.68$; scores larger than 3 indicate nontrivial kurtosis; Bentler, 2006), we employed robust

[1]This method also provides estimates of population means of variables. As these are not relevant to our hypotheses, they are not presented in results or figures.

statistics in model evaluation. This model provided suboptimal fit, Satorra–Bentler scaled $\chi^2(55, N = 178) = 109.92$, $p < .001$, with CFI = .94 and RMSEA = .108 (standardized parameters for the model are given in Figure 1).

Although each indicator of the three domains of child psychopathology provided good loadings onto their own factor, we observed considerable correlation among the three factors, suggesting that these three factors might have loaded onto a secondary factor that represented a latent dimension underlying the three forms of child psychopathology. Consequently, a second-order factor was included in which all three child psychopathology dimensions loaded significantly. We then tested this model by entering parental ADHD and parental depression variables as predictors of the second-order factor. The LM test suggested an improved model by the addition of a parameter between parental depression and the child internalizing factor. Consequently, the model was adjusted by allowing an additional path for the specific relationship between parental depression and the child internalizing factor. In addition, errors between the dependent variables of the child internalizing factor and attention problems were allowed to covary given that these were all drawn from the same instrument (CBCL). We also found that the correlation of the second-order factor disturbance and DISC ODD symptoms improved model fit. Paths between errors and observed variables are not shown in the figure. This final model presented in Figure 2 provided better fit than the initial model, Satorra–Bentler scaled $\chi^2(55, N = 178) = 49.28$, $p = .02$, CFI = .99, RMSEA = .058. As evidenced in Figure 2, there was a significant association between parental ADHD and the general child psychopathology factor. However, in this model, parental depression did not significantly predict the second-order child problems factor. Notably, the addition of a path from parental depression directly to the child internalizing factor was significant. These observed relationships suggested that parental ADHD was associated with multiple aspects of child psychopathology, but parental depression was specifically associated with child internalizing problems.

Discussion

We used SEM to estimate the unique contribution of parental depression and ADHD to multiple dimensions of child psychopathology. Based on previous conceptual and taxonomic evidence, we created a three-factor model to represent a priori domains of child psychopathology: internalizing, ADHD, and externalizing symptoms (Achenbach & Edelbrock, 1978; Hinshaw, 1987). We observed substantially high intercorrelations among internalizing, ADHD, and externalizing problems that necessitated the inclusion of a second-order factor of child psychopathology, common to these dimensions. The addition of the second-order child psycho-pathology factor and the subsequent model alterations significantly improved model fit. Both the initial and final model illustrated that parental ADHD was significantly associated with the second-order child problems factor, whereas parental depression was specifically associated with child internalizing problems.

The lack of specificity for parental ADHD and child psychopathology is consistent with previous work on the association of parental ADHD and negative parenting behaviors that are correlated with many forms of child psychopathology. For example, ADHD symptoms in expectant mothers were inversely associated with self-efficacy and positive expectations of

the infant and their future maternal role (Ninowski, Mash, & Benzies, 2007). Parenting self-efficacy was also inversely related to overreactive parenting in mothers of children with ADHD (Gerdes et al., 2007), and parents of children with ADHD reported lower self-efficacy than parents of children without ADHD (Rogers, Wiener, Marton, & Tannock, 2009). Among mothers with young infants, those with high levels of ADHD reported lower levels of parental satisfaction (Watkins & Mash, 2009). Thus, parental ADHD may have downstream effects on children partly because of their cognitive biases/distortions and poor self-efficacy. In a sample of 82 mothers with children aged 3 to 6, maternal ADHD was significantly associated with lower parenting self-esteem (i.e., deficit in perceived knowledge and skill required for effective parenting) and difficulties executing appropriate and necessary parenting behaviors (Banks, Ninowski, Mash, & Semple, 2008). Moreover, these same mothers self-reported higher levels of ineffective parenting, particularly inconsistent discipline styles. The impact of ADHD on parenting was further suggested when 23 mothers with ADHD who were treated with methylphenidate utilized significantly fewer negative parenting techniques (e.g., inconsistent parenting, corporal punishment; Chronis-Tuscano et al., 2008). Although the precise mechanism(s) underlying the association has not been fully discerned, results from our study further substantiated the nonspecific, but robust, contribution of parental ADHD to child psychopathology.

Although our study suggested that parental ADHD symptoms predicted multiple dimensions of child psychopathology, it also uncovered a unique relationship between parental depression and child internalizing problems, which was consistent with previous work (Pfiffner et al., 1999). However, our findings diverge from other studies that showed parental depression predicted ADHD and externalizing problems, although most previous studies test parental ADHD and depression simultaneously (e.g., Kane & Garber, 2009; Kashdan et al., 2004; Kepley & Ostrander, 2007; Trapolini et al., 2007; Tully et al., 2008), despite their frequent comorbidity. Therefore, comorbid ADHD may partially explain the observed relationship between parental depression and noninternalizing domains of child psychopathology previously reported (e.g., Trapolini et al., 2007; Tully et al., 2008).

A prominent area of focus for comorbid parental ADHD and depression has been parenting. Parental depression accounted for some variance in the positive association of parental ADHD and negative parenting (Harvey, Danforth, Eberhardt McKee, Ulaszek, & Friedman, 2003), suggesting that both disorders may relate to negative parenting behaviors. In a separate study, maternal ADHD significantly incremented predictions of inconsistent parental discipline strategies beyond the effect of maternal depression (Chronis-Tuscano et al., 2008). In light of previous research that parental ADHD was associated with negative parenting practices (Chronis-Tuscano et al., 2008; Harvey et al., 2003), it is possible that parenting may mediate the relationship between parental ADHD and child problems. Previous work suggested that the effects of poor parenting were non-specific, predicting childhood internalizing and externalizing problems (Berg-Nielsen, Vikan, & Dahl, 2002). The symptoms of ADHD (i.e., inattention, overactivity, and impulsivity) may negatively affect the ability of parents to effectively monitor their children (Kendziora & O'Leary, 1993). Case studies (Evans, Vallano, & Pelham, 1994) and larger empirical studies (Murray & Johnston, 2006) suggest that ADHD symptoms were associated with poorer parental

monitoring and inconsistent management techniques, which may constitute a potential pathway to a broad range of child problems as suggested by our results.

Another possible explanatory factor underlying the correspondence of parent and child psychopathology is shared genetic liability. Approximately, 25% to 30% of parents of children with ADHD have significant ADHD symptoms themselves (Barkley, 1990), a rate that far exceeds parents of children without ADHD (Smalley et al., 2000). Given that dimensions of ADHD and externalizing problems are significantly heritable (Levy, Hay, McStephen, Wood, & Waldman, 1997; Rhee & Waldman, 2002) and that parenting behaviors are at least moderately heritable (Hur & Bouchard, 1995), the association of parental ADHD and/or depression with child psychopathology may actually reflect multiple forms of gene–environment interplay, including gene–environment correlation and interaction (Jaffee & Price, 2007). A common polymorphism in the dopamine transporter gene was associated with observed negative maternal parenting behavior (Lee et al., 2010), and this same variant has been implicated in studies of ADHD and externalizing behavior (Waldman et al., 1998; Young et al., 2002). In other words, the genetic influences underlying parental and child psychopathology may be shared. Behavior genetic studies suggest that genetic factors influence the transactional network within families (Braungart, Fulker, & Plomin, 1992), with genetic factors significantly accounting for the association between parent negativity and child adjustment (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). Future research should utilize genetically informative designs to examine the plausibility of disrupted parenting as a mediator of parent and child psychopathology.

We note at least two important limitations of this study. First, the associations reported were based on cross-sectional data; thus, directionality of effects could not be clearly discerned, especially given the likelihood of “child effects” on parent psychopathology (Barkley 1989; Lytton, 1990). Previous work demonstrated that parents showed elevated anxiety, depression, and hostility after interacting with children with externalizing behavior problems (Pelham et al., 1997). Moreover, without establishing temporal ordering through repeated measures, we were unable to determine whether observed associations were potentially causal (Kraemer et al., 2001). Second, we used parent report for all psychopathology measures, which may bias the associations (i.e., shared method variance; Campbell & Fiske, 1959). For example, parental depression may bias reports of offspring ADHD and behavior problems (Chi & Hinshaw, 2002). Future research should adopt multi-informant method/approaches to parse the separate roles of psychopathology and potential reporting bias in the development of child psychopathology.

The current study observed a specific association of parental depression with child internalizing problems, whereas parental ADHD associated more generally to a broad range of child psychopathology. These findings suggest that parental ADHD should be considered more carefully in explanatory models of child psychopathology. Clinicians should carefully consider parental ADHD and depression in assessment and intervention. Specifically, parental measures of ADHD and depression may provide some traction on specific domains of child problems. We reiterate the suggestions of Pfiffner and colleagues (1999) that clinicians should focus on the amelioration of parental ADHD in the context of family or child-centered therapy. Multimodal interventions targeting parent and child psychopathology

may be more efficacious than an exclusive focus on child psychopathology. Finally, our study included linear representations of ADHD and depression, emphasizing the potential effect that nonclinical levels of ADHD and depression may have on child psychopathology. Thus, the exploration of the correlates of parental ADHD, in addition to parental depression, may be a rich area for future research given its robust association with multiple dimensions of child psychopathology.

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Bios

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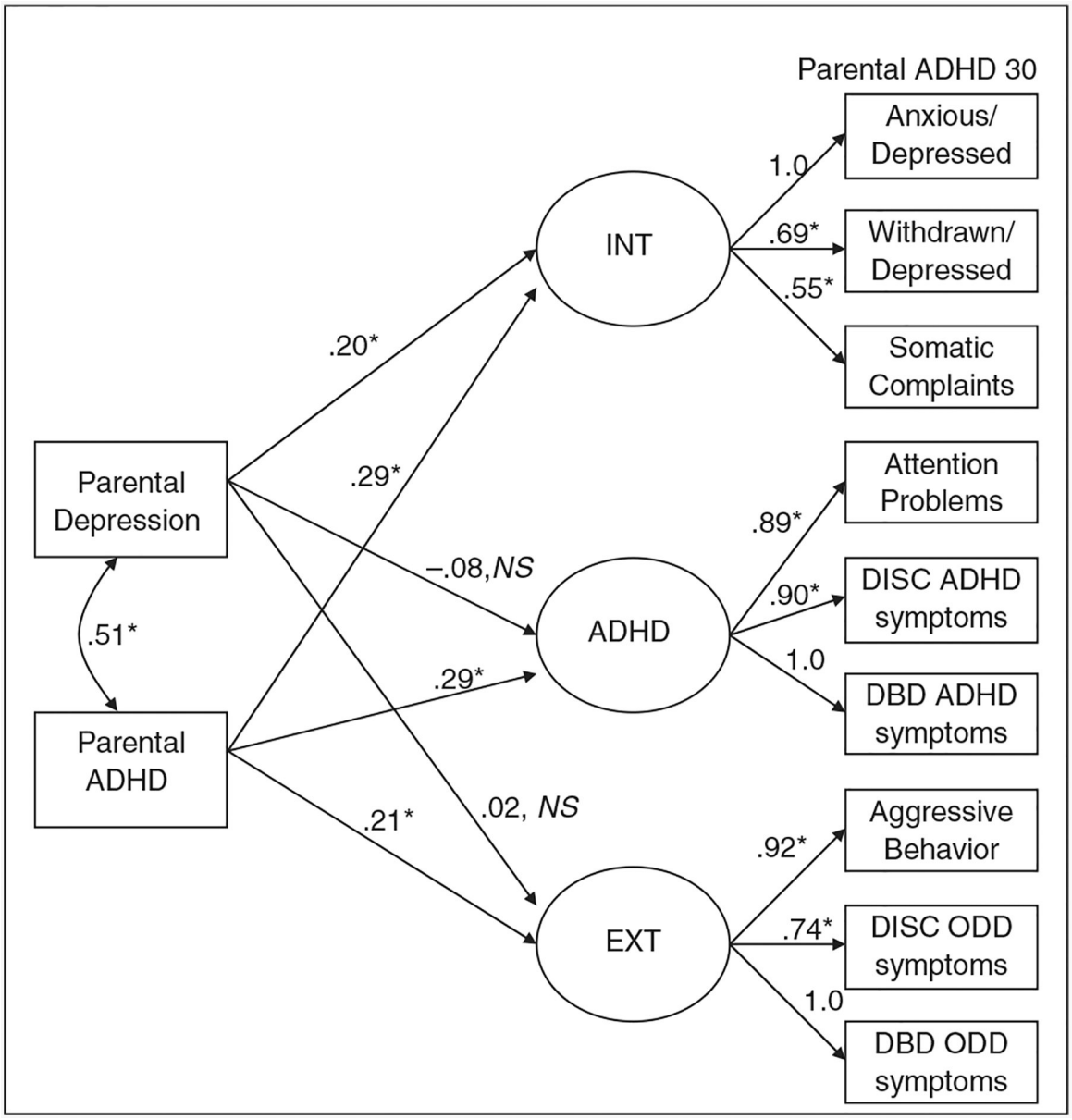

Figure 1.

Theoretical model of the association between parental depression and parental ADHD with child internalizing, ADHD, and externalizing symptom factors ($N = 178$) Note: INT = internalizing factor; EXT = externalizing factor. Standardized parameter estimations are shown (*$p < .05$ on unstandardized coefficients) and errors are not shown.

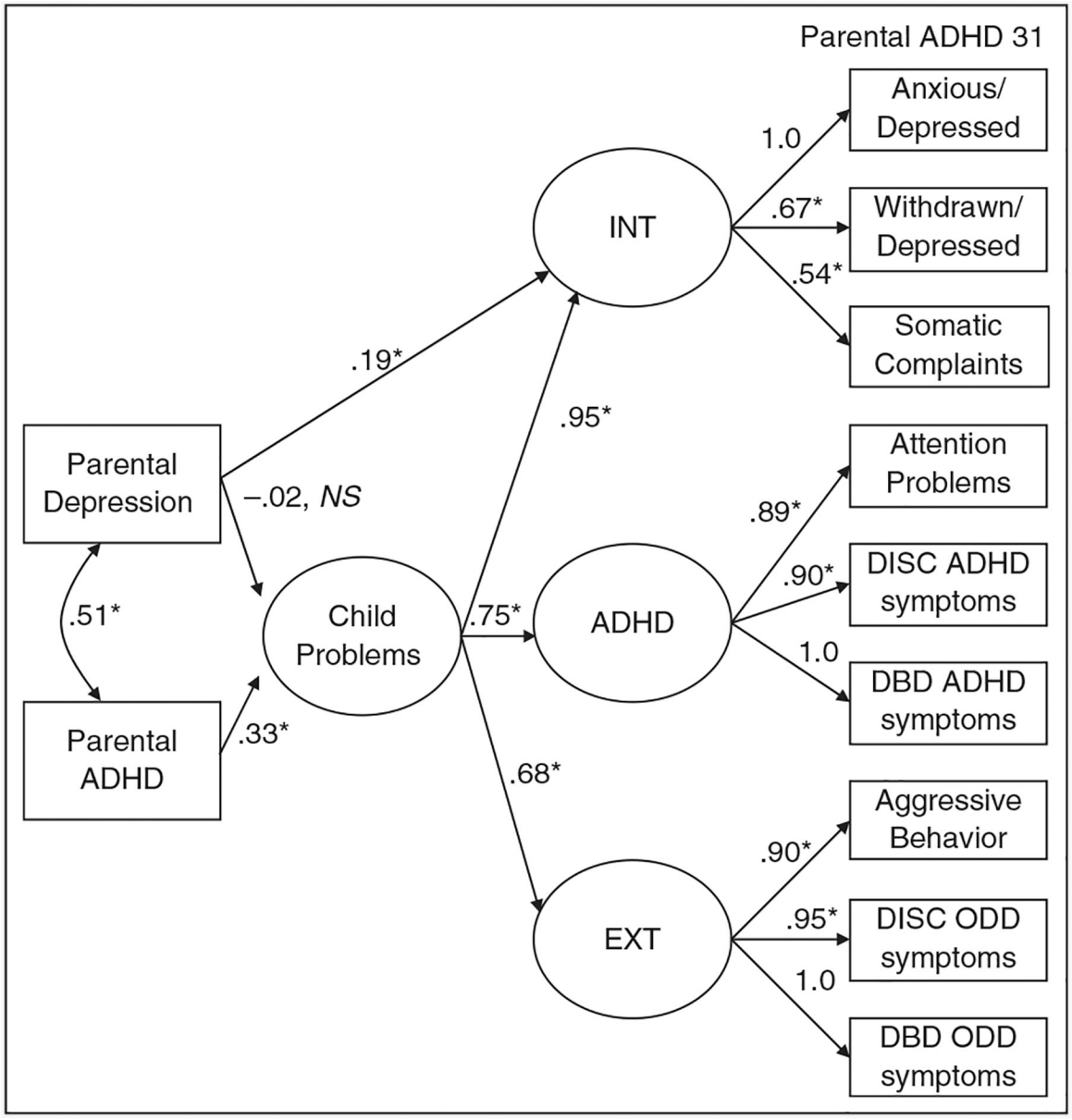

Figure 2.

Final model of the association between parental depression and parental ADHD with child internalizing, ADHD, and externalizing symptom factors ($N = 178$) Note: INT = internalizing factor; EXT = externalizing factor. Standardized parameter estimations are shown (*$p < .05$ on unstandardized coefficients) and errors are not shown.

Table 1.

Correlations Between Structural Equation Modeling Variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Parental depression	1	.49****	.26***	.17**	.25***	.05	−.00	.08	.07	.14*	.10
2. Parental ADHD		1	.33****	.16*	.28****	.20**	.16*	.21**	.16*	.19**	.16*
3. Anxious/depressed			1	.58****	.45****	.45****	.27****	.33****	.50****	.30****	.46****
4. Withdrawn/depressed				1	.31****	.44****	.15*	.24***	.48****	.30****	.45****
5. Somatic complaints					1	.35****	.24***	.26****	.33****	.21***	.29****
6. Attention problems						1	.78****	82****	.63****	.45****	.56****
7. DISC ADHD symptoms							1	.86****	.53****	.54****	.54****
8. DBD ADHD symptoms								1	.58****	.45****	.64****
9. Aggressive behavior									1	.68****	.77****
10. DISC ODD symptoms										1	.65****
11. DBD ODD symptoms											1

Note: DISC = Diagnostic Interview Schedule for Children; DBD = Disruptive Behavior Disorder Rating Scale; ODD = oppositional defiant disorder. Correlations were calculated prior to expectation-maximization for missing data imputation.

* $p < .10$.

** $p < .05$.

*** $p < .01$.

**** $p < .001$.

Table 2.

Descriptive Statistics of the Child Psychopathology Measures (N = 178)

	M (*SD*)	Range
Anxious/depressed	3.89 (3.33)	0–15
Withdrawn/depressed	1.60 (1.90)	0–8
Somatic complaints	1.96 (2.36)	0–11
Attention problems	7.74 (4.89)	0–19
DISC ADHD symptoms	7.95 (5.52)	0–18
DBD ADHD symptoms	21.24 (13.88)	0–53
Aggressive behavior	7.05 (6.18)	0–28
DISC ODD symptoms	2.26 (2.38)	0–8
DBD ODD symptoms	6.18 (4.98)	0–22

Note: DISC = Diagnostic Interview Schedule for Children; DBD = Disruptive Behavior Disorder Rating Scale; ODD = oppositional defiant disorder.