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Potential Cognitive, Parenting, and Developmental Mediators of the Relationship Between ADHD and Depression

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The authors investigated the role of parent behavior management and locus of control in mediating the relationships between ADHD (attention-deficit/hyperactivity disorder) and depression in a community sample of 232 children with ADHD and 130 community controls. Results supported the hypothesized path models to depression for older and younger subjects. For older subjects (10 years and older), a cognitive variable, locus of control, partially mediated the relationships between ADHD and parent management and depression. In addition, parent management partially mediated the relationships of ADHD with locus of control and depression. For the younger group (under 8 years old), however, locus of control did not mediate the effects of parent management and ADHD on depression. Consistent with developmental theories, only an environment variable, parent management, explained the relationship between ADHD and depression for this younger group. For children 8–9 years old, both locus of control and parent management partially mediated the ADHD–depression relationship; however, similar to the younger children, locus of control did not mediate the parent management–depression relationships. Implications for designing interventions and prevention strategies for children with ADHD are discussed.

Keywords: attention-deficit/hyperactivity disorder (ADHD), depression, comorbid disorders, multiple sources of information, community sample

A number of studies have documented a high rate of comorbidity between ADHD and depression (Angold, Costello, & Erkanli, 1999; Jensen et al., 2001;Jensen, Shervette, Xenakis, & Richters, 1993); moreover, depression appears to be equally prevalent among children with the inattentive versus combined subtype of ADHD (Crystal, Ostrander, Chen, & August, 2001; Powers, Costigan, Eiraldi, & Left, 2004). Despite the strong association between ADHD and depression, little is known about the mediators of this relationship. According to *The Diagnostic and Statistical Manual* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000), children with ADHD are typically viewed as having a disorder of early developmental origins; in contrast, depression appears to have a later onset with prevalence rates increasing with age (Kessler, 2002). These findings suggest that ADHD precedes the onset of depression, offering a clue about the development of the dual diagnosis. Understanding processes that intercede the two conditions could yield new insights about the nature and etiology of this particular type of comorbidity and lead to treatment and prevention innovations; indeed, some authors have suggested that the study of comorbidity is the most pressing issue in child psychopathology research and practice (Jensen, 2003). When one draws from extant literature regarding the development of depression in children and adults, two constructs emerge as possible mediators of the ADHD and depression relationship: child cognitions and parenting behaviors.

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Much of the literature on depression has focused on testing the cognitive hypothesis, that is, the prediction that cognitive variables are the most proximal causes of depressive symptoms (see Garber & Horowitz, 2002, for a review). In particular, researchers have found strong relationships between childhood depression and control-related beliefs such as external locus of control (McCauley, Mitchell, Burke, & Moss, 1988), low perceived competence (Cole, Peeke, Dolezal, Murray, & Canzoniero, 1999), and low perceived control and contingency (Weisz, Southam-Gerow, & McCarty, 2001). Yet, most studies to date have focused on older children or adolescents and have not explained the early developmental origins of depression-related cognitions.

In one of the few longitudinal studies, Nolen-Hoeksema, Girgus, and Seligman (1992) concluded that negative life events are directly linked to depression during the early childhood years and that negative cognitions occurring during late childhood reflect the scarring that evolves from early exposure to stressful interactions with the environment. As adolescence nears, proximal sources of stress may have a less direct effect on depression; eventually, negative cognitions emerging from early exposure to stress act as an independent risk factor for depression (Nolen-Hoeksema et al.).

These findings suggest that understanding the etiology of childhood depression will likely require a modification of some of the causal models that explain depression in adults. Cole and Turner (1993) have suggested that, whereas cognitive factors moderate the effects of stress in adults, cognitions may mediate the effects of these external factors on depression in children. That is, environmental factors may lead to distorted cognitive sets in children, which, in turn, make them vulnerable to depression. Supporting this hypothesis, Cole and Turner found that negative attribution styles and cognitive distortions mediated or partially mediated the

effects of negative life events on depression in 356 fourth, sixth, and eighth grade students.

One environmental stressor, parenting behaviors, may play a pivotal role in the development of child cognitive styles and, concomitantly, in the development of depression. Researchers have documented disrupted parent–child relationships in children who are depressed (Gerlsma, Emmelkamp, & Arrindell, 1990; Rapee, 1997; Stark, Humphrey, Crook, & Lewis, 1990; Walker, Garber, & Greene, 1993). Furthermore, theorists have proposed that negative family relations provide fertile ground for children to develop negative schemas known to be associated with depression (Beck & Young, 1985; Randolph & Dykman, 1998). Recent research has suggested that parenting behaviors marked by control, intrusiveness, inconsistency, and overprotection may compromise children's control-related beliefs (Carton & Nowicki, 1994; Chorpita, Brown, & Barlow, 1998; Rudolph, Kurlakowsky, & Conley, 2001; Skinner, Zimmer-Gembeck, & Connell, J. P., 1998). Predictable and contingent-responsive parenting, on the other hand, promotes an internalized locus of control (Schneewind & Pfeiffer, 1978).

Although previous studies have established strong links between parenting behaviors and control-related beliefs, very few studies have investigated control beliefs as a mediator of the causal path from parenting behaviors to depression (Chorpita & Barlow, 1998). Initial tests of the causal path have produced inconsistent findings. Chorpita and colleagues (Chorpita et al., 1998) tested this model with a sample of ninety-three 6 to 18 year olds and found parenting behaviors were associated with lower perceived control in the child, which, in turn predicted child depression and anxiety symptoms. This relationship has not been found uniformly across children at all developmental levels. Muris and others (Muris, Meesters, Schouten, & Hoge, 2004) focused on a rather narrow age range (12–13-year-olds) and found perceived control beliefs in children were associated with mood symptoms; however, these beliefs did not mediate the effects of parenting behaviors on depression or anxiety. These findings would suggest that future studies rely on a developmental framework (Muris et al).

Extending theories regarding childhood depression to understanding depression among children with ADHD is speculative but consistent at least with the sequence of the presentation (i.e., depression usually following ADHD). There is both empirical and theoretical support for the proposition that the central impairment associated with ADHD is related to deficiencies in self-control (Barkley, 1998); therefore, it is reasonable to expect children with ADHD to believe that they cannot influence the events that happen to them. Consistent with this premise, Linn and Hodge (1982) have reported that children diagnosed with ADHD are more inclined to report an external locus of control. Yet, children with ADHD do not uniformly report negative cognitions. Recent research would suggest that **c**hildren with ADHD typically judge themselves in an overly optimistic manner (Hoza, Pelham, Dobbs, Owens, & Pillow, 2002). Interestingly, this "positive illusionary bias" is attenuated in those with depression (Hoza et al., 2004), suggesting that children with ADHD and comorbid depression have negative self-appraisals that closely parallel the harsh judgments of them made by others. There is also evidence suggesting that control-related beliefs are differentially represented in children with externalizing symptoms and depend on the degree to which internalizing symptoms are also manifested. In a recent study, children with externalizing symptoms did not believe that they could control their behavior; however, this belief was present only if they also displayed high levels of internalizing symptoms (Han, Weisz, & Weiss, 2001). This line of research suggests that the relatively large subset of children with ADHD who develop depression might possess negative views concerning their ability to control important events in their life; moreover, there may be a realistic basis for these negative beliefs.

Although genetic or other early neurodevelopment factors are clearly implicated in the etiology of ADHD, family interactions may be important in understanding psychiatric comorbidity in children with ADHD (Barkley, 1998). For example, the impulsive, inattentive, and demanding behavior that typifies children with ADHD frequently elicits very negative, coercive, and inconsistent responses from parents and others (Barkley & Cunningham, 1979; Danforth, Barkley, & Stokes, 1991; Fischer, 1990; Mash & Johnston, 1990). These parenting behaviors are similar to those used to explain the emergence of negative cognitions associated with depression (Rudolph et al., 2001).

When considered together, the respective studies examining families of children who are depressed or who have ADHD suggest that the cognitive and behavioral dysregulation that typifies children with ADHD may lead to negative, inconsistent, and coercive exchanges with their parents. These disrupted parenting practices may contribute to the belief that events in the environment are noncontingent and uncontrollable; in turn, an external locus of control may help explain the unusually high levels of depression displayed in children with ADHD. Although these are tenable hypotheses, we are unaware of any study that has examined whether parenting practices or control-related beliefs mediate the relationship between ADHD and depression.

Normal developmental transformations also may affect the nature of the relationships between ADHD, parenting behaviors, child cognitions, and depression. For example, family factors may be more directly linked to child well-being in early childhood when children are dependent on parents for regulating affect (see Cicchetti & Toth, 1995; Ostrander, Weinfurt, & Nay, 1998; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). Although parental influence remains important throughout the childhood years, during the later stages of childhood, these influences wane as the children's social network expands (Larson & Richards, 1991; Steinberg & Silverberg, 1986). At the same time, the role of cognitions in enhancing or interfering with well-being likely grows over time. Cognitive appraisals in early childhood are unstable and more directly linked to immediate and concrete events in the environment (Cicchetti & Toth, 1995; Dweck & Leggett, 1988; Fincham & Cain, 1986; Nicholls & Miller, 1984). Cognitions become more crystallized between 8 and 9 years of age (Cicchetti & Toth; Fincham & Cain, 1986). During late childhood and early adolescence, researchers have documented stabilized cognitions that endure beyond the immediate situation (Cicchetti & Toth; Dweck & Leggett; Fincham & Cain, 1986; Harter, 1989; Ostrander, Nay, Anderson, & Jensen, 1995). Evidence of transient cognitions in early childhood makes it questionable whether cognitions can play a significant role in mediating the relationship between parenting behaviors and depression in younger-aged children (Cicchetti & Toth; Fincham & Cain, 1986). Consistent with this contention, Nolan-Hoeksema et al. (1992) found that life events in third grade (approximately 8 years of age) predicted

depressive symptoms in fourth grade even after controlling for cognitions. Other studies have documented a mediating role of cognitions for children around the age of 10 or 11 (Cole & Turner, 1993). In short, developmental changes likely play an important role in determining how family and cognitive factors explain the relationship between depression and ADHD.

On the basis of these developmental considerations, we expected a weaker influence of the cognitive variable and predicted that only parent behavior management would mediate the path between ADHD and depression for children under 8-years-old. For children ages 8 to 9, we expected to find evidence of increasing cognitive influence over the path from ADHD to depression as cognitions begin to stabilize during this age frame. We hypothesized that behavior management and locus of control would mediate the path between ADHD diagnosis and depressive symptoms in older children similar to findings from previous research about the development of depression in the general population (Cole & Turner, 1993). In addition, because family interactions appear to have an impact on children throughout the elementary school years (Larson & Richard, 1991; Steinberg & Silverberg, 1986), we expected parent management practices to play an important role in explaining the relationship between ADHD and depression for all groups.

Method

Participants

A community population of 7,231 children, initially in Grades 1–4, attending 22 schools, were screened by using a sequential, two-stage assessment strategy (see August, Realmuto, Crosby, & MacDonald, 1995, for a detailed description). On the basis of parent and teacher ratings, each exceeding 1.75 *SD* units above the mean on the 10-item Hyperactivity Index (HI) of the Revised Conners Rating Scales (Goyette, Conners, & Ulrich, 1978), 309 (4.3%) children were screened positive and exhibited high levels of problematic behavior across settings. A comparison sample of children, not rated as problematic by either parent or teacher, was also identified, which included 144 children selected through a stratified random sampling procedure to be proportionally equivalent to the problematic children in terms of school, age, and gender. The beginning phase of the identification process began in the early spring, and the final phase was completed 8 months later.

After the screening process, the sample consisted of 309 problem and 144 nonproblem children (total $N = 453$), ranging in age from 6.60 to 11.75 years. The sample was 79% male, 95% Caucasian and predominantly middle class, although all socioeconomic levels, as determined by the Hollingshead (1975) index, were represented.

Procedures

Several months after the screening, parents completed mailed questionnaires, and teachers completed questionnaires distributed in the schools. At the same time, child psychiatric diagnoses were generated through use of the Diagnostic Interview for Children and Adolescents–Revised-Parent Version (DICA-R-P; Reich, Shayla, & Taibelson, 1992; Reich & Welner, 1990). During the interval between screening and selection, one school district's catchment area was redrawn, and some students were assigned to a new school district that did not participate in the study. Reassignment to a nonparticipating school district was the primary reason that 75 positive screens were unable to be interviewed with the DICA-R-P. A comparison of the participants and nonparticipants found the groups to be indistinguishable on socioeconomic status (SES), family size, single parent status, the ages of parents and children, and their scores on the respective parent and teacher HI screening measures. The DICA-R-P was administered to parents over the telephone by eight trained research assistants (see August et al., 1995, for additional details). Research assistants participated in an intensive training program that included video training and role-play. An independent rater who assessed 20% of each assistant's interviews was used to obtain interrater reliability. Interrater reliability for the *DSM–III–R* diagnoses of ADHD was .97.

Selection Criteria for Children With ADHD

Because the DICA-R-P is based on *DSM–III–R* criteria, it was necessary to modify the criteria for ADHD so as to maximize concordance with *DSM–IV–TR* diagnostic criteria. Creating this analog to the *DSM–IV–TR* criteria involves a method that is consistent with the approach we have reported elsewhere and that has demonstrated excellent discriminant and convergent validity (Crystal et al., 2001; Ostrander, Weinfurt, Yarnold, & August, 1998). This procedure divided the ADHD sample into 43% combined and 50% inattentive subtypes; the predominantly hyperactive subtype represented only 6%. Interrater reliability (kappa) for the reconstructed subtypes was 0.96 for the inattentive and hyperactive–impulsive subtypes and 1.00 for the combined subtype. At the end of the identification process, the sample of children with ADHD consisted of 109 ADHD–combined type, 123 ADHD–inattentive type, and 16 ADHD–hyperactive/impulsive type ($n = 248$). The respective subtypes of ADHD did not differ in terms of age or gender distribution. Furthermore, a chi-square analysis of the parents' reports on Hollingshead's (1975) index of SES showed no significant difference among the groups in their overall distribution across Hollingshead's categories. Because of the relatively small numbers and the limited empirical support for the impulsive–hyperactive subtype, these potential participants were not included in the study, resulting in a slight reduction in the ADHD sample ($n = 232$).

Selection Criteria for Non-ADHD Community Controls

All negative screens were included as potential community controls ($n = 6,589$). Final selection of non-ADHD community controls was determined by the following additional criteria. Of students scoring less than 1 *SD* above the mean on the teacher version of the HI, 10% were randomly selected and were further stratified to match the proportional representation of the participants with ADHD according to school, grade, and gender (August et al., 1995). Control subjects were ultimately identified if they also reported no history of psychotropic medication use and had no prior history of clinical assessment for behavioral problems. Diagnostic interviews were not administered to comparison students. However, the mean scores on the parent and teacher screening measure (HI) were at floor levels, suggesting only a remote possibility that any of these children would be diagnosed with a disruptive behavioral disorder. There were 144 subjects that were originally identified as community controls; however, a total of 130 agreed to participate in the study and completed at least some of the assessment tools. Most nonresponders were lost because of reassignment to another school district or move from their original school district. The final sample included 232 children with ADHD and 130 community controls.

Instruments

Parent Measures

Behavior Management Self-Assessment (BMSA; August, Realmuto, Crosby, & MacDonald, 1995). The BMSA was completed by the parents and is a 15-item Likert-type questionnaire ($\alpha = .81$) rating parenting skills related to the effective and consistent management of disruptive behavior, use of positive reinforcement, and monitoring child's whereabouts. The items on this scale were adapted from the Parental Practices Scale (Stray-

horn & Weidman, 1988). Representative items include "I praise my child for doing something I approve of"; "I am not consistent in disciplining my child"; "I do a good job of disciplining my child"; "When I ask my child to do something, I am clear and to the point in my request"; "I have to nag and/or scold my child to get him/her to do something I have asked"; "I yell or scream at my child when he or she gets on my nerves"; and "When I give my child commands, I do not follow through to see that he or she obeys." Higher scores on the BMSA reflect less effective behavioral management and are associated with high levels of parent-reported family conflict as well as low levels of family cohesion and expressiveness (August et al., 1995). In addition, high scores are also associated with poorer child adjustment in areas reflecting self-concept, social skills, and problem behaviors (August et al., 1995).

Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL Parent Rating Scale contains 118 items, each scored on a 3-point scale. The scale includes two broad dimensions, internalizing and externalizing. The CBCL subscales have evidence of adequate internal consistency, test–retest reliability, concurrent validity, and discriminant validity (Achenbach). The Depression–Anxiety subscale was used in the present study.

Behavioral Assessment System for Children (BASC; Reynolds & Kamphaus, 1992). The BASC is a multisource and multidimensional assessment system that includes separate report measures that are derived from respective self, parent, and teacher respondents. The BASC was constructed by using structural equation modeling; as such, the nonoverlapping content of the respective scales reflects a "pure" index of the constructs of interest. This allowed for greater precision in the respective scale construction; thus, the individual scales should correlate with each other only to the extent predicted from the correlations between the constructs underlying the scales (and not because of overlapping items or item content that overlaps with an adjacent construct). The need for item purity is particularly important when examining the relationship between constructs that are expected to be highly related (e.g., depression and negative cognitions).

BASC-Parent Report (BASC-PRS; Reynolds & Kamphaus, 1992). The BASC-PRS is a multidimensional measure designed to assess adaptive and problem behaviors in children and adolescents. The parents participating in the study completed the child version of the BASC-PRS, which comprises 130 items rated on 4-point frequency scales ranging from 0 (*never*) to 3 (*always*). The Depression subscale was used in the present study, and the manual reports very good internal ($\alpha = .86$) and test–retest reliabilities ($r = .87$). In addition, the PRS Depression subscale has good convergence with other measures reflecting affective disturbance, and this relationship was distinct from measures of externalizing disorders.

Child Measures

BASC-Self-Report Scale (BASC-SRS; Reynolds & Kamphaus, 1992). The BASC-SRS is a self-report measure for children ages 8 to 11 years and comprises 152 items that are rated as either true or false by the child. A subset ($n = 86$) of the participants was between 7 and 8 years of age when this instrument was administered. However, no significant differences between scores of children below 8 years and those above 8 years old were found for any of the self-report measures used in the present investigation. The correlation between the two scales used in this study (depression and locus of control) was identical ($r = .83$) for the under-8 group and a slightly older age group (8.0 to 8.5 years). Moreover, the correlations between these scales and the other scales that constitute the BASC-SRS were not significantly different across these two age groups ($p > .05$). Thus, children under the age of 8 seemed to respond to this self-report in a very similar manner as children who were slightly older. These findings suggest that the discriminant and convergent validity of the BASC was very similar for children under and over 8 years of age. A more detailed description of the respective BASC-SRS scales used in this study follows:

Locus of Control subscale. The Locus of Control subscale of the BASC-SRS comprises 16 items that assess the individual's perception of who or what controls the various events of one's life. High scores denote an external locus of control, defined as the perception that success or failure is determined by forces beyond one's control. Representative items include "I am blamed for a lot of things I don't do"; "My parents expect too much of me"; "My parents have too much control over my life"; "I can't seem to control what happens to me"; "Things go wrong for me, even when I try hard"; and "Bad things just happen." The manual documents high levels of test–retest reliabilities ($r = .76$ over 8 weeks) and internal consistency ($\alpha = .87$), as well as good discriminant and convergent validity.

Depression subscale. The Depression subscale of the BASC-SRS comprises 17 items that assess feelings of unhappiness, inability to experience pleasure, and dejection. The manual reports excellent internal ($\alpha = .88$) and test–retest reliability ($r = .75$). Good convergent and discriminate validity was found when the depression scale was compared with emotional and behavioral functioning derived from other self, parent, and teacher measures.

Child Depression Inventory (CDI; Kovacs, 1992). The CDI is a 27-item self-report instrument designed to assess the number and extent of depressive symptoms. Scores range from 0 to 54, with higher scores reflecting greater depression. The CDI has acceptable internal consistency and validity (Kovacs, 1992).

Composite Measure

Depression composite. To ensure that the strength of the relationship between the respective independent and dependent variables was not differentially influenced by shared method variance (i.e., source variance), the depression composite score was derived from two child-report (the CDI and BASC-SRS Depression subscale) and two parent-report measures (BASC-PRS Depression subscale and the CBCL Depression–Anxiety subscale). The composite was calculated by standardizing each measure and summing these standardized values into a single composite score. This compensatory data aggregation strategy helped minimize source bias (e.g., overinflated estimates of shared variance attributable to reliance on a single informant) between the composite measure and all other study variables. In addition, a simple aggregation of sources and instruments has proven to result in a more practical and valid means of combining data sources than alternate methods that rely on statistical or clinically derived weighting procedures (Piacentini, Cohen, & Cohen, 1992). For children under 8, the α on this composite measure was .74, the item-to-total correlation ranged from .40 to .69, and the average interscale correlation was .43. For children 8 to 10, α was .77, the item-to-total correlation ranged from .42 to .65, and the average interscale correlation was .47. The α level for the oldest age group (ages 10 and over) was .82, the item-to-total correlation ranged from .52 to .73, and the average interscale correlations was .55. Higher scores reflected a higher level of depressive symptoms.

Results

Data Analysis Overview

According to Baron and Kenny (1986), a mediating model must meet the following criteria: The mediating variable (e.g., parent management) must significantly relate to the independent variable (e.g., ADHD), the dependent variable (e.g., depression) must significantly relate to the independent variable, and the dependent variable must significantly relate to the mediating variable. A mediating role is suspected when a previously significant relationship between the independent variable (e.g., ADHD) and dependent variable (e.g., depression) is substantially reduced when the mediator variable (e.g., parent management) is entered into the equation (Baron & Kenny).

We first calculated univariate relations between study variables to satisfy the first requirement of mediation (e.g., significant univariate relations between variables). Next, we conducted a hierarchical regression analysis to calculate the effects of all study variables on the dependent variables with the proposed mediators in the model. Any significant standardized betas in the final step of the hierarchical regression were used as path coefficients to construct the final path model (see Klem, 1995).

Of the individual measures used in this study, the BMSA had the highest percentage of missing data (29%); in contrast, all the participants completed self-report data (i.e., CDI and BASC-SRS), and a high percentage of the participants completed the parent CBCL (11% missing data). Missing data were associated with lower SES. Though this effect was very small, it may limit the extent to which findings can be generalized to samples with a lower SES (Cohen, Cohen, West, & Aiken, 2003). Missing data were unrelated to other demographic variables or any of the respective dependent and independent variables. In addition, participants with and without missing data did not differ on any of the individual scales that constitute the CBCL-P, BASC-PRS, or BASC-SRS. Therefore, omitting cases with missing data from our analysis allowed us to achieve relatively unbiased estimates (Cohen et al.).

Preliminary Analyses

Descriptive statistics and preliminary Pearson correlation analyses were calculated to determine the univariate relations among study variables. As expected, there were significant relationships among all study variables. The strongest relationships were found between the depression composite and ADHD ($r = .68$), locus of control ($r = .63$), and parent management ($r = .48$). The significant univariate relationships between ADHD, parent management, locus of control, anxiety, and depression variables satisfied the first requirement of mediation analysis (i.e., independent variables, dependent variables, and mediators should be significantly related) suggesting that mediational analysis could proceed (Baron & Kenny, 1986).

Mediation and Path Analyses

To test whether locus of control mediated the relationship between ADHD and parent-management variables and depression, we conducted a series of hierarchical regression analyses with depression as the criterion by using the following steps: (a) ADHD–IV diagnosis (1 = a diagnosis of ADHD, 0 = controls) was entered in the first step to determine its relationship with depression without the proposed mediators in the model; (b) parent management was added at the second step to determine whether the significant relations that emerged from the first step disappeared or decreased when we controlled for the proposed mediator; and (c) locus of control was entered at Step 3 to determine variables with direct effects on depression, controlling for all other variables. We repeated Steps 1 and 2 using locus of control as the criterion and Step 1 using parent management as the criterion. All analyses were conducted three times, once with the younger group ($n = 52$; 6.6–7.9 years old), once with the middle group ($n = 113$; 8.0–9.9 years old), and once with the older group ($n = 58$; ranging from 10.0–11.7 years old).

The model for the older group was tested first. ADHD, parent management, and locus of control were entered, in order, as predictors of depression (see Table 1). All independent variables

Table 1
Summary of Mediation Analyses for ADHD, Depression, Locus of Control, and Parent Management Variables in Older Children (10 Years Old and Older)

Variable	β	*B*	*SE*	$p <$	R^2	$R^2\Delta$
Model 1: Predicting depression ($n = 58$)						
Step 1:					.47***	
ADHD	.68	4.98	0.71	.000***		
Step 2:					.56***	.09***
ADHD	.50	3.67	0.77	.000***		
Parent management	.36	0.15	0.04	.001**		
Step 3:					.68***	.12***
ADHD	.36	2.65	0.71	.000***		
Parent management	.22	0.09	0.04	.025*		
Locus of control	.41	1.45	0.34	.000***		
Model 2: Predicting locus of control						
Step 1:					.25***	
ADHD	.50	1.06	0.25	.000***		
Step 2:					.33***	.08**
ADHD	.34	.71	0.27	.012*		
Parent management	.33	.04	0.02	.015*		
Model 3: Predicting parent management						
Step 1:					.25***	
ADHD	.50	8.87	2.06	.000***		

Note. ADHD = attention-deficit/hyperactivity disorder.
* $p < .05$. ** $p < .01$. *** $p < .001$.

significantly predicted depression in the final step and, collectively, accounted for over two thirds of the variance in depression scores (R^2 = .68). When locus of control was added at Step 3, though, a notable drop in β was observed for all previously added predictors (ADHD β dropped from .50 to .36 and parent management β dropped from .36 to .22). A follow-up Sobel test using the standardarized and unstandardized betas and the associated error terms from Table 1 revealed that these reductions were due to a significant mediation effect of locus of control on parent management (Sobel = 2.17; $p < .03$) and ADHD (Sobel = 2.23, $p < .026$). In addition, the ADHD–depression relationship was partially mediated by parent management (Sobel = 3.04; $p < .002$).

The same analyses were repeated with locus of control as criterion. In combination, ADHD and parent management explained 33% of the variance in locus of control scores. A follow-up Sobel test indicated that the visual reduction in the β for ADHD from Step 1 to Step 2 (a .16% decrease) was due to the mediation of parent management (Sobel = 2.17; $p < .030$).

The model for the middle group was tested next. ADHD, parent management, and locus of control were entered, in order, as predictors of depression (see Table 2). All independent variables significantly predicted depression in the final step and collectively accounted for two thirds of the variance in depression scores (R^2 = .68). When locus of control was added at Step 3, though, the β for ADHD dropped .14 from the previous step, representing a significant mediation effect (Sobel = 2.99; $p < .003$). In addition, parent management significantly mediated the ADHD–depression relationship (Sobel = 1.97; $p < .048$). Unlike the results with the older children, though, locus of control did not mediate parent management's relationship with depression. In other words, for the middle group, the path from ADHD to depression was explained by parent management and locus of control; however, locus of control did not exert a mediating influence on parent management in this path.

The model for the younger group was tested last. ADHD, parent management, and locus of control were entered, in order, as predictors of depression (see Table 3). All independent variables significantly predicted depression in the final step and collectively accounted for nearly two thirds of the variance in depression scores (R^2 = .65). However, subsequent analysis with locus of control as criterion found that parent management and ADHD were unrelated to locus of control, violating the first prerequisite for mediation. Thus, locus of control did not mediate the ADHD–depression path and was dropped from subsequent analysis. Parent management, on the other hand, did mediate the ADHD–depression relationship for younger children (Sobel = 2.29, $p < .022$). In other words, for the younger group, the path from ADHD to depression was explained by parent management alone; locus of control did not exert a mediating influence on this path. Final path models for each group are depicted in Figures 1, 2, and 3.

Discussion

In the present study, we investigated the linkages between ADHD and depression, particularly the role of development in qualifying parenting and cognitive influences on the relationship. Results supported the hypothesized path models for younger and older children. Consistent with developmental theories, only an environment variable, parent management, was needed to explain the relationship between ADHD and depression for the younger group (under 8-years-old). For older children (10 years and older), parent management continued to uniquely account for part of the

Table 2
Summary of Mediation Analyses for ADHD, Depression, Locus of Control, and Parent Management Variables in Middle Children (8–9 Years Old)

Variable	β	*B*	*SE*	$p <$	R^2	$R^2\Delta$
Model 1: Predicting depression (n = 113)						
Step 1:					.48***	
ADHD	.69	4.53	0.45	.000***		
Step 2:					.51***	.03**
ADHD	.64	4.16	0.46	.000***		
Parent management	.18	0.08	0.03	.012*		
Step 3:					.68***	.17***
ADHD	.50	3.28	0.39	.000***		
Parent management	.14	0.06	0.02	.018*		
Locus of control	.44	1.36	0.18	.000***		
Model 2: Predicting locus of control						
Step 1:					.11***	
ADHD	.34	0.71	0.19	.000***		
Step 2:					.12***	.01
ADHD	.31	0.65	0.20	.002**		
Parent management	.09	0.01	0.01	.318		
Model 3: Predicting parent management						
Step 1:					.10***	
ADHD	.21	4.70	1.36	.001***		

Note. ADHD = attention-deficit/hyperactivity disorder.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3
Summary of Mediation Analyses for ADHD, Depression, Locus of Control, and Parent Management Variables in Younger Children (Under 8 Years Old)

Variable	β	*B*	*SE*	$p <$	R^2	$R^2\Delta$
Model 1: Predicting depression ($n = 52$)						
Step 1:					.46***	
ADHD	.68	3.82	0.58	.000***		
Step 2:					.54***	.08**
ADHD	.53	2.99	0.61	.000***		
Parent management	.32	0.10	0.04	.005**		
Step 3:					.65***	.09***
ADHD	.49	2.78	0.54	.000***		
Parent management	.22	0.07	0.03	.031*		
Locus of control	.36	1.06	0.27	.000***		
Model 2: Predicting locus of control						
Step 1:					.05	
ADHD	.23	0.44	0.26	.097		
Step 2:					.11	.06
ADHD	.10	0.20	0.29	.495		
Parent management	.28	0.03	0.02	.072		
Model 3: Predicting parent management						
Step 1:					.21***	
ADHD	.46	8.01	2.18	.001***		

Note. ADHD = attention-deficit/hyperactivity disorder.
* $p < .05$. ** $p < .01$. *** $p < .001$.

path to child depressive symptoms, but a cognitive variable, locus of control, emerged as a significant mediator as well.

Findings supported our general expectation of increasing cognitive influence during the childhood years. For the middle group (8–9 years of age), parent management and locus of control independently mediated the relationship between ADHD and depression. Near the end of childhood, parent management and locus of control became more closely associated and the relationship between ADHD and depression became more complex. Accordingly, after the age of 10, locus of control not only independently mediated the relationship between ADHD and depression but also contributed to elevated levels of depression by partially mediating the effects of poor parenting practices.

Prior researchers have described middle childhood as a transition period for moving from diffuse and general to more stable beliefs about the self and world (see Cole, Jacquez, & Maschman, 2001). For instance, around the age of 8, children begin to differentiate domains of their self-concept (Harter, 1989; Marton, Golombek, Stein, & Korenblum, 1988). These changes in cognitions may explain the findings for the middle group in our study. In addition, it is possible that other unmeasured environmental mediators emerge during middle childhood. For instance, other family variables, such as the extent to which parents encourage or discourage independence, and other social influences, such as teacher and peer relationships, may be especially important during middle childhood. Significant changes in time spent with and the

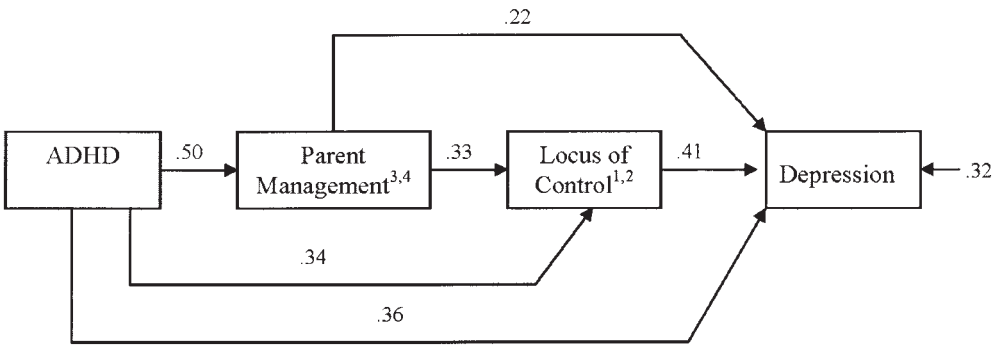

Figure 1. Path model from ADHD to depression for older children. ADHD = attention-deficit/hyperactivity disorder. [1] Locus of control mediates parent–depression (Sobel = 2.17, $p < .030$). [2] Locus of control mediates ADHD–depression (Sobel = 2.23, $p < .026$). [3] Parent management mediates ADHD–depression (Sobel = 3.04, $p < .002$). [4] Parent management mediates ADHD–locus of control (Sobel = 2.17, $p < .030$).

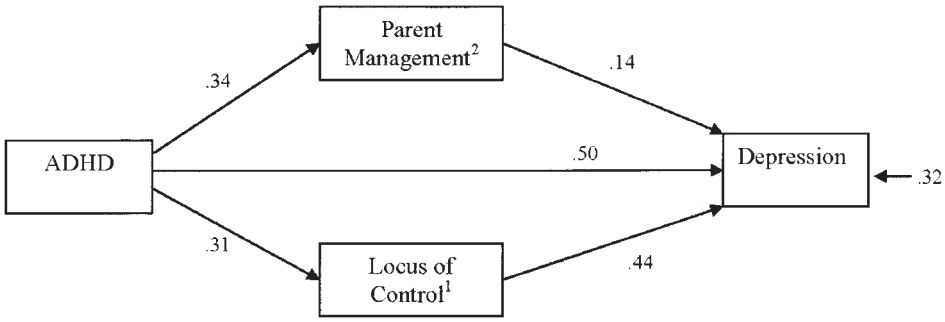

Figure 2. Path model from ADHD to depression for middle children. ADHD = attention-deficit/hyperactivity disorder. [1] Locus of control mediates ADHD–depression (Sobel = 2.99, $p < .003$). [2] Parent management mediates ADHD–depression (Sobel = 1.97, $p < .048$).

influences of the family versus same-aged peers emerge during middle childhood as well (Larson & Richards, 1991; Steinberg & Silverberg, 1986). Perhaps these influences were particularly salient for the middle group.

The findings support Beck's and others' contention (Beck & Young, 1985; Randolph & Dykman, 1998) that the cognitive distortions that influence depression in adulthood emerge during childhood, in part as a consequence of early chaotic family relationships. The timing of this emergence, however, has only recently entered scholarly discourse as an important area of exploration. Developmental theories suggest that children do not have the stable cognitive sets seen in adulthood until middle or late childhood (Cicchetti & Toth, 1995; Dweck & Leggett, 1988; Fincham & Cain, 1986; Harter, 1989; Marton et al., 1988; Ostrander et al., 1995, Ostrander, Weinfurt, & Nay, 1998). Children under 10 years old in the present study may not have developed stable cognitions, and thus the cognitive variable, locus of control, did not mediate the effects of the environment on depressive symptoms for these children. A chaotic, inconsistent, and ineffective family environment may lead directly to depressive symptoms in younger children with ADHD. For older children, however, the influence of parenting behaviors on depressive symptoms was partially mediated by locus of control. It is important to note that even for younger children, locus of control was related to depressive symptoms even though it did not explain the relationship between parent behaviors or ADHD and depression.

On the basis of these findings, effective interventions to treat and prevent depression in children with ADHD may vary depending on the child's age. Interventions to ameliorate or prevent depressive symptoms in younger children may best be targeted at altering dysfunctional parenting styles. On the basis of the parent scale used in the present study, parenting marked by inconsistent expectations and unpredictable consequences were associated with depressive symptoms in children with ADHD. Parent management training to promote consistency, structure, and monitoring—either alone or in combination with cognitive interventions for the child—may help alleviate some of the internalizing symptoms in younger children with ADHD. For older children with ADHD who are also depressed, interventions need to expand beyond a focus on parenting practices and include altering negative cognitions around lack of perceived control.

A strength of the present study includes its large community sample of participants with ADHD. Caron and Rutter (1991) recommended conducting research with community samples for a more accurate depiction of characteristics associated with comorbid conditions. It is unclear, however, whether the findings in the present study are applicable to children found in clinical practice. In addition, the population selected for the present study was predominantly Caucasian, male, and middle class, another limit on the generalizability of the findings.

Additional limitations concern the nature of constructs and measurement strategies selected for this study. We focused on locus of control as our cognitive construct because of previous research linking parenting behaviors to external locus of control; furthermore, most investigations have conceptualized locus of control as a unidimensional construct (Han et al., 2001; Weisz & Stipek, 1982). However, selection of this cognitive construct diverges from recent directions in cognitive research focusing on specific aspects of perceived control (Han et al.; Weisz et al., 2001; Weisz & Stipek). For example, the present study did not consider whether children with ADHD were more inclined to possess an external locus of control as a result of beliefs about their own incompetence or as a result of beliefs that the environmental contingencies were unrelated to their behavior. Future researchers

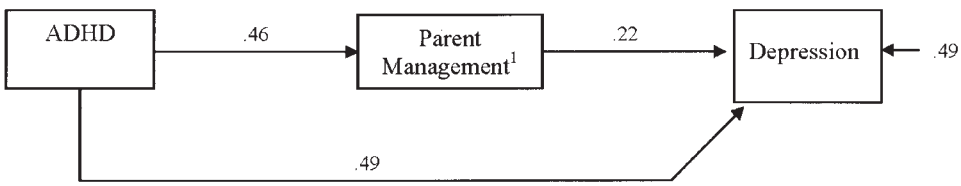

Figure 3. Path model from ADHD to depression for younger children. ADHD = attention-deficit/hyperactivity disorder. [1] Parent management mediates ADHD–depression (Sobel = 2.29, $p < .022$).

should consider the multidimensional nature of control-related beliefs in explaining the relationships between ADHD, parenting practices, and depression.

We chose the parenting measure in the present study because it had many items pertaining to structure and consistency, aspects of parenting that we speculated would be most related to children's perceptions of their own control. However, the measure was limited because it relied solely on the parent's perspective. Parents are able to report on parent–child interactions across a variety of circumstances; because of this, parent-report measures have been viewed as a feasible method of assessing parenting practices (Arnold, O'Leary, Wolff, & Acker, 1993; Locke & Prinz, 2002). Unfortunately, there is no agreed-upon standard for measuring parenting practices, and it is unclear whether parent-reports converge with their actual parenting practices (Locke & Prinz). Therefore, future researchers will need to determine whether the parenting practices assessed in the present study reflect actual parent–child interactions. Subsequent studies should supplement parent reports with child reports and observational data to provide a more robust measure of parenting behavior.

Because of the cross-sectional nature of this study, the present findings can only suggest, not prove, a causal pathway between ADHD, parenting, and depression. We provided a credible theoretical and empirical foundation for our ordering of relationships among the key variables; even so, longitudinal investigations are needed to assess the timing of the proposed relationships and confirm the causal sequence. In addition, further studies are needed to examine the relationships between the variables in the present investigation and other related variables, including life stressors, social competence, and additional components of attributional style and family environment. Further clarification of the developmental pathway to depression for children with ADHD may lead to improved prevention and treatment interventions for these children.

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