The Role of Family Experiences and ADHD in the Early Development of Oppositional Defiant Disorder

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Abstract

Objective—The present study examined the role of family experiences in the early development and maintenance of oppositional defiant disorder (ODD) symptoms in preschool aged children with behavior problems.

Method—Participants were 199 3-year-old children with behavior problems who took part in four annual child and family assessments.

Results—Children with behavior problems who were exposed to overreactive parenting practices, maternal depression, marital conflict, and lower family income tended to have more ODD symptoms 3 years later. Moreover, initial changes in paternal overreactivity, and changes in maternal depression corresponded to initial changes in ODD symptoms. Children who met criteria for ADHD at age 6 were less likely to show improvement in ODD symptoms from age 3 to 6, and were more likely to have been exposed to negative parenting practices, marital conflict, and parental depression during the preschool years. Maternal depression and overreactivity mediated the relation between early hyperactivity and later ODD symptoms.

Conclusions—Results point to the importance of early family functioning in the development of ODD.

Keywords

oppositional defiant disorder; attention-deficit hyperactivity disorder; parenting; preschool aged children

Oppositional defiant disorder (ODD) is characterized by frequent hostility and defiance, and is one of the most common childhood behavior disorders (American Psychiatric
Theoretical models of the etiology of behavior problems emphasize the importance of early childhood experiences. In particular, ODD is thought to develop when family dysfunction coincides with difficult child characteristics early in life (e.g., Campbell, Shaw, & Gilliom, 2000; Lahey & Waldman, 2003). One key difficult child characteristic may be hyperactivity/impulsivity, which has been proposed to contribute to the development of ODD by eliciting negative family functioning (Barkley, 1990). In support of this model, high comorbidity between ODD and attention-deficit/hyperactivity disorder (ADHD) has been well-documented, particularly for ADHD combined and predominantly hyperactive/impulsive types (Burns & Walsh, 2002; Waschbush, 2002).

Numerous studies, including many with longitudinal designs, have demonstrated that children who experience early family dysfunction are more likely to show behavior problems (Burke, Loeber, & Birmaher, 2002). Although these studies provide an important first step in supporting etiological models of ODD, their findings may be largely driven by child effects or third variables. Early family functioning may be associated with later behavior problems because early difficult child characteristics both elicit early family dysfunction and lead directly to later behavioral difficulties. Similarly, third variables such as shared genetics may underlie both family dysfunction and behavior problems. A stronger test of etiological models of ODD involves studying ODD as it emerges early in development among children who demonstrate the difficult child characteristics that, in conjunction with family dysfunction, are hypothesized to lead to the development of ODD.

A Need for Longitudinal Research on Families of Young Preschool Children With Behavior Problems

There are multiple pathways in the development of ODD, one of the most problematic of which involves the emergence of behavior problems early in life, with approximately 5 to 7% of the population showing stable antisocial behavior beginning during the preschool years (Campbell et al., 2000). At the same time, an estimated half of preschoolers with behavior problems outgrow their difficulties (Campbell, Breaux, Ewing, & Szumowski, 1986), suggesting that the preschool years may be a sensitive period during which environmental influences may cause children with difficult child characteristics to follow diverging pathways. Only a handful of studies have examined whether family variables play a key role in helping preschool children with behavior problems outgrow their difficulties (Campbell, Ewing, Breaux, & Szumowski, 1986; Campbell, Pierce, Moore, & Marakovitz, 1996; Heller, Baker, Henker, & Hinshaw, 1996; Lavigne et al., 1998; Olson & Hoza, 1993; Palfrey, Levine, Walker, & Sullivan, 1985), with even fewer focusing on children showing behavior problems before age 4 (Campbell, Ewing et al., 1986; Campbell et al., 1996; Lavigne et al., 1998). Although there is considerable variability in the age of onset of ODD (Todd, Huang, & Henderson, 2008), impairing symptoms often begin during the early preschool years (e.g., Keenan & Wakschlag, 2000). Stability of behavior problems tends to be higher during the later preschool years than during the early preschool years (Campbell, Ewing et al., 1986; Harvey, Youngwirth, Thakar, & Errazuriz, 2009; Lahey et al., 2004), suggesting that by age 4 or 5, many children may be firmly on their pathways, and the family processes that led them there may have already largely occurred. Thus, large scale studies with comprehensive family assessments are needed to follow children with behavior problems beginning during the early preschool years when ODD often first emerges.

A Need to Examine Change Over Multiple Time Points

Historically, longitudinal research on family functioning and behavior problems often focused on two time points. The advent of growth curve modeling brought recognition of the value of assessing change using multiple time points. Growth curve modeling has been used in studies of community samples to examine how family functioning during the preschool or
early elementary years is associated with subsequent changes in externalizing problems (e.g., NICHD Early Child Care Research Network, 2004). Parallel process latent growth curve modeling (Cheong, MacKinnon, & Khoo, 2003) can also be used to address the less-commonly studied, but critical question of whether trajectories of family functioning predict trajectories of child functioning. Applying these approaches in an at-risk preschool sample may advance our understanding of the role that family functioning plays in the development and maintenance of early behavior problems.

A Need to Distinguish Among Different Types of Behavior Problems

A growing body of research points to the importance of distinguishing between ADHD and ODD (Waschbusch, 2002), and there is evidence that even as early as age 3, it may be important to differentiate between children with hyperactivity/impulsivity and/or oppositional defiance/aggression (Harvey, Friedman-Weieneth, Goldstein, & Sherman, 2007). Examining the interplay between ADHD and ODD symptoms early in development is key to understanding the role that ADHD may play in the development of ODD. The few longitudinal studies that have analyzed ADHD and ODD symptomatology separately provide support for the importance of disentangling these comorbid disorders. For example, symptoms of ADHD have been found to increase the likelihood that conduct problems will persist beyond the preschool years (Speltz, McClellan, DeKlyen, & Jones, 1999). Moreover, family functioning (maternal parenting and depression) predicted later conduct problems among young children (ages 4 to 7) with ADHD (Chronis et al., 2007). Research is needed to build on these studies by directly examining changes in both ODD symptoms and family functioning (including fathers’ functioning) and their relation to ADHD, beginning during the early preschool years.

A Need for Research on Fathers

Fathers play an important role in child development (Loeber, 1990). Few longitudinal studies of preschool children with behavior problems have examined how fathers’ functioning is linked with children’s later behavior problems, reflecting a general pattern in developmental psychopathology research in which fathers continue to be underrepresented (Phares, Fields, Kamboukos, & Lopez, 2005). Fully understanding the role of family dysfunction in the development of ODD requires more research on fathers.

The Present Study

The present study sought to address these gaps in the literature by examining child and family predictors of later ODD symptoms among 3-year-old children with behavior problems, focusing on two important types of early family experiences: parenting practices and family adversity. The link between parenting practices and disruptive behavior problems has been well documented (e.g., Rothbaum & Weisz, 1994). Research also points to a number of other family variables that may be implicated in the development of ODD including marital conflict (e.g., Shaw, Owens, Vondra, & Keenan, 1996), socioeconomic status (Campbell, Breaux et al., 1986), single parenthood (Palfrey et al., 1985), and maternal psychopathology (Shaw et al., 1996).

The present study sought to test key components of theoretical models of the etiology of ODD (e.g., Barkley, 1990; Lahey & Waldman, 2003), which posit that ODD symptoms develop when family dysfunction is paired with difficult early child characteristics, including symptoms of ADHD. In particular, the following hypotheses were tested:

**Question 1: Do early family experiences and changes in family experiences predict improvement in ODD symptoms among preschool aged children with behavior problems?**—It was hypothesized that age 3 parenting practices and family
adversity (including maternal and paternal psychopathology, socioeconomic status, single parenthood, and marital conflict) would predict trajectories of ODD symptoms among preschool aged children with behavior problems, such that more family dysfunction at age 3 would be associated with more ODD symptoms at age 6 and with less improvement in ODD symptoms over time. It was also hypothesized that changes in parenting practices and family adversity from age 3 to 6 would predict trajectories of ODD symptoms among preschool aged children with behavior problems, such that deterioration in family functioning over time would be associated with more ODD symptoms at age 6 and with less improvement in ODD symptoms over time. Because children with ADHD often need assistance from their environments to control their behavior (Barkley, 1990), they may be particularly sensitive to disruptions in their environment. Therefore, ADHD status was predicted to moderate these relations, such that relations between family functioning and ODD symptoms would be stronger among children who met criteria for ADHD at age 6.

Question 2: Do children with ADHD experience more early family dysfunction?—A key part of the process by which ADHD may lead to the development of ODD involves the tendency for children with ADHD to elicit negative family interactions. It was predicted that families of children with ADHD would show more negative parenting practices and more family adversity at age 3 and would show worse trajectories of family functioning over the preschool years due to cumulative effects of ADHD on the family over time.

Question 3: Are children with ADHD at greater risk for developing and/or maintaining ODD symptoms during the preschool years?—If ADHD plays an important role in the development of ODD, one should see evidence of ODD emerging among children with ADHD during the preschool years. It was predicted that children with ADHD would show less improvement in ODD symptoms from age 3 to 6 and would show more symptoms of ODD at age 6 compared to children without ADHD.

Question 4: Do family experiences mediate the relation between early hyperactivity and subsequent ODD symptoms?—Existing models posit that early symptoms of ADHD may elicit negative family functioning which in turn leads to the development of ODD symptoms. Therefore it was predicted that negative parenting practices and family adversity would mediate the relation between early hyperactivity and subsequent ODD symptoms.

**Method**

**Participants**

Participants were 199 children (107 boys, 92 girls) and their 199 mother figures and 158 father figures who took part in a 4-year longitudinal study of preschool aged children with behavior problems. Children were 3 years old at screening and 36 to 50 months (M = 44 months, SD = 3) at the first home visit (T1). Data were collected from families at 1-year (T2; n = 184), 2-year (T3; n = 163), and 3-year (T4, n = 168) follow-up visits. The sample included European American (49.7%), Latino American (21.6%; mostly Puerto Rican), African American (12.6%), and multiethnic (16.1%) children. The median family income at T1 was $47,110. Most mothers (84.4%) and fathers (88.8%) had high school diplomas, and 33.2% of mothers and 29.2% of fathers had bachelor’s degrees. All mothers lived with their children; fathers who did not live with their children full-time were invited to participate if they spent time with their children on a regular basis each week. All mothers participated at T1; 16 of the 158 fathers did not participate at T1, but participated at one or more later time points.
Procedure

Children with significant externalizing problems were recruited from 3-year-old children \( (n = 1752) \) whose parents completed a screening packet which they received through mail (via state birth records), pediatrician offices, child care centers, and community centers. A smaller group of non-problem children were also recruited but are not the focus of this study. Inclusion criteria were (a) no evidence based on parent report of mental retardation, deafness, blindness, language delay, cerebral palsy, epilepsy, autism, or psychosis; (b) parent reported concern about the child’s activity level, defiance, aggression, or impulse control; and (c) Behavioral Assessment System for Children – Parent Report Scale (BASC-PRS; Reynolds & Kamphaus, 1992) Hyperactivity and/or Aggression subscale \( T \) scores at or above 65. Parents were told that the goal of the study was to understand factors that help young children with behavior problems outgrow their difficulties and they were paid for participating. Fifty-nine percent of behavior problem children whom we sought to recruit participated. Written informed consent was obtained from all parents who participated. The study was conducted in compliance with the authors’ Internal Review Board.

Measures

**Single parenthood**—Families were classified at T1 as married/living together (coded 1) or as single (coded 0) based on parent report.

**Family income**—Family income at T1 was based on the total income of adults living with the child. Missing data were estimated by predicting income from years of education.

**Parent depression**—Parent depression was measured at each time point using the Millon Clinical Multiaxial Inventory – III (MCMI-III; Millon, Davis, & Millon, 1997), a 175-item questionnaire of symptoms of DSM-IV disorders. The internal consistency for the MCMI-III scales in a clinical population ranged from .66 to .90; test-retest reliabilities ranged from .84 to .96 (Millon et al., 1997). The full MCMI-III was administered at T1, and only subscales that assess Major Depression, Dysthymia, and Depressive Personality were administered at T2, T3, and T4. Since one of the objectives of the current study was to analyze changes in depression over time, 8 of the 33 items were not included because they assess symptoms over a long time-frame. Prototypical items for the Major Depression scale (Millon et al., 1997) were double-weighted. Combined raw scores of these three scales were used; higher scores indicated greater depression.

**Marital conflict**—At T1, married or cohabiting couples completed the Conflicts and Problems-Solving Scales – Violence Form (CPS-V; Kerig, 1996), which includes 69 items describing positive and negative conflict tactics. Members of the couple independently rated the degree to which each tactic was used in the relationship by themselves and their partners. The 138 items (69 self and 69 partner) were averaged to create an overall conflict strategy score. High scores indicated greater use of negative conflict strategies. Cronbach’s alpha was .95 for mothers and for fathers. Mothers’ and fathers’ conflict scores were significantly correlated, \( r (125) = .52, p < .001 \), and were standardized and averaged. When only one member of the couple completed the measure, that individual’s standardized score was used to assess conflict strategies.

**Parenting practices**—Parenting was assessed separately for mothers and fathers using self-report and audiotaped observation. Self-report of parenting was assessed at each time point using the Parenting Scale (Arnold, O’Leary, Wolff, & Acker, 1993), which is a 30-item scale that yields scores for laxness and overreactivity, with high scores indicating dysfunctional parenting. The Parenting Scale has demonstrated good internal consistency (\( \alpha = .83 \) for laxness and \( .82 \) for overreactivity), good test-retest reliability (.83 for laxness and...
82 for overreactivity), and has been found to correlate with observations of parenting and child behavior (Arnold et al., 1993).

At T1, mothers and fathers were asked to record their interactions with their children at home using two 60 min tapes per parent. Parents were instructed to select times that tended to be challenging and to behave as they normally would. The first 15 min of each side of one tape was coded for each parent because a preliminary review of the tapes suggested that this was sufficient to capture a wide variety of behavior. Trained research assistants who were unaware of the child’s behavior status rated warmth and negative affect separately for mothers and fathers, and two raters overlapped for one-fourth of participants. Warmth referred to being positively attentive to the child; using praise, encouragement, and terms of endearment; conveying affection; being supportive and available; being cheerful in mood and tone of voice; and/or conveying interest, joy, enthusiasm, and warmth in interactions with the child. Global ratings of parent warmth (intraclass correlation [ICC] = .53) were made every 5 min and ranged from 1 (not warm) to 7 (extremely warm). Negative affect ratings indicated irritation, annoyance, frustration, or anger. Each statement that was judged to exhibit negative affect was rated on a scale from 1 (slight) to 6 (strong), and these ratings were summed across the 30 min of interaction (ICC = .60). A square root transformation was conducted on negative affect to reduce skewness. This method of recording parenting has been shown to be sensitive to detecting changes in parent-child interactions following parent training (Danforth, Harvey, Ulaszek, & McKee, 2006).

To further evaluate the validity of these parenting measures, the present sample of children with behavior problems was compared to the 59 nonproblem children who took part in the larger study. Parents of children in the behavior problem sample showed significantly more negative parenting practices than parents of nonproblem children on six of the eight parenting variables (all ps < .05). The difference for mothers’ self-reported laxness and fathers’ observed negative affect approached significance (p = .09 and p = .05, respectively). The average effect size for comparisons between the two groups was d = .50.

**ODD symptoms**—At T1, T2, and T3, the ADHD and ODD sections of the NIMH-Diagnostic Interview Schedule for Children-IV (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) were administered to parents, with minor modification to school-related questions. The full version of the DISC-IV was administered at T4. When both parents took part in the interview, primary caregivers’ responses were used in the rare cases of open disagreement. The ODD section of the DISC-IV assesses whether the child frequently displays each of 8 DSM-IV symptoms; DSM-IV criteria for ODD require the presence of 4 or more symptoms (American Psychiatric Association, 2000). ODD symptom counts (which could range from 0 to 8) were used at each time point, and showed no substantial skewness (skewness coefficients ranged from −.10 to .54).

**T4 diagnoses**—Clinicians (trained psychology graduate students) assigned diagnoses of ADHD and ODD based on an examination of DISC symptom counts, elevations on teacher and parent BASC subscales (T scores > 65), and evidence of impairment on the DISC and a psychosocial interview. Convergent evidence of developmentally deviant symptoms (elevations on most measures) was needed to make diagnoses. ADHD diagnoses were given if clinically significant symptoms were evident at home or at school. Two clinicians reviewed T4 materials and made independent diagnoses. Discrepancies were discussed and a consensus diagnosis was reached. Kappa was .78 for ADHD and .75 for ODD. Of the 168 behavior problem children who completed T4, 36 (20 boys, 16 girls) met criteria for ADHD only, 22 (13 boys, 9 girls) for ODD only, and 39 (26 boys, 13 girls) for ADHD and ODD. Of the 75 children who met criteria for ADHD, 6 met criteria for ADHD predominantly
inattentive type, 13 for ADHD predominantly hyperactive/impulsive type, and 56 for ADHD combined type.

**T1 hyperactivity**—Children’s hyperactivity at T1 was measured using mothers’ BASC-PRS Hyperactivity T-scores. The BASC-PRS is a widely used scale of child psychopathology, which has demonstrated good reliability and validity (Reynolds & Kamphaus, 1992).

### Results

All models were run using MPLUS (Muthén & Muthén, 1998–2010) and were conducted separately for mothers and fathers because not all families were two-parent families. One-tailed tests were used for a priori predictions.

### Descriptive Statistics

Intercorrelations and descriptive statistics for predictor variables are presented in Table 1. Parenting and family experience variables were generally moderately correlated for mothers, but were more modestly correlated for fathers. Family adversity variables were consistently related to one another in expected directions. In fact, the strongest correlations for fathers involved relations between paternal depression and other measures of family adversity.

ODD symptoms showed a pattern of growing decline over the four time points (T1 $M = 4.535$, $SD = 2.007$; T2 $M = 4.215$, $SD = 2.189$; T3 $M = 3.750$, $SD = 2.280$; T4 $M = 2.570$, $SD = 2.239$), with ODD symptoms decreasing nearly a full standard deviation from T1 to T4.

### Unconditional Growth Models

**ODD unconditional growth models**—Two components of ODD trajectories were of interest: (a) rate of change in ODD symptoms and (b) level of ODD symptoms at T4. Unconditional growth models (Figure 1a) were used to estimate ODD trajectories and consisted of three latent growth factors (intercept, linear, and quadratic). These models revealed significant curvature in ODD trajectories (see below), which complicated the measurement of rate of change in ODD symptoms, because the instantaneous linear rate of change in ODD symptoms varied across time. Linear rate of change at T1 was selected as the rate of change outcome, because of conceptual interest in predictors of early improvement in ODD symptoms. Because the two outcomes of interest (initial [T1] linear rate of change in ODD and T4 ODD symptom level) required estimating ODD trajectories centered at both T1 and T4, two sets of growth models were constructed: (a) *T1 Models*, in which Time was centered at T1 (child age in years [months/12] minus T1 age) to estimate the initial linear rate of change in ODD per year; and (b) *T4 Models*, in which Time was centered at T4 (child age minus T4 age) to estimate T4 ODD symptom levels.

Figure 2 presents the average growth trajectory of ODD symptoms. The average of the quadratic growth factor (representing acceleration in the rate of change/curvature in trajectories) was significantly different from zero based on both the T1 ($\beta = -0.214$, $SE = 0.061$, $p < .001$) and T4 ($\beta = -0.234$, $SE = 0.063$, $p < .001$) unconditional growth models. The average initial linear rate of change in ODD was not significantly different from zero ($\beta = -0.007$, $SE = 0.194$, $p = .97$). Thus, average ODD symptoms did not show a significant initial linear change, but showed a gradually accelerating decrease over the next 3 years. The latent growth factors that were conceptually of greatest interest as ODD outcome variables (T4 model intercept and T1 model linear, which are identified in Figure 2) both demonstrated significant variability across individuals ($\sigma^2 = 2.622$, $SE = 1.315$, $p = .046$, and $\sigma^2 = 1.932$, $SE = 0.978$, $p = .048$, respectively). Thus, although the average initial linear rate...
of change in ODD was not significantly different from zero, there was significant variability in initial linear rates of change. There was not significant variability in the quadratic growth factor for either T1 or T4 models (\( \sigma^2 = 0.130, SE = 0.078, p = .093 \), and \( \sigma^2 = 0.149, SE = 0.114, p = .193 \), respectively).

**Family experience unconditional growth models**—Unconditional growth models were also estimated, separately for mothers and fathers, for overactivity, laxness, and depression (Time centered at T1), to determine whether there was significant curvature in these trajectories. The quadratic latent growth factors for maternal laxness, \( \beta = 0.048, SE = 0.020, p = .017 \), and paternal overactivity, \( \beta = -0.077, SE = 0.026, p = .003 \), were significant, so quadratic latent growth factors were included for subsequent models involving these two family variables. In the presence of a significant quadratic factor, the linear latent growth factor for maternal laxness and paternal overactivity represented initial linear rate of change per year (because Time was centered at T1). Exploratory analyses indicated that these family experience quadratic growth factors were not significant predictors of ODD trajectories, so ODD growth factors were not regressed on these family quadratic growth factors in the final models.

The linear latent growth factors were significant for maternal laxness, \( \beta = -0.226 (\sigma^2 = .070), SE = 0.067, p = .001 \), and paternal overactivity, \( \beta = 0.239 (\sigma^2 = .425), SE = 0.084, p = 0.004 \), suggesting that, on average, mothers showed an initial decrease in laxness and fathers showed an initial increase in overactivity at T1. Linear latent growth factors were not significant for maternal depression, \( \beta = -0.238 (\sigma^2 = 1.304), SE = 0.132, p = .071 \), paternal depression, \( \beta = 0.004 (\sigma^2 = .718), SE = 0.131, p = .977 \), maternal overactivity, \( \beta = -0.024 (\sigma^2 = 0.011), SE = 0.020, p = .229 \), or paternal laxness, \( \beta = 0.001 (\sigma^2 = 0.115), SE = 0.087, p = .987 \), suggesting that the average levels of these variables remained unchanged over time.

**Question 1: Do early family experiences and changes in family experiences predict improvement in ODD symptoms among preschool aged children with behavior problems?**

**Model construction**—Figure 1b depicts models that were estimated for predictor variables that were measured only at T1. For family variables that were available at multiple time points, parallel process latent growth curve models (Cheong et al., 2003) were estimated (Figure 1c). Note that the ODD quadratic growth factor was regressed on the predictor variables and if this path was not significant, it was omitted from the final model. Table 2 presents results of these models. In the interest of space, only coefficients of the predictor variables and if this path was not significant, it was omitted from the final model.

**Predictors of initial linear rate of change in ODD**—Lower levels of T1 maternal overactivity, \( \gamma = 0.340, SE = 0.154, p = .013 \), and paternal overactivity, \( \gamma = 0.267, SE = 0.139, p = .027 \), were associated with initial linear decreases in ODD symptoms. Moreover, initial linear decreases in fathers’ overactivity were associated with initial linear decreases in ODD symptoms, \( \gamma = 0.568, SE = 0.269, p = .017 \). Lower maternal depression at T1, \( \gamma = 0.035, SE = 0.010, p < .001 \), and linear decreases in maternal depression, \( \gamma = 0.240, SE = 0.097, p = .007 \), were associated with initial linear decreases in ODD symptoms.

**Predictors of T4 ODD levels**—Lower levels of T1 maternal overactivity, \( \gamma = 1.035, SE = 0.423, p = .007 \), and paternal overactivity, \( \gamma = 0.774, SE = 0.359, p = .015 \), as well as greater initial decreases in paternal overactivity, \( \gamma = 1.594, SE = 0.744, p = .016 \), were associated with fewer T4 ODD symptoms. Lower T1 maternal depression, \( \gamma = 0.108, SE = 0.031, p < .001 \), and greater linear decreases in maternal depression, \( \gamma = 0.715, SE = 0.299, p = .009 \), were associated with fewer T4 ODD symptoms. Higher T1 income, \( \gamma = -0.008, SE = .024 \).
Predictors of curvature in ODD trajectories—Paternal warmth was the only family experience variable that significantly predicted the ODD quadratic growth factor (T1 Model, $\gamma = 0.187$, $SE = 0.090$, $p = 0.038$; T4 Model, $\gamma = 0.285$, $SE = 0.124$, $p = 0.022$), indicating that greater paternal warmth was associated with less acceleration in the decrease in ODD symptoms (i.e., flatter trajectories).

ADHD status as a moderator of the relation between family experiences and ODD symptoms—Because the relation between family functioning and ODD symptoms may vary as a function of ADHD status, we examined whether ADHD status (1 = ADHD; 0 = not ADHD) interacted with family variables to predict ODD trajectories. Children with ADHD inattentive type were coded as not having ADHD, because hyperactivity, rather than inattention, is thought to play a role in the development of ODD (Burns & Walsh, 2002). T4 ADHD status and an ADHD × family predictor variable product term were entered as predictors of ODD growth factors. These variables were dropped if the interaction term was not significant.

ADHD status interacted with T1 negative affect in predicting initial linear rate of change in ODD for mothers, $\gamma = -0.131$, $SE = 0.043$, $p = 0.002$, and fathers, $\gamma = -0.168$, $SE = 0.090$, $p = 0.03$, and in predicting T4 ODD levels for mothers only, $\gamma = -0.391$, $SE = 0.127$, $p = 0.001$, indicating that the relations between negative affect and ODD were significantly less positive for children with ADHD than for children without ADHD. Negative affect coefficients were used to further interpret these interactions. Coefficients in Table 2 represent relations for children without ADHD (ADHD status = 0); models were rerun with ADHD status coded 0 for ADHD to test the relations for children with ADHD. Among children without ADHD, lower maternal T1 negative affect was associated with fewer T4 ODD symptoms, $\gamma = 0.299$, $SE = 0.099$, $p = 0.001$, and with initial decreases in ODD symptoms, $\gamma = 0.086$, $SE = 0.034$, $p = 0.006$. In contrast, among children with ADHD, maternal negative affect was not significantly associated with T4 ODD symptoms, $\gamma = -0.091$, $SE = 0.080$, $p = 0.126$, or with initial linear rate of change in ODD, $\gamma = -0.045$, $SE = 0.028$, $p = 0.051$. For fathers, although the significant interaction indicated that the relation between paternal negative affect and initial linear rate of change in ODD was significantly less positive for children with ADHD than for children without ADHD, the relations were not significantly different from zero for either children with ADHD, $\gamma = -0.104$, $SE = 0.071$, $p = 0.071$) or for children without ADHD, $\gamma = 0.063$, $SE = 0.055$, $p = 0.124$. In sum, parental negative affect appeared to be more detrimental for children without ADHD than for children with ADHD, although the relation between parental negative affect and poor ODD outcome in children without ADHD reached significance only for mothers.

Question 2: Do children with ADHD experience more early family dysfunction?

Both T4 ADHD status (combined and hyperactive/impulsive type) and T1 maternal BASC-PRS Hyperactivity scores were examined as predictors of each family experience variable, because T4 ADHD status was thought to provide the most accurate measure of underlying ADHD, whereas T1 hyperactivity fits better temporally with conceptual models that point to early hyperactivity as a key ingredient in the development of ODD. For family experience variables that were measured only at T1, each family variable was regressed first on T1 hyperactivity and then on T4 ADHD status, using linear regression. For multi-time point family variables, T1 hyperactivity and T4 ADHD status were entered separately as predictors of family variable intercept and linear growth factors.
T1 hyperactivity and T4 ADHD were associated with lower maternal warmth, more maternal negative affect, higher paternal laxness, and higher maternal and paternal depression at T1 (Table 3). T1 hyperactivity was also associated with higher maternal overreactivity, maternal laxness, and paternal negative affect at T1. T4 ADHD status was associated with more T1 marital conflict and with linear increases in paternal depression.

**Question 3: Are children with ADHD at greater risk for developing and/or maintaining ODD symptoms during the preschool years?**

T1 hyperactivity and T4 ADHD status were entered (in separate models) as predictors in the model shown in Figure 1b (in place of the Family Predictor). Children with ADHD at T4 had more T4 ODD symptoms, $\gamma = 1.379, SE = 0.335, p < .001$, and were less likely to show initial linear decreases in ODD, $\gamma = 0.449, SE = 0.108, p < .001$. T1 hyperactivity was also associated with more T4 ODD symptoms, $\gamma = 0.032, SE = 0.013, p = .005$, but did not significantly predict initial linear rate of change in ODD, $\gamma = 0.005, SE = 0.006, p = .224$.

**Question 4: Do family experiences mediate the relation between early hyperactivity and subsequent ODD symptoms?**

Latent growth factors can also be treated as variables involved in meditational relations (Cheong et al., 2003; Selig & Preacher, 2009). Because marital conflict, maternal overreactivity, maternal depression, and paternal overreactivity predicted ODD symptoms, T1 hyperactivity was added to the models shown in Figure 1b and 1c, to examine whether these family variables would mediate the observed relation between T1 hyperactivity and T4 ODD symptoms. Income and negative affect were not examined in mediation analyses because income was not thought to be influenced by children’s hyperactivity, and negative affect was a predictor of ODD only for children without ADHD. In particular, the ODD intercept and linear growth factors in the T4 Models were regressed on T1 hyperactivity and on the family experience variables, and the family experience variables were regressed on T1 hyperactivity. There was evidence that T1 maternal overreactivity partially mediated and that T1 maternal depression mediated the relation between T1 hyperactivity and T4 ODD symptoms (Table 4). There was no evidence that paternal overreactivity, marital conflict, or changes in maternal depression mediated the relation between T1 hyperactivity and T4 ODD symptoms.

**Discussion**

The present study examined child and family predictors of the early development of ODD symptoms among young preschool aged children with behavior problems. The results of this study provide some support for etiological models of ODD that suggest that early family experiences play a key role in the development and maintenance of early behavior problems (Lahey & Waldman, 2003). In particular, 3-year-old children with behavior problems who were exposed to overreactive parenting practices, maternal depression, marital conflict, and lower family income, were less likely to show initial improvement in ODD symptoms and/or tended to have more ODD symptoms at age 6. However, single parenthood; paternal depression; and parental negative affect, laxness, and lack of warmth were not predictive of more ODD symptoms. In addition, there was some support for the notion that ADHD may place children at risk for developing ODD symptoms by disrupting family functioning. Children with ADHD showed less improvement in ODD symptoms and poorer family functioning during the preschool years than children without ADHD, although only maternal depression and maternal overreactivity mediated the relation between early hyperactivity and later ODD.
symptoms. The present study did not find evidence that the effects of family adversity were greater among children with ADHD.

Perhaps the strongest support for the importance of early family experiences in this sample came from evidence that linear changes in maternal depression and initial linear changes in paternal overreactivity were linked to initial linear changes in ODD symptoms at age 3, although these findings did not extend to other family experience variables. Documenting that maternal depression and paternal overreactivity covary with ODD symptoms within families over time reduces the possibility that time invariant third variables (e.g., genetics or socioeconomic status) account for the link between these aspects of family functioning and child behavior. However, it remains unclear whether changes in family functioning over time are a cause or an effect of children’s changing symptomatology. In fact, existing theory and research suggest that the relation between parenting and child behavior is bidirectional (Shaw & Bell, 1993).

The present study extends previous research by examining the role that ADHD may play in the development of ODD. ADHD symptoms, measured at both age 3 and age 6, were associated with more ODD symptoms at age 6. Children who met criteria for ADHD at age 6 also showed more problematic initial linear changes in ODD symptoms; however, hyperactivity at age 3 was not significantly related to initial changes in ODD symptoms. Measuring hyperactivity at age 3 may include transient, developmental hyperactivity, which may not have the same effect on the development of ODD as chronic hyperactivity underlying ADHD. Nonetheless, the finding that children who later met criteria for ADHD were less likely to show improvement in ODD symptoms as young preschoolers suggests that the preschool years may be a critical time for the development of comorbidity between ADHD and ODD.

Our findings corroborate and extend longitudinal research with older preschool aged children (Chronis et al., 2007) and suggest that family functioning plays at least some role in the development of comorbid ADHD/ODD. Children with ADHD and/or early hyperactivity experienced disruptions in many domains of family functioning early in life, although only two of these domains (early maternal overreactivity and depression) mediated the relation between early hyperactivity and later ODD symptoms. It is important to consider other possible causal mechanisms that may explain these findings. For example, given the strong genetic component underlying ADHD (see Barkley, 1990), disruptions in family functioning among children with ADHD may be a marker for genetic risk for ADHD, and these disruptions in family functioning may in turn lead to the development of ODD. It is also possible that genetics may directly account for the development of ODD among children with ADHD (Wood, Rijsdijk, Asherson, & Kuntsi, 2009), and family dysfunction may be caused by a combination of shared genetic and child effects. A third possible causal explanation is that family dysfunction leads to the development of both ADHD and ODD, although this is not consistent with previous research documenting a strong genetic/biological component of ADHD. Further research is needed to explore these possibilities.

The present findings generally corroborate previous research suggesting that fathers play an important role in the development of behavior problems (Loeber, 1990), though paternal functioning was less consistently linked to ODD symptoms than was maternal functioning. In particular, fathers’ overreactivity predicted initial linear changes in ODD symptoms and ODD symptom outcome, which is consistent with previous research linking fathers’ functioning to ODD symptoms among children with ADHD (e.g., Johnston, 1996). This study also suggests that fathers of children with ADHD may experience disruptions in their
own functioning when children are very young, well before children have been diagnosed with ADHD. However, fathers’ functioning was not found to mediate the relation between early hyperactivity and later ODD symptoms. Thus, although fathers’ functioning appears to be tied to both ADHD and ODD, it does not appear to explain the link between the two. Interestingly, although paternal warmth was not associated with initial linear change or later levels of ODD symptoms, it was associated with less curvature in ODD trajectories. Fathers who showed less warmth had children who initially showed marginally smaller linear decreases in ODD symptoms, but showed greater acceleration in improvement than children of warmer fathers. Perhaps lack of paternal warmth leads to positive changes in other family members who may attempt to compensate for low paternal warmth, which in turn leads to later improvement in children. Alternatively, fathers of preschoolers presenting with behavioral challenges may shift to a more structuring (and seemingly less warm) role in interacting with their children, which results in a more rapid decrease in ODD symptoms. More research is needed to understand this finding and to examine the interplay among family variables early in children’s development.

Clinical Implications

This study provides moderate support for existing models of the development of ODD, and provides clinically relevant information regarding the family variables that may be most helpful to target in prevention programs for children with early behavior problems. In particular, early overreactive parenting and maternal depression both showed clinically meaningful relations with later ODD symptoms. Parents who were one standard deviation above the mean on initial levels of or changes in depression or overreactivity would be predicted to have children with 1.25 to 2.08 more ODD symptoms (.56 to .93 of a standard deviation) at age 6 than parents who were one standard deviation below the mean on these parent factors. These findings support parent training interventions that provide parents with positive alternatives to harsh parenting (Webster-Stratton & Taylor, 2001), but also support previous efforts to target family functioning more broadly (Parsons & Alexander, 1973; Patterson, 2002). In particular, targeting maternal depression in addition to parenting skills may better prevent the development and maintenance of ODD in young children. This study also provides additional support for the importance of involving fathers in parent training (Lundahl, Tollefson, Risser, & Lovejoy, 2008). Finally, this study suggests that early family intervention with young children at risk for developing ADHD may hold some promise for preventing the development of comorbid ODD among children with ADHD. Although there has been growing interest in the use of stimulant medication for preschoolers with ADHD (Zito, Safer, dosReis, Gardner, Boles, & Lynch, 2000), relatively little attention has been given to developing psychosocial interventions for preschoolers with ADHD.

Limitations

The results of the present study should be interpreted in the context of several limitations. First, method variance may account for some of the observed relations in this study. In cases in which parents reported on both their own functioning and their children’s behavior, shared method variance may have inflated the observed associations. Similarly, associations between parental depression and child behavior may be accounted for by reporting bias among depressed parents. Second, interrater reliability for audiotaped parenting data was somewhat low, which may have made it difficult to detect effects. Third, although the sample was ethnically diverse, there were not sufficient numbers of participants from each ethnic group to examine the moderating effect of ethnicity; thus, it is not clear whether results are generalizable to all ethnic groups represented in this sample. Fourth, the recruitment rate for this study was relatively low (59%), limiting the generalizability of these findings. Finally, results may have been different if another method of recruitment had

J Consult Clin Psychol. Author manuscript; available in PMC 2012 December 1.
been used, such as recruiting in a clinical setting, using teacher reports to identify children, or using epidemiological sampling.

Acknowledgments

This research was supported by National Institute of Mental Health Grant R01MH60132.

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a. Unconditional Latent Growth Curve Model

b. Single Predictor Latent Growth Curve Model
c. Parallel Process Latent Growth Curve Model

Unconditional, single predictor, and parallel process latent growth curve models. T1 = Time 1. T4 = Time 4. Separate models were estimated for mothers and fathers. Separate models were estimated for overreactivity (Over), laxness (Lax), and depression. Individually varying time points were used. This model was estimated with time (in years) centered at T1 (T1 Model) and then again with time centered at T4 (T4 Model). For T1 Models, ODD Linear represented the instantaneous linear rate of change at T1 and was the outcome of interest. For T4 Models, ODD Intercept represented T4 ODD symptom level and was the outcome of interest. For T1 Models, parameters b1 and b2 were of interest and for T4 Models, parameters a1 and a2 were of interest and are presented in Tables. Paths c1 and c2 were omitted from the model if they were not significant.

Figure 1.
Figure 2.
Average ODD trajectory from Time 1 (Time = 0) to Time 4 (Time = 3).
Table 1

Intercorrelations, Means, and Standard Deviations of Predictor Variables for Mothers (Below Diagonal) and Fathers (Above Diagonal)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mothers M (SD)</th>
<th>Father M (SD)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. T1 overreactivity&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.803 (0.741)</td>
<td>2.593 (0.809)</td>
<td>.084</td>
<td>-.137</td>
<td>.121</td>
<td>.011</td>
<td>.120</td>
<td>.089</td>
<td>.229**</td>
<td></td>
</tr>
<tr>
<td>2. T1 laxness&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.990 (1.000)</td>
<td>2.840 (0.892)</td>
<td>.131</td>
<td>-.102</td>
<td>.000</td>
<td>.217*</td>
<td>.037</td>
<td>-.271**</td>
<td>.009</td>
<td></td>
</tr>
<tr>
<td>3. T1 warmth</td>
<td>4.397 (1.003)</td>
<td>4.613 (0.810)</td>
<td>-.111</td>
<td>-.363***</td>
<td>-.286**</td>
<td>.005</td>
<td>.025</td>
<td>.135</td>
<td>.042</td>
<td></td>
</tr>
<tr>
<td>4. T1 negative affect</td>
<td>2.938 (2.575)</td>
<td>1.810 (1.561)</td>
<td>.264***</td>
<td>.280***</td>
<td>-.526***</td>
<td>.086</td>
<td>.259**</td>
<td>-.181</td>
<td>.017</td>
<td></td>
</tr>
<tr>
<td>5. T1 depression&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.081 (6.506)</td>
<td>3.707 (5.232)</td>
<td>.202**</td>
<td>.330***</td>
<td>-.298***</td>
<td>.253**</td>
<td>.403**</td>
<td>-.270**</td>
<td>-.217**</td>
<td></td>
</tr>
<tr>
<td>6. T1 marital conflict</td>
<td>0.722 (0.241)</td>
<td></td>
<td>.242***</td>
<td>.171*</td>
<td>-.182*</td>
<td>.052</td>
<td>.308***</td>
<td>a</td>
<td>a</td>
<td></td>
</tr>
<tr>
<td>7. T1 single parenthood (1 = intact; 0 = single)</td>
<td>0.688 (0.464)</td>
<td></td>
<td>-.070</td>
<td>-.266***</td>
<td>.357***</td>
<td>-.270***</td>
<td>-.265***</td>
<td>-.276**</td>
<td>a</td>
<td></td>
</tr>
<tr>
<td>8. T1 family income (K)</td>
<td>54.423 (38.514)</td>
<td></td>
<td>.049</td>
<td>-.259***</td>
<td>.297***</td>
<td>-.108</td>
<td>-.245**</td>
<td>-.220**</td>
<td>.363**</td>
<td></td>
</tr>
</tbody>
</table>

Note. T1 = Time 1.

<sup>a</sup>These are the same for fathers and mothers because they are family-level variables; they are presented below the diagonal.

* \( p < .05 \)

** \( p < .01 \)

*** \( p < .001 \)
## Table 2

Family Predictors of T4 ODD Symptoms and Initial Linear Rate of Change in ODD Symptoms

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Predicting T4 ODD Symptoms (T4 Model Intercept Growth Factor; Paths a1/ a2 in Figure 1b &amp; 1c)</th>
<th>Predicting Initial Linear Rate of Change Per Year in ODD Symptoms (T1 Model Linear Growth Factor; Paths b1/b2 in Figure 1b &amp; 1c)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \gamma ) (SE)</td>
<td>95% CI</td>
</tr>
<tr>
<td>T1 family income (K)</td>
<td>(-0.008 ) (0.004) *</td>
<td>([-0.016, -0.000])</td>
</tr>
<tr>
<td>T1 single parenthood</td>
<td>(-0.293 ) (0.350)</td>
<td>([-0.979, 0.393])</td>
</tr>
<tr>
<td>T1 marital conflict</td>
<td>(1.134 ) (0.656) *</td>
<td>([-0.152, 2.420])</td>
</tr>
<tr>
<td><strong>Mothers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overreactivity</td>
<td>T1 overreactivity</td>
<td>(1.035 ) (0.423) **</td>
</tr>
<tr>
<td></td>
<td>Overreactivity linear change</td>
<td>(3.531 ) (4.273)</td>
</tr>
<tr>
<td>Laxness</td>
<td>T1 laxness</td>
<td>(0.144 ) (2.155)</td>
</tr>
<tr>
<td></td>
<td>Initial linear change in laxness (a)</td>
<td>(2.465 ) (12.621)</td>
</tr>
<tr>
<td>T1 Warmth</td>
<td>(-0.165 ) (0.170)</td>
<td>([-0.497, 0.167])</td>
</tr>
<tr>
<td>Negative affect</td>
<td>T1 negative affect</td>
<td>(0.299 ) (0.099) **</td>
</tr>
<tr>
<td></td>
<td>T1 negative affect × T4 ADHD</td>
<td>(-0.391 ) (0.127) **</td>
</tr>
<tr>
<td><strong>Maternal depression</strong></td>
<td>T1 depression</td>
<td>(0.108 ) (0.031) ***</td>
</tr>
<tr>
<td></td>
<td>Depression linear change</td>
<td>(0.715 ) (0.299)</td>
</tr>
<tr>
<td><strong>Fathers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overreactivity</td>
<td>T1 overreactivity</td>
<td>(0.774 ) (0.359) *</td>
</tr>
<tr>
<td></td>
<td>Initial linear change in overreactivity (a)</td>
<td>(1.594 ) (0.744) *</td>
</tr>
<tr>
<td>Laxness</td>
<td>T1 laxness</td>
<td>(0.613 ) (0.418) *</td>
</tr>
<tr>
<td></td>
<td>Laxness linear change</td>
<td>(7.325 ) (8.085)</td>
</tr>
<tr>
<td>T1 Warmth</td>
<td>(0.457 ) (0.273)</td>
<td>([-0.078, 0.992])</td>
</tr>
<tr>
<td>Negative affect</td>
<td>T1 negative affect</td>
<td>(0.104 ) (0.145)</td>
</tr>
<tr>
<td></td>
<td>T1 negative affect × T4 ADHD</td>
<td>(-0.168 ) (0.090)</td>
</tr>
<tr>
<td><strong>Paternal depression</strong></td>
<td>T1 depression</td>
<td>(0.072 ) (0.065)</td>
</tr>
<tr>
<td></td>
<td>Depression linear change</td>
<td>(0.390 ) (0.508)</td>
</tr>
</tbody>
</table>

Note. T1 = Time 1. T4 = Time 4. Models were estimated separately for each parenting variable and separately for mothers and fathers.

\(a\) Linear change represent initial linear rate of change per year for these models.
The ODD quadratic growth factor was also significantly predicted by paternal warmth in both the T1 Model, \( \gamma = 0.187, SE = 0.090, p = 0.038 \), and T4 Model, \( \gamma = 0.285, SE = 0.124, p = 0.022 \).

* \( p < .05 \).

** \( p < .01 \).

*** \( p < .001 \).

† \( p < .10 \).
Table 3

Relations Between T4 ADHD/T1 Hyperactivity and Family Experience Variables

<table>
<thead>
<tr>
<th>Family Experience Variable (as outcome)</th>
<th>T4 ADHD Status as Predictor</th>
<th>T1 BASC Hyperactivity as Predictor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>γb (SE) 95% CI</td>
<td>γb (SE) 95% CI</td>
</tr>
<tr>
<td>T1 marital conflict</td>
<td>0.105 (0.041) **</td>
<td>0.003 (0.002) †</td>
</tr>
<tr>
<td>Mothers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overreactivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 overreactivity</td>
<td>0.043 (0.115) −0.182, 0.268</td>
<td>0.009 (0.004) *</td>
</tr>
<tr>
<td>Overreactivity linear change</td>
<td>0.065 (0.041) −0.156, 0.145</td>
<td>−0.002 (0.002) −0.006, 0.002</td>
</tr>
<tr>
<td>Laxness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 laxness</td>
<td>0.150 (0.145) −0.134, 0.434</td>
<td>0.020 (0.005) **</td>
</tr>
<tr>
<td>Initial linear change in laxness</td>
<td>0.041 (0.042) −0.041, 0.123</td>
<td>−0.002 (0.002) −0.006, 0.002</td>
</tr>
<tr>
<td>T1 warmth</td>
<td>−0.461 (0.154) −0.763, −0.159</td>
<td>−0.031 (0.006) **</td>
</tr>
<tr>
<td>T1 negative affect</td>
<td>0.798 (0.411) −0.008, 1.604</td>
<td>0.065 (0.015) **</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 depression</td>
<td>2.078 (1.058) [0.004, 4.152]</td>
<td>0.117 (0.038) **</td>
</tr>
<tr>
<td>Depression linear change</td>
<td>0.113 (0.273) −0.442, 0.648</td>
<td>0.007 (0.011) −0.015, 0.029</td>
</tr>
<tr>
<td>Fathers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overreactivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 overreactivity</td>
<td>0.072 (0.131) −0.185, 0.329</td>
<td>−0.006 (0.005) −0.016, 0.004</td>
</tr>
<tr>
<td>Initial linear change in overreactivity</td>
<td>0.080 (0.056) −0.030, 0.190</td>
<td>0.004 (0.002) †</td>
</tr>
<tr>
<td>Laxness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 laxness</td>
<td>0.347 (0.144) 0.065, 0.629</td>
<td>0.014 (0.006) **</td>
</tr>
<tr>
<td>Laxness linear change</td>
<td>−0.064 (0.056) −0.174, 0.046</td>
<td>−0.001 (0.002) −0.005, 0.003</td>
</tr>
<tr>
<td>T1 warmth</td>
<td>−0.026 (0.161) −0.342, 0.290</td>
<td>−0.007 (0.007) −0.020, 0.006</td>
</tr>
<tr>
<td>T1 negative affect</td>
<td>0.437 (0.317) −0.184, 1.058</td>
<td>0.035 (0.012) **</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 depression</td>
<td>1.907 (0.824) [0.292, 3.522]</td>
<td>0.124 (0.033) **</td>
</tr>
<tr>
<td>Depression linear change</td>
<td>0.780 (0.276) [0.239, 1.321]</td>
<td>0.001 (0.012) −0.023, 0.025</td>
</tr>
</tbody>
</table>

Note. T1 = Time 1, T4 = Time 4. For marital conflict, warmth, and negative affect, each family variable was regressed on T4 ADHD (1 = ADHD, 0 = not ADHD) and then on T1 hyperactivity in separate linear regression models. For overreactivity, laxness, and depression, family experience growth factors were estimated with time centered at T1. These factors (intercept growth factors as estimates of T1 family variable level and linear growth factors as estimates of linear rate of change per year in the family variable) were then each predicted by T4 ADHD status and T1 hyperactivity in separate models.

* p < .05;
** p < .01;
*** p < .001,
† p < .10.

J Consult Clin Psychol. Author manuscript; available in PMC 2012 December 1.
Table 4

Tests of Family Experience Variables as Mediators of the Relation Between T1 Hyperactivity and T4 ODD Symptoms

<table>
<thead>
<tr>
<th>Family Experience Variable</th>
<th>Direct Path from T1 Hyperactivity to Family Experience Variable $\gamma_a (SE)$</th>
<th>Direct Path from Family Experience Variable to T4 ODD Symptoms $\gamma_b (SE)$</th>
<th>Direct Path from T1 Hyperactivity to T4 ODD Symptoms $\gamma_c (SE)$</th>
<th>Indirect Effect&lt;sup&gt;a&lt;/sup&gt; $\gamma_a \times \gamma_b (SE)$ [95% confidence]</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 marital conflict</td>
<td>0.003 (0.002)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.870 (0.610)</td>
<td>0.029 (0.013)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.003 (0.003) [-0.002, 0.008]</td>
</tr>
<tr>
<td>T1 maternal overreactivity</td>
<td>0.009 (0.004)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.952 (0.354)&lt;sup&gt;**&lt;/sup&gt;</td>
<td>0.032 (0.014)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.009 (0.006) [-0.001, 0.018]</td>
</tr>
<tr>
<td>T1 maternal depression</td>
<td>0.120 (0.037)&lt;sup&gt;***&lt;/sup&gt;</td>
<td>0.064 (0.029)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.020 (0.018)</td>
<td>0.008 (0.004) [0.000, 0.016]</td>
</tr>
<tr>
<td>Maternal depression linear rate of change</td>
<td>0.006 (0.010)</td>
<td>1.377 (1.107)</td>
<td>0.020 (0.018)</td>
<td>0.008 (0.015) [-0.022, 0.038]</td>
</tr>
<tr>
<td>T1 paternal overreactivity</td>
<td>-0.006 (0.005)</td>
<td>0.418 (0.236)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>0.032 (.034)</td>
<td>-0.0030 (0.003) [-0.007, 0.002]</td>
</tr>
<tr>
<td>Paternal overreactivity linear rate of change</td>
<td>0.004 (0.002)&lt;sup&gt;*&lt;/sup&gt;</td>
<td>3.508 (8.176)</td>
<td>0.032 (.034)</td>
<td>0.014 (0.033) [-0.051, 0.080]</td>
</tr>
</tbody>
</table>

Note. T1 = Time 1. T4 = Time 4.

<sup>a</sup>Sobel t (Sobel, 1982) was used to test significance of indirect effect. This approach is a widely used and conservative method of testing significance of indirect effects (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002).

<sup>*</sup>p < .05;
<sup>**</sup>p < .01;
<sup>***</sup>p < .001.

<sup>f</sup>p < .10.