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Longitudinal Associations between Parental and Children's Depressive Symptoms in the Context of Interparental Relationship Functioning

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Abstract

Using longitudinal, multi-informant data from the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development, the present study tested associations between trajectories of parental and child depressive symptoms from ages 11 to 15 years. Consistent with predictions, changes in mothers' and fathers' depressive symptoms were positively associated with change in children's depressive symptoms over time. In addition, youth characteristics of sex and pubertal development moderated the trajectories, with children more advanced on pubertal development showing higher initial levels of depressive symptoms, and girls demonstrating steeper slopes of depressive symptoms over time. The context of interparental relationship functioning (i.e., marital conflict, marital conflict resolution) moderated both the trajectories of child depressive symptoms and the interplay between parental and child depressive symptoms in ways largely consistent with hypotheses. Implications of the findings are discussed in terms of treating youth depressive symptoms with a consideration of the broader family context, including parental and interparental functioning.

Keywords

Childhood depression; Longitudinal design; Marital functioning; Multilevel modeling; Parental depression

Introduction

One of the most persistent aims of psychological inquiry is to capture how children develop over time, especially with respect to problematic outcomes and distressing symptoms. A central motivation for this interest is the increased risk of subsequent and more severe problems as a function of earlier adjustment: Across domains such as affective and behavioral problems (including aggression), personality disorders, and psychotic symptoms, early distress has been shown to predict later dysfunction and diagnoses (e.g., Babinski, Hartsough, & Lambert, 1999; Loeber & Hay, 1997; Poulton, Caspi, Moffitt, Cannon, Murray, & Harrington, 2000). Depressive symptoms have shown particular developmental continuity and warrant further investigation for their connection to psychosocial dysfunction (e.g., self-esteem, coping skills) and subsequent diagnosable disorders, including depression and substance use (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Moreover, knowledge of longitudinal symptom trajectories may reveal family-wide correlates of children's chronic depression and need for clinical treatment (Rivas-Vazquez, Saffa-Biller, Ruiz, Blais, & Rivas-Vazquez, 2004).

Childhood Depressive Symptoms

Improving our understanding of the course of psychological distress during the transition to adolescence is crucial. First, distress is relatively common at this point: Depression occurs at substantial rates in adolescence with 20–40% of youth likely to report depressed mood symptoms (Petersen, Compas, Brooks-Gunn, Stemmler, Ey, & Grant, 1993). In addition, longitudinal data suggest that pubertal timing and changes are linked with changes in depressive symptoms, with earlier physical maturation predictive of elevated depression symptoms (Ge et al., 2003). Furthermore, research suggests that youth from both clinical and nonclinical samples are affected, with the developmental course (including diagnostic prevalence and recurrence) of depression in young adults from community-versus clinic-based samples appearing more similar than disparate. Specifically, Dunn and Goodyer (2006) found that children's subsequent adult depression diagnosis was not accounted for childhood depression status, but rather was predicted by the timing and severity of symptoms experienced across childhood.

Predictors of Trajectories of Child Depression Symptoms

Youth characteristics—Models of child depressive symptoms over time should include youth characteristics of puberty and sex, which have been shown in past research to potentially affect the intercept (i.e., starting level) and slope (i.e., change over time) of symptom trajectories. Whereas pre-pubertal sex difference are small, girls are more likely than boys to show increases in depressive symptoms in contexts of stress (e.g., family conflict; Essex, Klein, Cho, & Kraemer, 2003; Nolen-Hoeksema & Girgus, 1994) and pubertal development (Ge, Conger, & Elder, 1996; Stice, Presnell, & Bearman, 2001). Moreover, twice as many females receive a depression diagnosis as compared to men, a difference that begins in adolescence (see Hankin, Wetter, & Cheely, 2008). Using large community samples of females (10–15 years of age) from the United States and Australia, Patton et al. (2008) showed that advanced pubertal stage is associated with higher risks for the onset and persistence of depressive symptoms; social distress (e.g., family conflict) also predicted the course of depressive symptoms.

Mother and father depressive symptoms—Elevated levels of parental depressive symptoms place children at risk for a wide range of maladaptive outcomes, including internalizing and externalizing problems along with cognitive, social, and physical disruptions (see reviews in Downey & Coyne, 1990; Gunlicks & Weissman, 2008). Children of depressed parents are more likely than children of nondepressed parents to have diagnosable disorders, especially depression (Hammen, 1991). Connell and Goodman's (2002) meta-analysis documented stronger associations between parents' mental health and child internalizing problems (e.g., depressive symptoms) for mothers than fathers, although there is evidence for the importance of both (see Kane & Garber, 2004). However, associations between parent and child symptoms were significant but small, with parents' mental health accounting for less than 5% of variance in child behavior problems, suggesting that the study of additional family factors is well warranted (Connell & Goodman, 2002).

The Context of Interparental Relationship Functioning

The quality of the interparental relationship is an important developmental environment and interacts with parental psychological distress to predict child functioning (Cummings, Davies, & Campbell, 2000). Previous findings point to complex pathways among the family risk factors, suggesting that family variables beyond parental symptoms alone predict child depression over time (e.g., Shelton & Harold, 2008). Marital functioning plays an important role in how youth depression trajectories unfold over time, with conflict processes having

received the most attention (e.g., Keller, Cummings, Peterson, & Davies, 2009). The present study considers marital conflict and marital conflict resolution jointly, in line with calls for a multidimensional approach to interparental relationship functioning (Fincham & Linfield, 1997), as they may differentially interact with parental symptoms to predict trajectories of child depressive symptoms.

The Current Study

In line with the developmental psychopathological perspective, the current study considers the developmental course of depressive symptoms (rather than diagnoses) in the context of youth, parental, and family factors (Cicchetti, 2006; Cummings et al., 2000). The aim of the study was two-fold. The first aim was to model child trajectories of depressive symptoms from age 11 to 15 as predicted by youth characteristics and parental depressive symptoms. On the basis of past research, youth sex and pubertal development were predicted to moderate such that youth who were more advanced on pubertal development would evidence higher levels of depressive symptoms, and that girls would show steeper increases than boys in depressive symptoms across the transition to puberty. In addition, mother and father depressive symptoms were expected to be associated with higher levels of youth depressive symptoms. Following Connell and Goodman (2002), mothers' symptoms were hypothesized to be more closely linked than fathers' to child depression over time. The second aim was to test whether the family-level process of interparental relationship functioning moderated youth depression trajectories (i.e., intercept or slope) or the links between parent and youth depression over time. Marital conflict and marital conflict resolution were expected to moderate differentially, with conflict hypothesized to amplify child depressive symptom trajectories, on the one hand, and conflict resolution predicted to attenuate symptom trajectories, on the other hand. In addition, marital functioning was expected to similarly moderate associations between parent and child depression symptoms, with conflict predicted to strengthen parent-child depression links, and conflict resolution expected to weaken the interplay between parent and child depressive symptoms.

Method

Study Design

Participants for this study were a subset of families drawn from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD), a longitudinal sample of 1,364 children born at 31 hospitals near 10 U.S. research sites during 1991 (NICHD Early Child Care Research Network, 2001). The original 1,364 families were selected following a conditional random-sampling plan designed such that the participants reflected the economic, ethnic, and educational diversity of each research site. The SECCYD – designed primarily to examine linkages between variations in childcare characteristics and children's developmental outcomes over time – collected a wealth of information on childcare, family, and school contexts, and child development. Detailed description of data collection is available at http://secc.rti.org.

At each assessment point, a standardized interview was conducted with mothers to obtain information on family living arrangements and other demographic data. Mothers provided information regarding their partner relationship status, including whether they were married, separated, widowed, or divorced. Based on this data, measures of interparental relationship functioning (described below) were administered to mothers who were living with a husband or partner, and to the mother's husband/partner.

Participants

The present study utilized data from 699 families (51.2% of the 1,364 enrolled in the study at birth), who contributed to the data at least (1) one youth depression score between 11 and 15 years of age, and (2) one parent-reported interparental relationship index at age 11. In the present study's analysis sample, maternal age at the 0.1-year assessment ranged from 18 to 46 years ($M = 29.21$, $SD = 5.38$). An income-to-needs ratio, calculated by dividing the total family income by the U.S. poverty threshold for the appropriate family size, averaged 3.22 ($SD = 2.65$) at the 0.1-year assessment. Mothers reported the following racial/ethnic distribution of their child: American Indian, Eskimo, and Aleut (0.1%); Asian or Pacific Islander (1.3%); Black or African American (7.9%); White (86.4%); and other (4.3%). Forty-three children (6.2%) were Hispanic. A dichotomous ethnicity variable (0 = *White*, 1 = *Racial/Ethnic Minority*) was created for analytic purposes. The children included in this sample consisted of 343 (49.1%) boys.

Overall, the SECCYD maintained very high retention, with over 1,000 (> 70%) of the children and families followed through age 15. Attrition analyses compared the demographic characteristics of the full sample of 1,364 families to the subset included in the present analyses. Based on the 0.1-year assessment, included families had older mothers ($M = 29.21$ years vs. 26.96 years, $t = 7.52$, $p < .001$) and higher financial resources (i.e., income-to-needs ratio) ($M = 3.22$ vs. 2.47, $t = 5.16$, $p < .01$). Also, included mothers were more likely than non-included mothers to be White, 86% vs. 74%, $\chi^2(1, N = 1364) = 32.61$, $p < .01$. Comparisons further indicated that non-included families were more likely than included families to have boys, 54% vs. 49%, $\chi^2(1, N = 1364) = 3.93$, $p = .047$. Relevant to the current study's goal of testing longitudinal linkages between parental and youth depressive symptom trajectories, previous analysis of longitudinal SECCYD data revealed no systematic attrition based on maternal depression scores at the 0.1-year assessment (Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007).

Measures

The Child Depression Inventory-Short Form (CDI; Kovacs, 1992) is a self-report instrument for measuring depressive symptoms in children and adolescents that typically yields similar results as the 27-item form (Kovacs, 1981). Children respond to items assessing sadness, self-blame, and interpersonal relationships by selecting which of the three descriptions best fits how they have been feeling during the past two weeks (e.g., "I do most things O.K., I do many things wrong, I do everything wrong") for 10 items. The CDI has demonstrated acceptable reliability and validity properties in past research (Sitarenios & Kovacs, 1999) and is most appropriately treated as a continuous measure of mood (Matthey & Petrovski, 2002). All three waves of the CDI were completed by 90% of the children in the sample ($n = 629$), two waves by 8.2% of the sample ($n = 57$), and one wave by 1.9% of the sample ($n = 13$). Cronbach alphas at ages 10, 11, and 15, respectively, were .73, .76, and .81.

In addition to youth sex (343 male, 356 female), pubertal development at age 11 was included as a covariate. Mothers completed the appropriate version (male or female) of the Pubertal Development Scale (PDS; Peterson, Crockett, Richards, & Boxer, 1988). The PDS consists of items concerning whether specific physical changes have occurred (pimply skin, growth spurt, breast development, facial hair), which are rated on a scale from 1 (*no development*) to 4 (*development already past*). A pubertal development score was computed as the average of the item responses, with higher values indicating more advanced pubertal development. Table 1 shows the descriptive statistics of the youth characteristics.

Maternal and paternal depressive symptoms were assessed using the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977), a twenty-item self-

report instrument. Respondents indicated how frequently they have been bothered by the listed depressive symptoms during the past week, ranging from 0 (*less than one day*) to 3 (*five to seven days*). Examples include "I felt that everything I did was an effort" and "I was bothered by things that usually don't bother me." Individuals diagnosed with clinical depression score higher on the CES-D than non-depressed individuals. The CES-D is widely used to assess depressive symptoms among normative samples, including in research focused on linkages between mental health and family functioning (e.g., Brody & Flor, 1997). Mothers' and fathers' CES-D scores demonstrated high internal consistency in the current sample: Cronbach alphas ranged from .87 to .92.

The Partner Relationship Questionnaire includes the Braiker and Kelley (1979) conflict subscale, which consists of 5 items that tap disagreement and negativity (e.g., "How often do you feel angry or resentful toward your partner?"). Responses to items ranged from 1 (*not at all*) to 9 (*very much*), and the scale score was computed by averaging responses to the 5 items. Thus, scores could range from 1 to 9, with higher scores indicating greater partner relationship conflict. The Partner Relationship Questionnaire also includes Kerig's (1996) conflict resolution scale, which consists of 13 questions that assess the degree of positivity in the handling of interparental disagreements. A sample item is, "We have fun making up with each other." Responses to items ranged from 0 (*never*) to 3 (*usually*), with the scale score computed by following a proportional weighting procedure in which items were multiplied by −2 (reverse coded), 1, or 2. The final scale score was a sum of responses to the 13 items, with higher scores indicating more positive affect in the resolution of conflict. Both measures have demonstrated sound psychometric properties (Felmlee, Sprecher, & Bassin, 1990; Kerig, 1996). Across interparental relationship functioning subscales, mother and father Cronbach alphas exceeded .80 in the present study. Mothers' and fathers' ratings of marital conflict, $r\,(n = 603) = .41$, $p < .001$, and marital conflict resolution, $r\,(n = 601) = .44$, $p < .001$, were significantly correlated. Accordingly, when mother and husband/partner scores both were available, an average of the scores was used for analytic purposes; a single score was retained when marital functioning measures were available for only one partner. Table 1 shows the descriptive statistics of the interparental relationship functioning variables.

Results

Data Analysis

The present study's hypotheses were tested with multilevel modeling using Hierarchical Linear Modeling (HLM) statistical software (version 6.08, Raudenbush, Bryk, Cheong, & Congdon, 2004). Multilevel modeling is ideally suited to test hypotheses concerning variables that have been collected repeatedly over time, and offers the advantages of allowing uneven interval spacing between assessments and accommodating individually-varying numbers of data points (Papp, 2004; Raudenbush & Bryk, 2002). Specifically, related to the present hypotheses, multilevel modeling derives parameters from repeated measurements collected from each parent and child over time (in Level 1), which are averaged simultaneously across all participants and related to youth- and family-level characteristics (in Level 2). Specifically, HLM estimated longitudinal trajectories (i.e., intercept and slope parameters) of child depressive symptoms as predicted by other time-varying variables (i.e., parental depression, child age) for each participating family (in Aim 1), and then related these averaged trajectories to family-level characteristics (i.e., youth characteristics, interparental relationship functioning) (in Aim 2). Results below report unstandardized coefficients, and all available data were utilized due to maximum likelihood estimation (Raudenbush, 2001).

To address the first aim, multilevel modeling was utilized to model child depressive symptoms as predicted by mother depressive symptoms, father depressive symptoms, and change over time (i.e., child age at assessment). The Level 1 model included repeated assessments of CDI scores, which demonstrated non-normal distributions on average, as the outcome variable. Accordingly, results were obtained using hierarchical generalized linear modeling (HGLM), an extension of HLM that estimates a Poisson sampling model to appropriately predict dependent variables with non-normal distributions (Raudenbush & Bryk, 2002). To address the second aim of testing whether marital functioning moderated the trajectories of child depressive symptoms and the longitudinal linkages between parent depression and child depression, interparental relationship functioning variables (i.e., marital conflict, marital conflict resolution) were added to Level 2 of the HGLM identified in Aim 1.

Family Members' Depressive Symptoms over Time

On average, children reported relatively low levels of depression symptoms at age 11 (M = 1.26, SD = 2.00, range 0–15), age 12 (M = 1.39, SD = 2.22, range 0–19), and age 15 (M = 1.97, SD = 2.66, range = 0–18). Results from HGLM confirmed that child depressive symptom scores increased over time (coeff = 0.104, t = 6.41, p < .001). HGLM results further revealed that mothers' and fathers' respective depressive symptom scores also increased over time (coeff = 0.028, t = 3.38, p = .001; coeff = 0.046, t = 4.90, p < .001).

Longitudinal Linkages between Parent and Child Depressive Symptoms

An unconditional HGLM that regressed on child depressive symptoms the predictors of maternal depression, paternal depression, and child age (i.e., timing of each assessment) indicated positive linkages between depressive symptoms for mothers and children (coeff = 0.017, t = 3.76, p < .001) and fathers and children (coeff = 0.032, t = 5.58, p < .001). However, as noted above, the aim was to test the hypothesis in a model that appropriately included relevant youth characteristics that might account for the level of or change in child depressive symptoms.

The Poisson distribution assumes the expected value (E) and variance (V) of child depressive symptoms given the event rate (λ). At Level 1, a child's depressive symptoms score was predicted by child age (in years, centered at age 11), and mother and father depressive symptoms (matched by child age), such that the resulting Level 1 Poisson model is:

$$\mathrm{E}(\text{Child depressive symptoms score}|\beta)=\lambda$$
$$\mathrm{V}(\text{Child depressive symptoms score}|\beta)=\lambda$$
$$\log[\lambda]=\beta_{0ij}+\beta_{1ij}(\text{Mother Depressive Symptoms})+\beta_{2ij}(\text{Father Depressive Symptoms})+\beta_{3ij}(\text{Child Age})$$

At Level 2, child sex (0 = *male*, 1 = *female*) and pubertal development score (centered on the sample mean) were entered as covariates that predicted the depressive symptoms score intercept, or the starting value (β_{00}), and the depressive symptoms score slope, or the change over time (β_{30}):

$$\beta_{00j}=\gamma_{000}+\gamma_{001}(\text{Child PDS})+\gamma_{002}(\text{Child Sex})$$
$$\beta_{10j}=\gamma_{100}+U_{10}$$
$$\beta_{20j}=\gamma_{200}+U_{20}$$
$$\beta_{30j}=\gamma_{300}+\gamma_{301}(\text{Child PDS})+\gamma_{302}(\text{Child Sex})$$

In sum, this hierarchical generalized linear model yields the longitudinal associations between mothers' and children's depressive symptoms (γ_{100}) and fathers' and children's depressive symptoms (γ_{200}), accounting for the effects of development over time (in Level 1) and previously identified youth characteristics (in Level 2).

As shown in Table 2, youth characteristics moderated the child depressive symptoms trajectories such that (1) higher pubertal development scores predicted a higher depressive symptoms intercept, or starting point, at age 11, and (2) trajectories of depressive symptoms increased more steeply during the transition to adolescence among girls than boys. Both covariates were retained in all subsequent analyses. As predicted, depressive symptoms of both mothers and fathers were associated with higher child depressive symptom scores over time (see Table 2). Hypothesis testing indicated that fathers' depressive symptoms were a stronger predictor than mothers' depressive symptoms of child depressive symptoms, $\chi^2(1, N = 699) = 5.15$, $p = .022$.

The next model tested whether the youth covariates (i.e., pubertal development score, child sex) moderated the parental depression – child depression linkages (β_{10j} and β_{20j}). The HGLM results indicated no significant moderator effects (ps > .05). Hence, child sex and pubertal development scores were not retained as moderators, thereby maximizing the statistical power of subsequent model tests.

The Context of Interparental Relationship Functioning

To investigate whether interparental relationship functioning moderated the trajectories of child depressive symptoms (i.e., intercept and slope), and the longitudinal linkages between parent depression and child depression, the previous Level 1 model was retained and marital functioning variables were added to Level 2:

$$\beta_{00j}=\gamma_{000}+\gamma_{001}(\text{Pubertal Development})+\gamma_{002}(\text{Child Sex})+\gamma_{003}(\text{Marital Functioning})$$
$$\beta_{10j}=\gamma_{100}+\gamma_{101}(\text{Marital Functioning})+U_{10}$$
$$\beta_{20j}=\gamma_{200}+\gamma_{201}(\text{Marital Functioning})+U_{20}$$
$$\beta_{30j}=\gamma_{300}+\gamma_{301}(\text{Pubertal Development})+\gamma_{302}(\text{Child Sex})+\gamma_{303}(\text{Marital Functioning})$$

The moderating contexts of marital conflict and marital conflict resolution were tested in separate models. As shown in the left-hand column of Table 3, HGLM results indicated that marital conflict moderated change over time in child depressive symptom scores such that depressive symptoms increased at higher levels of marital conflict. However, marital conflict did not moderate the intercept of the child depressive symptoms trajectories, or the positive associations between mother-child depressive symptoms and father-child depressive symptoms.

The marital conflict resolution model (shown in the right-hand column of Table 3) revealed that the positive link between mother-youth depressive symptoms was not moderated by marital conflict resolution, but the father-child depressive symptoms association was moderated such that the positive link was attenuated for higher levels of positive affect in the resolution of interparental conflict. Consistent with predictions, marital conflict resolution moderated change over time in youth depressive symptoms such that the positive trend over time was weakened at higher levels of marital conflict resolution. In addition, child depressive scores at the initial timepoint (age 11) were positively associated with marital conflict resolution ratings by parents. However, follow-up HGLMs indicated that marital conflict resolution was not associated with youth depressive symptoms at age 12 or age 15 (ps > .05, results not shown).

Discussion

The present study investigated trajectories of depressive symptoms across 11 to 15 years of age in a relatively large community-based sample of families, and tested whether youth characteristics and parental depressive symptoms moderated the trajectories (i.e., initial levels or slopes). In addition, indicators of interparental relationship functioning (i.e., marital conflict, marital conflict resolution) were tested as moderators of the trajectories and the longitudinal linkages between parental and child depression. Results from multilevel modeling indicated that trajectories of depressive symptoms across the transition to adolescence vary as a function of youth characteristics and parental depressive symptoms. Specifically, the current results aligned with previous findings that indicate more advanced pubertal development reflects a transition to risk (Haynie, 2003; Patton et al., 2008; Stice et al., 2001), whereby children who reported higher levels of pubertal development at age 11 also evidenced higher levels of concurrent depressive symptoms. In addition, depressive symptoms changed over time depending on youth sex, with girls showing steeper increases than boys in symptoms, replicating previous work based on community samples (Ge, Lorenz, Conger, Elder, & Simons, 1994). However, child sex and pubertal development level did not moderate the positive parent-child depression associations.

Considering the context of interparental functioning added to the models and contributed information to the understanding of trajectories of children's depressive symptoms. Specifically, indicators of how parents handled their marital disagreements (at age 11) were associated with how children's depressive symptoms changed over the transition to puberty, with elevated marital conflict related to steeper increases over time, and greater conflict resolution related to attenuated increases. Marital conflict resolution also moderated the longitudinal linkage between father-child depressive symptoms, such that conflict resolution buffered the strength of the positive association over time. That is, parents' successful handling of marital conflict (e.g., feeling closer to one another after the disagreement, resolving the issue) when children were 11 years of age had a protective effect on the positive interplay between fathers and children's psychological symptoms over time. Contrary to predictions, marital conflict did not moderate the linkages between parent and child depressive symptom trajectories, suggesting that the variance in child depressive symptoms was accounted for by youth characteristics and parent symptoms over time. It is possible that longitudinal assessments of conflict (rather than at age 11 alone) would have better captured how family mental health and relationship functioning co-vary over time.

The results support developmental psychopathology notions that the course of child depressive symptoms is related to more than parental symptomatology alone, and that multiple domains of family functioning must be considered to understand the development of youth mental health processes (Cummings et al., 2000). The results also point to potentially disparate mechanisms through which maternal versus paternal psychological distress relates to youth functioning, a process that should continue to be explored in future research. In addition, positive and negative aspects of marital functioning had differential effects on children's depressive symptoms trajectories, consistent with multidimensional views of interparental relationship functioning.

Limitations

Although the original study families reflected the economic, educational, and ethnic diversity of each data collection site, they were not representative of U.S. families in general. Indeed, the study's eligibility criteria, namely that the mother was 18 years or older, did not plan to move, and had no known substance abuse problem or serious illness, may have precluded the involvement of participants with the highest risk for evidencing depressive and other symptoms. Moreover, secondary data analysis, while efficient and

valuable, is often accompanied by decisions resulting in missing data (McKnight & McKnight, 2011). As noted above, all of the marital functioning variables were not assessed at all timepoints, which limits how they could be included in the statistical models. In addition, the current study included data from only 3 timepoints, which limited the shape of the symptom trajectories and the number of variance parameters that could be estimated freely. In addition, the current study did not include a genetic component: Recent findings indicate that genetic factors may be more predictive of longitudinal child depressive symptoms when family conflict levels are higher (Rice, Harold, Shelton, & Thapar, 2006).

Treatment Implications

Despite these limitations, the findings offer implications for the treatment of childhood depressive symptoms in the family context. Treatment of parental psychological symptoms has been shown to reduce children's symptoms (see Gunlicks & Weissman, 2008), supporting consideration of within-family psychological adjustment. Not only did the present findings implicate parental symptoms in the course of youth depressive symptoms, as predicted, but they also demonstrated the importance of interparental relationship functioning. Hence, clinicians who treat children's symptom distress are encouraged adopt a family-wide perspective and consider multiple domains of parental and interparental functioning. Furthermore, results from this study contribute to a growing literature that suggests that even subclinical levels of family members' symptoms may have distressing and long-term consequences (Beach & Fincham, 1998), supporting efforts to prevent and treat the longitudinal interplay between family members' psychological distress and interparental relationship functioning for the benefit of parents, couples, and children.

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Table 1

Descriptive Statistics: Youth Characteristics and Interparental Relationship Functioning

Variable	*M* or %	*SD*	Range
Youth characteristics			
Pubertal development	1.54	0.49	1.00–3.40
Sex	49.1% male		
Interparental relationship functioning			
Marital conflict	3.44	1.26	1.00–8.20
Marital conflict resolution	5.49	10.79	−35.00–23.00

Table 2

Study Aim 1: Longitudinal Linkages between Parental and Children's Depressive Symptoms

Fixed Effect	Coeff.	*t*	*df*	*p*
Intercept, γ_{000}	−0.127	−1.24	1751	.216
Pubertal development, γ_{001}	0.244	2.10	1751	.036
Sex, γ_{002}	−0.102	−0.84	1751	.402
Intercept, γ_{100}	0.017	3.66	698	<.001
Intercept, γ_{200}	0.033	5.62	698	<.001
Intercept, γ_{300}	0.016	0.60	1751	.546
Pubertal development, γ_{301}	−0.035	−0.99	1751	.321
Sex, γ_{302}	0.164	4.55	1751	<.001

Note. Coeff. = coefficient.

Table 3

Study Aim 2: Longitudinal Linkages between Parental and Children's Depressive Symptoms in the Context of Interparental Relationship Functioning

	Marital conflict model				Marital conflict resolution model			
Fixed Effect	**Coeff.**	***t***	***df***	***p***	**Coeff.**	***t***	***df***	***p***
Intercept, γ_{000}	−0.145	−1.45	1747	.148	−0.168	−1.62	1745	.105
Pubertal devel., γ_{001}	0.241	2.08	1747	.037	.211	1.84	1745	.066
Sex, γ_{002}	−0.100	−0.82	1747	.410	−0.089	−0.74	1745	.462
Marital funct., γ_{003}	−0.093	−1.32	1747	.186	0.017	2.08	1745	.038
Intercept, γ_{100}	0.018	3.83	697	<.001	0.018	3.84	696	<.001
Marital funct., γ_{101}	−0.001	−0.29	697	.772	0.0003	0.77	696	.443
Intercept, γ_{200}	0.033	5.42	697	<.001	0.034	5.47	696	<.001
Marital funct., γ_{201}	0.006	1.29	697	.199	−0.001	−2.10	696	.036
Intercept, γ_{300}	0.018	0.67	1747	.503	0.014	0.50	1745	.616
Pubertal devel., γ_{301}	−0.034	−0.97	1747	.334	−0.026	−0.73	1745	.467
Sex, γ_{302}	0.164	4.56	1747	<.001	0.169	4.66	1745	<.001
Marital funct., γ_{303}	0.032	2.41	1747	.016	−0.005	−2.96	1745	.004

Note. Coeff. = coefficient.