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ORIGINAL PAPER

Associations of Parent–Child Anxious and Depressive Symptoms When a Caregiver Has a History of Depression

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Abstract We examined the associations between parent and child anxious and depressive symptoms controlling for co-occurring symptoms in both. One hundred and four families participated, including 131 9–15 year old children considered at risk for anxiety and/or depression due to a history of depression in a parent. Parents and children completed questionnaires assessing depressive and anxious symptoms. Linear Mixed Models analyses controlling for the alternate parent and child symptoms indicated that both parent and child depressive symptoms and parent and child anxious symptoms were positively associated. Parental depressive symptoms were not positively associated with child anxious symptoms, and parental anxious symptoms were not positively associated with child depressive symptoms. The findings provide evidence for positive specific links between parent and child development of same-syndrome, but not cross-syndrome, symptoms when a caregiver has a history of depression.

Keywords Parent · Child · Anxious symptoms · Depressive symptoms

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Introduction

Major depressive disorder (MDD) is currently recognized as one of the leading causes of disease-related disability worldwide and is predicted to be *the* leading cause by 2020 (Hirschfield 2001; World Health Organization 2007). Unfortunately, the debilitating effects of MDD (Kessler et al. 2003) are not limited to the individuals diagnosed; when parents are depressed, their depression has the potential to adversely impact their children. Over the past 30 years, literature has amassed indicating that children and adolescents living with a depressed caregiver are at a substantial risk for a variety of developmental and adjustment difficulties. These difficulties can emerge and persist at any point from infancy through adulthood and include an increased risk for internalizing and externalizing problems (for reviews see Goodman and Gotlib 1999; Goodman 2007; Kane and Garber 2004).

Both during an episode and when subthreshold levels of symptoms persist into remission, parental depressive symptoms have been associated with child internalizing problems, generally (e.g., Foster et al. 2008), and depressive symptoms, specifically (e.g., Abela et al. 2006). However, not all research with this population has supported a relation between parental depressive symptoms and negative child outcome (e.g., Langrock et al. 2002). The mixed findings highlight the importance of conducting investigations with appropriate controls to ascertain the role of parental depressive symptoms in child outcome when a caretaker has a history of depression.

Of relevance, depressive and anxious symptoms and disorders frequently co-occur in adults (e.g., Goodman 2007; Kessler et al. 2003) and children (e.g., Angold and Costello 1993; Rudolph et al. 2006). Because of the overlap between these two disorders, it is difficult to

determine whether anxious symptoms alone, depressive symptoms alone, or the combination of these two sets of symptoms contribute to negative outcomes among children of depressed parents. Thus, it is important to try to understand which set of symptoms has which "effect" in order to better understand the mechanism of risk transmission. In order to disentangle the role of these two types of symptoms, it is necessary to control for co-occurring parent and child anxious and depressive symptoms (see Colletti et al. 2009; Shanahan et al. 2008, for a discussion of this issue). Without such controls, conclusions about associations between the sets of symptoms in parents and children cannot be reached.

Although not conducted with caregivers with a history of depression and not focusing on depressive and anxious symptoms specifically, the recent Shanahan et al. (2008) study represents a rare example of a study controlling for *both* co-occurring parental symptoms and co-occurring child symptoms. In contrast to the previous literature reviewed by Shanahan et al. (2008) that did not control for these variables, the authors found that most risk factors were related to one specific child problem behavior rather than the typically reported diffuse negative child outcomes (e.g., West and Newman 2003). Of particular interest, parental depression was related to child depression but not to a combined measure of child anxiety disorders (Shanahan et al. 2008). This provides some evidence that *specific* symptoms in parents are related to *specific* outcomes for children.

As Goodman (2007) has noted, research examining the role of comorbid disorders and symptoms in child outcomes when a caregiver has a history of depression is limited but necessary. Additional research in the vein of Shanahan et al. (2008) discussed above would make a valuable contribution to an understanding of the specificity of each disorder as well as their combined impact. In one of a very few such studies, McClure et al. (2001) examined the role of parental anxiety in a sample of children for whom two-thirds of the mothers were at high risk for lifetime depression. A parental anxiety diagnosis without a co-occurring depression diagnosis was associated with a child diagnosis of anxiety. In contrast, a parental depression diagnosis without a co-occurring anxiety diagnosis was not associated with a child diagnosis of anxiety.

The findings of Shanahan et al. (2008) and McClure et al. (2001) suggest that when controlling for co-occurring diagnoses, parental depressive and anxiety disorders have specific associations with child depressive and anxiety disorders, respectively. Since sub-threshold symptoms of disorders in parents can be as important for child outcome as symptoms above the diagnostic threshold (e.g., Foster et al. 2008), the study of both parental depressive and anxious symptoms when a parent has a history of depression warrants attention. As suggested in the Shanahan et al. (2008) and McClure et al. (2001) studies, these two clusters of symptoms may be differentially associated with child outcome; it may be that *only* parent–child depressive symptoms and *only* parent–child anxious symptoms are significantly associated. However, on a symptom level, there may be cross-cluster associations.

There is some evidence for cross-cluster links, particularly for parent depressive symptoms and child anxious symptoms (see Colletti et al. 2009, for a review); however, these relationships have emerged primarily when the alternate symptoms (e.g., depressive symptoms when examining anxious symptoms) are not controlled. Although disorders rather than symptoms were examined, a recent study by Biederman et al. (2006) points to the importance of controlling for alternate symptoms. These investigators did not find an association between parent depression and child anxiety when controlling for parent panic disorder. When comparing their findings to those of other investigators who found a significant association between parent depression and child anxiety but did not control for comorbid parent anxiety disorders, Biederman et al. (2006) noted that the comorbid anxiety disorders may have accounted for the difference. That is, the relation between parent depression and child anxiety in earlier studies may have resulted from comorbid parent anxiety disorders.

We conducted this study with children at risk for internalizing symptoms due to having a parent with a history of depression. This study had three purposes. First, we examined the relation between parent and child depressive symptoms while controlling for anxious symptoms of both dyadic members. Second, we examined the association between parent and child anxious symptoms while controlling for depressive symptoms of both dyadic members. Third, we examined the following cross-cluster links: parent depressive symptoms-child anxious symptoms, controlling for parent anxious and child depressive symptoms; and parent anxious symptoms-child depressive symptoms, controlling for parent depressive and child anxious symptoms. Based on the literature reviewed, we hypothesized significant positive same-cluster, but not cross-cluster, links between parents and children when controlling for symptoms from the alternate cluster.

In order to provide a rigorous evaluation of the proposed hypotheses, we utilized parent report of their own depressive and anxious symptoms and child report of their own depressive and anxious symptoms. This approach avoided the issue of shared reporter variance and eliminated the issue of parental depressive and anxious symptoms distorting parents' views of their children's behavior (Kroes et al. 2003).

Methods

Participants

We used baseline data of a randomized controlled trial designed to test a preventive intervention with the intent of preventing mental health problems among children of depressed parents (Compas et al. 2009). To be eligible for participation, families were required to have: (1) a target parent with a history of a MDD or dysthymia diagnosis during the lifetime of their oldest participating child; and (2) at least one child between the ages of 9 years and 15 years, 11 months. The criteria for exclusion were: (a) parental history of bipolar I disorder, schizophrenia, or schizoaffective disorder; (b) child history of autism spectrum disorders, mental retardation, bipolar I disorder, or schizophrenia; and (c) child current diagnosis of conduct disorder or alcohol/substance abuse or dependence. In addition, eligible families were deferred for later reassessment if: (a) a parent was currently suicidal; (b) a parent had current MDD with a Global Assessment of Functioning score $\leq$50; (c) a parent had current alcohol/substance abuse or dependence with a Global Assessment of Functioning $\leq$50; or (d) a participating child had current MDD. Deferred families were re-screened every 2 months until they screened eligible and could be invited to the next stage of recruitment.

The criteria for exclusion and deferral for parents were utilized to obtain a sample of parents with a history of depression who did not also have bipolar disorder and who were sufficiently high-functioning to ensure relatively consistent participation in the intervention. The criteria for exclusion and deferral of children were utilized to obtain a sample at-risk for, but not clinically diagnosed with, major depression or another severe mental illness.

Demographic data for the sample are presented in Table 1. The sample consisted of 104 parents (91 mothers, 13 fathers; *M* age = 41.5 years, *SD* = 8.1 years) with a history of MDD or dysthymia and their 131 children (64 female, 67 male; *M* age = 11.5 years, *SD* = 2.0 years). Families were recruited from Burlington, Vermont, and Nashville, Tennessee, and their surrounding areas. Recruitment strategies included mental health care provider and primary care physician referrals, local newspaper and radio advertisements, and flyers posted in the community. Participating target parents were largely Caucasian (79.8%), well educated (83.7% reported at least some college), and married or living with a partner (57.7%). Sixty-one parents had more than one eligible child; all eligible children participated. Seven participants were not included in the current sample due to missing data on one of the primary variables of interest.

Table 1 Descriptive statistics for demographic variables

	M	SD	%
Parent gender			87.5 (female)
Parent age	41.5	8.1	
Parent race			
Caucasian, non-hispanic			79.8
Black or African American			13.5
Asian			1.0
Latino or hispanic			1.9
American Indian or Alaska native			1.0
Mixed			2.9
Parent marital status			
Married or living with a partner			57.7
Widowed			1.0
Divorced			21.2
Separated			7.7
Never married			12.5
Parent education level			
Less than high school			6.7
High school or equivalent			9.6
Some college or technical school			37.5
College graduate			25.0
Graduate education			21.2
Household income level			
Under $5,000			6.7
$5,000–$9,999			5.8
$10,000–$14,999			1.9
$15,000–$24,999			11.5
$25,000–$39,000			18.3
$40,000–$59,999			18.3
$60,000–$89,999			19.2
$90,000–$179,999			14.4
Over $180,000			3.8
Child gender			48.9 (female)
Child age	11.5	2.0	

$N = 104$ parents and 131 children

Measures

Demographic data. The following demographic variables were collected from the parent: parent gender, age, ethnicity/race, marital status, education level, and annual family income, child gender, and child age.

Parent Diagnoses. The Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition (SCID-I/P; First et al. 2001) was used to assess parental psychopathology for purposes of study inclusion and exclusion. The SCID-I/P is a semi-structured interview designed to reliably measure Diagnostic and Statistical Manual-Fourth Edition (DSM-IV; APA, 1994) Axis I diagnoses. The Major Depressive Episode (MDE) section

of the Mood Episodes Module of the SCID-I/P was used to establish a current or previous diagnosis of MDE in the target parent. If the parent did not meet criteria for current or past MDE, then the Dysthymic Disorder section of the Mood Episodes Module was administered. The Manic Episode and Hypomanic Episode sections of the Mood Episodes Module, the Delusions and Hallucinations sections of the Psychotic Symptoms Module, and the Alcohol and Substance Abuse and Dependence sections of the Substance Use Disorders Module were also administered to assess for lifetime bipolar disorder, schizophrenia, and alcohol and substance use to determine exclusion and deferral criteria. Adequate reliability and validity have been demonstrated for the SCID-I/P (e.g., Williams et al. 1992).

Parental Depressive Symptoms. The Beck Depression Inventory-II (BDI-II; Beck et al. 1996) was used in the current study to assess current levels of parental depressive symptoms. The BDI-II is a well-established, widely-used 21-item self-report inventory designed to assess cognitive, affective, and physiological symptoms of depression. Participants were asked to indicate which of four statements reflecting varying degrees of symptom severity was representative of how they had been feeling over the past 2 weeks. Higher scores are indicative of more severe depressive symptoms, with scores ranging from 14 to 19, 20 to 28, and 29 to 63 indicating mild, moderate, and severe depression, respectively. The BDI-II has good convergent and discriminant validity (Osman et al. 2005), stable internal consistency ($\alpha = .90$; Beck et al. 1996), and high test–retest reliability over a one week period (i.e., $r = .93$; Beck et al. 1996). Internal consistency for the current sample was high ($\alpha = .92$).

Parental Anxious Symptoms. The Beck Anxiety Inventory (BAI; Beck and Steer 1996) was used in the current study to assess current levels of parental anxious symptoms. The BAI is a well-established, widely-used, 21-item self-report inventory designed to assess current anxiety symptoms in adults. Participants were asked to indicate which of four statements reflecting varying degrees of symptom severity was representative of how they were feeling over the past 2 weeks. Higher total scores are indicative of more severe anxious symptoms, with scores ranging from 8 to 15, 16 to 25, and 26 to 63 indicating mild, moderate, and severe anxiety, respectively. The BAI has excellent internal consistency ($\alpha = .92$) and correlates with other measures of anxiety (Beck and Steer 1996). Internal consistency for the current sample was high ($\alpha = .92$).

Child Diagnoses. The Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children—Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997) was used to assess child psychopathology for purposes of study inclusion and exclusion. The K-SADS-PL is a semi-structured diagnostic interview designed to assess child and adolescent current and past psychopathology. Parent and child are separately interviewed regarding the child's symptoms, after which summary scores are calculated. For the current study, the K-SADS-PL modules of interest for purposes of inclusion and exclusion were current conduct disorder and current alcohol and substance abuse and dependence, as these diagnoses excluded children. Current major depressive disorder (MDD) was also assessed via the K-SADS-PL as this diagnosis required families to be rescreened at a later time. The K-SADS-PL has demonstrated good test–retest reliability over a 1–5 week period (e.g., current MDD, $\kappa = .90$; current conduct disorder, $\kappa = .74$), and has well-supported concurrent validity with CBCL/6-18 broadband and syndrome scale scores (e.g., Kaufman et al. 1997).

Child Anxious Symptoms. The DSM-Oriented Anxiety Problems scale from the Youth Self-Report for Ages 11-18 (YSR/11-18; Achenbach and Rescorla 2001) was used in the current study as a self-report measure of current child anxious symptoms. Youths rated themselves on how true each item was for them within the last 6 months using a three-point response scale. Raw scores were used in all analyses to allow for maximum variance. Nine and 10 year old children completed the YSR to allow for complete data on all measures. The internal consistency for the YSR for this age group has been adequate with the current sample (see Compas et al. 2009). Internal consistency ($\alpha = .67$) and test–retest reliability ($r = .68$) estimates for the Anxiety Problems scale have also been shown to be acceptable. Internal consistency for the current sample was adequate ($\alpha = .66$).

Child Depressive Symptoms. The DSM-Oriented Affective Problems scale from the Youth Self-Report for Ages 11–18 (YSR/11-18; Achenbach and Rescorla 2001) was used in the current study as a self-report measure of current child depressive symptoms. Raw scores were used in all analyses. Internal consistency ($\alpha = .81$) and test–retest reliability ($r = .80$) estimates for the Affective Problems scale are generally good (Achenbach and Rescorla 2001). Internal consistency for the current sample was adequate ($\alpha = .79$).

Interviewer Training for Diagnoses

Interviewers underwent approximately 25 h of training prior to administering the SCID-I/P (First et al. 2001) and the K-SADS-PL (Kaufman et al. 1997). Training included the following steps: (1) participating in a detailed overview of both instruments followed by practice with a previously trained and reliable interviewer; (2) listening to and scoring a previously administered interview; (3) resolving

discrepancies from the original scoring of that interview with a master trainer; (4) completing a reliability check, achieved by administering an interview with the master trainer (SCID-I/P) or a community parent and child (K-SADS-PL); (5) resolving discrepancies through discussion between the interviewer and master trainer; and (6) participating in periodic mandatory interviewer refresher meetings to prevent interviewer drift. For example, reliability checks, conducted on a randomly selected subset of parent and child interviews, indicated 96% ($\kappa = .76$) and 93% ($\kappa = .71$) agreement for the K-SADS-PL and SCID—I/P, respectively, for MDD.

Procedures

All study procedures were approved by the IRBs at Vanderbilt University and the University of Vermont. All prospective target parents were initially screened via diagnostic telephone interview for symptoms of current or past depression, lifetime history of bipolar I and II, lifetime schizophrenia, and current alcohol and substance abuse and dependence. Diagnoses were made using the SCID-I/P (First et al. 2001). In addition, parents participating in the telephone screen were asked to report on the child's current depression (i.e., symptoms occurring within the past month), current conduct disorder, current alcohol and substance use, lifetime bipolar disorder, lifetime schizophrenia, and pervasive developmental disorder. Child and adolescent diagnoses were made using relevant sections of the K-SADS-PL (Kaufman et al. 1997).

Families meeting initial eligibility criteria based on the telephone screen were invited to participate in a more comprehensive in-person assessment at either the University of Vermont or Vanderbilt University. This assessment included administration of the SCID-I/P and K-SADS-PL interviews to make a final decision regarding eligibility. The baseline assessment also included a battery of questionnaires, including the BDI, BAI, and YSR, completed either during the assessment session or at home and returned within 1 week of the assessment session. All procedures were repeated for each participating child in a family, and parents and each child were compensated $40 each for their participation.

Results

Preliminary Analyses

Descriptive statistics were computed for all primary variables. The mean BDI-II score for target parents was 19.34 ($SD = 11.91$), indicating that parents, on average, reported depressive symptoms at the cutoff between mild and moderate levels. The mean BAI score for target parents was 11.90 ($SD = 10.60$), indicating that parents, on average, reported mild levels of anxiety symptoms. Although raw scores were used in analyses, mean *T*-scores for child anxious and depressive symptoms on the YSR/11–18 were calculated for normative comparison and were 55.4 and 56.6, respectively, both of which fall in the normative range.

Prior to conducting analyses involving demographic variables, three of the variables were modified. Parent education level and household income were significantly correlated ($r = .49$, $p < .01$), and thus were standardized and combined to form a measure of family socioeconomic status (SES) (Ensminger and Fothergill 2003). Parent marital status was transformed into a two-category variable reflecting whether or not a second parent or a partner lived in the home. Similarly, due to the low frequency of target parents identifying with a race/ethnicity other than Caucasian, parent race/ethnicity was transformed into a two-category variable indicating whether or not the target parent was Caucasian/non-Hispanic.

Zero-order correlations were computed to examine the relations between continuous demographic variables (i.e., target parent socioeconomic status and child age) and the criterion variables (i.e., child anxious symptoms and child depressive symptoms). Because of the nested nature of the data, correlations were computed after individual cases had been weighted. For example, when correlating target parent socioeconomic status and child anxiety scores in a family with two participating children, the value for socioeconomic status was weighted at one-half. One-way analyses of variance were computed to examine the relations between dichotomous demographic variables (i.e., target parent gender, race, and marital status, and child gender) and the criterion variables. Again, these analyses were conducted after individual cases had been weighted to account for the nested nature of the data.

Parental marital status and parental race were significantly ($p < .05$) related to child depressive symptoms such that being a single parent and not being Caucasian/non-Hispanic were both associated with more depressive symptoms. Parental marital status and family socioeconomic status were significantly ($p < .05$) related to child anxious symptoms such that being a single parent and having a lower socioeconomic status were each associated with more anxious symptoms. Thus, these demographic variables were controlled in the primary analyses.

Weighted correlations were conducted among the independent and dependent variables. Parental depressive symptoms and anxious symptoms ($r = .61$, $p < .01$) and child depressive symptoms and anxious symptoms ($r = .61$, $p < .01$) were related. Parental depressive symptoms were not related to child depressive ($r = .15$) or

anxious ($r = .14$) symptoms. Similarly, parental anxious symptoms were not related to child anxious ($r = .17$) or depressive ($r = .02$) symptoms.

Primary Analyses

Because multiple children from the same family were included in data analyses, Linear Mixed Models (LMM) analyses were used in SPSS to examine the relations between the primary variables of interest. LMM accounts for the correlation of data within families by assuming a compound symmetry covariance structure and using an iterative, or repeated measures, procedure to estimate parameters of the model. In this way, mixed model analyses account for the assumed correlations among children within the same family.

In a linear mixed-effects model, responses from a subject are considered to be the sum of fixed- and random-effects. Effects of the independent variables (e.g., parental depressive symptoms) on each dependent variable (e.g., child depressive symptoms) are considered fixed. Fixed effects, in other words, are represented by the regression coefficients. In contrast, effects associated with the sampling procedure (i.e., sampling data from multiple children within the same family) are considered random. Although the fixed-effects are typically of interest, it is necessary to account for the random-effects of the data, which represent random deviations for a given subject or cluster from the overall fixed effects (West et al. 2006).

Two sets of LMM analyses were conducted in which child anxious symptoms or child depressive symptoms were regressed on relevant demographic variables, the alternate type of child symptoms (e.g., child depressive symptoms when child anxious symptoms was the outcome measure), and parent anxious and depressive symptoms. By entering all variables simultaneously, the analyses allowed for same-cluster (e.g., parent depressive symptoms-child depressive symptoms) and cross-cluster (e.g., parent anxious symptoms-child depressive symptoms) relations to be examined while controlling for the parent alternate symptoms, child alternate symptoms, and relevant demographic variables. The variables initially were entered in two blocks (control variables and predictor variables); however, as only the final block with all variables was of interest, this block is presented in Table 2 (when child anxious symptoms served as the dependent variable) and Table 3 (when child depressive symptoms served as the dependent variable).

As reported in Table 2, after controlling for demographic variables and child depressive symptoms, higher levels of parental anxious, but not depressive, symptoms predicted higher levels of child anxious symptoms ($B = .04$, $p < .05$). As reported in Table 3, after controlling for demographic variables and child anxious symptoms, higher levels of parental depressive symptoms ($B = .08$, $p < .05$) and, unexpectedly, lower levels of parental anxious symptoms ($B = -.06$, $p < .05$) predicted higher levels of child depressive symptoms.

Table 2 Parental anxious and depressive symptoms predicting child anxious symptoms

	B	SE	*t*	*p*
Parent marital status	−.12	.40	.29	ns
Family SES	−13	.11	1.18	ns
Child depressive symptoms	.37	.04	9.28	<.01
Parent anxious symptoms	.04	.02	2.02	<.05
Parent depressive symptoms	.03	.02	1.41	ns

$N = 104$ families; beta weight and standard error rounded to two decimal places; Family SES = family socioeconomic status; Child depressive symptoms = child Affective Problems from the Youth Self-Report; Parent anxious symptoms = Beck Anxiety Inventory; Parent depressive symptoms = Beck Depression Inventory-II; Child anxious symptoms = child Anxiety Problems from the Youth Self-Report

Table 3 Parental depressive and anxious symptoms predicting child depressive symptoms

	B	SE	*t*	*p*
Parent race	1.10	.71	1.55	ns
Family SES	.66	.60	1.09	ns
Child anxious symptoms	1.06	.12	9.06	<.01
Parent depressive symptoms	.08	.04	2.20	<.05
Parent anxious symptoms	−.06	.03	2.14	<.05

$N = 104$ families; beta weight and standard error rounded to two decimal places; Family SES = family socioeconomic status; Child anxious symptoms = child Anxiety Problems from the Youth Self-Report; Parent depressive symptoms = Beck Depression Inventory-II; Parent Anxious Symptoms = Beck Anxiety Inventory; Child depressive symptoms = child Affective Problems from the Youth Self-Report

Discussion

We found support for significant links between parent and child depressive symptoms and parent–child anxious symptoms when controlling for the alternate symptom cluster. Interestingly, in correlation analyses, when the alternate parent and child symptoms were not controlled, the associations between parent and child anxious symptoms and parent–child depressive symptoms were not significantly related. In contrast to our findings, Shanahan et al. (2008) found *fewer* associations among risk factors and child outcomes when other risk factors and comorbid child outcomes were controlled. Although there are several potential explanations for the discrepant findings in the two

studies (e.g., examining symptoms vs. disorders, examining two specific parent and child symptoms vs. numerous risk factors and child outcomes, different assessment instruments being utilized), the important and consistent point across studies is that controlling for co-occurring symptoms or disorders in the study of risk factors and child outcomes does influence the conclusions reached. One likely explanation for the findings in the current study is that controlling parent and child symptoms that co-occur but are not part of a particular symptom cluster removes "noise" or random variance, which allows for a more accurate assessment of the relationship between the constructs being examined (e.g., parent and child depressive symptoms) (Cohen et al. 2003).

Significant positive relations across symptom clusters (e.g., parental depressive symptoms-child anxious symptoms) did not emerge. Our findings, taken in conjunction with the significant same-cluster associations, are consistent with a hypothesis of specific, rather than diffuse, associations between parental risk factors and child outcomes. That is, higher levels of either parental depressive or anxious symptoms are significantly related *only* to higher levels of the same type of child symptoms. These findings are generally congruent with those reported by Shanahan et al. (2008) and McClure et al. (2001), as parent depression was associated only with child depression and parent anxiety only with child anxiety in the former and latter studies, respectively. Of significance, both studies controlled for co-occurring symptoms in one (McClure et al.) or both (Shanahan et al.) members of the parent–child dyad. The findings also add to a growing interest in the research literature on specificity of child outcomes in the context of risk factors (e.g., McKee et al. 2008; McMahon et al. 2003; Shanahan et al. 2008) and provide data consistent with a specificity hypothesis.

It is interesting to note that neither parental depressive nor parental anxious symptoms were correlated with child depressive and anxious symptoms in preliminary analyses. However, once demographic variables and the alternate child *and* parent symptoms were taken into account, significant positive relations emerged for parent–child anxious symptoms and parent–child depressive symptoms. Not surprisingly, parental depressive and anxious symptoms were highly correlated as were child depressive and anxious symptoms. By removing these sources of shared variance, an uncontaminated relationship between the independent and dependent variables of interest could be examined. The finding underscores the importance of controlling for the alternate parent and child symptoms, as well as demographic variables associated with the dependent variable, when examining relationships between similar symptoms of parents and children. However, it is important to note that, once symptom overlap is controlled, the unique variance which remains in the parent–child depressive symptom relationship and the parent–child anxious symptom relationship is not consistent with traditional views of child anxiety and depression. As we will consider later, there is substantial overlap in symptoms of the two disorders (e.g., Watson 2005). Our findings highlight the unique symptoms of child anxiety and depression and how each set of symptoms are related to similar symptoms of parents.

An additional finding was that once parent depressive symptoms, child anxious symptoms, and demographic control variables were included in the regression model examining the relation of parental anxious symptoms and child depressive symptoms, an unexpected significant relationship emerged: parental anxious symptoms were *negatively* associated with child depressive symptoms. This finding was not only unexpected but counter-intuitive and inconsistent with prior research on cross-symptom relationships between parents and children (see Colletti et al. 2009). Offering an explanation seems premature until the finding is replicated taking into account both parental depressive symptoms and child anxious symptoms; nevertheless, the finding does suggest the importance of examining not only same cluster of symptoms but also cross-cluster relations when studying parents and children.

There are several limitations of the current study. First, the study was cross-sectional in nature, which prevents any conclusions about causality. The majority of the existing literature assumes that the primary direction of effect is from parent symptoms to child symptoms; however, child symptoms could also influence parental symptoms (e.g., parental anxious symptoms may increase in response to a child's anxiety about school attendance) and, most likely, the direction of effect is bidirectional rather than unidirectional. Second, all data were self-report. Third, as has been noted, the sample was constituted by parents with a history of clinical depression and children who were at risk for, but not clinically diagnosed with, depression; both of these sample characteristics may limit generality of the findings to other groups of parents and children. Replication of the findings in other clinical samples and in community samples will be an important next step. Fourth, the relations that emerged between parent and child symptoms are restricted by the child's age. For example, different associations may have emerged for parental depressive symptoms and child anxious symptoms for younger children (see Colletti et al. 2009). Finally, the emergence of significant relationships in regression analyses, where third variables are controlled, but not in zero-order correlations is consistent with a classical suppression effect (Cohen et al. 2003). There is a long history of caution about interpretation of suppression effects (e.g., Wiggins 1973) which should be noted with the current findings.

Several strengths of the study already have been noted but deserve mention again. First, having different reporters for parent and child variables eliminates the issue of common reporter variance. In addition, controlling for the alternate symptoms (e.g., anxious symptoms) of the parent *and* child when examining the association of one set of symptoms (e.g., depressive symptoms) in both parent and child represents an important step in the research process identifying links between parent and child psychological symptoms.

Our findings suggest that, in caregivers with a history of depression, there are links between parent and child depressive symptoms and between parent and child anxious symptoms. Moreover, these links appear to have a degree of specificity to them. However, the mechanisms that underlie the associations were not addressed in this study. Both genetic and environmental (e.g., parenting, modeling) factors may be implicated and warrant attention in future research.

Finally, ongoing debate about the classification of anxiety and depression deserves mention. Moffitt et al. (2007) have made numerous arguments for the close association of the two diagnoses, and Watson (2005) has gone a step further by advocating that we re-evaluate our current classification of these disorders in light of sufficient evidence for collapsing them into one group of disorders. This study, although focused exclusively on symptoms rather than diagnoses, provides evidence that is both congruent and incongruent with these authors' arguments. With regard to congruence, we found a close association of anxious and depressive symptoms, as the correlation between these two clusters of symptoms was .61 for both parents and children. However, with regard to incongruence, Moffitt et al. (2007) proposed that one type of support for classifying anxiety and depression together would be having similar risk factors. Our findings indicate that child anxious and depressive symptoms have different risk factors in the form of differential parental symptom clusters. This suggests that at a symptom level there is a utility to considering these two symptom clusters separately, a conclusion not dissimilar to recent findings of Ebesutani et al. and The Research Network on Youth Mental Health (2010).

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