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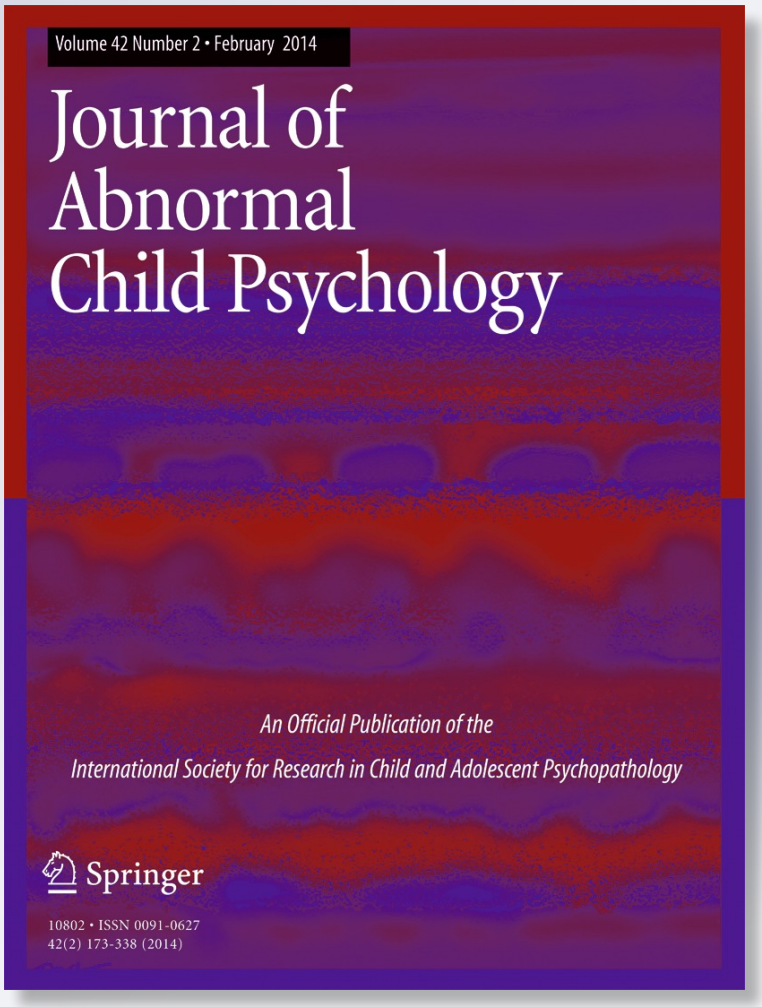

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Relation Between Parent Psychiatric Symptoms and Youth Problems: Moderation through Family Structure and Youth Gender

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Abstract Links between parents' psychiatric symptoms and their children's behavioral and emotional problems have been widely documented in previous research, and the search for moderators of this association has begun. However, family structure (single versus dual-parent households) has received little attention as a potential moderator, despite indirect evidence that risk may be elevated in single-parent homes. Two other candidate moderators—youth gender and age—have been tested directly, but with inconsistent findings across studies, perhaps in part because studies have differed in whether they used youth clinical samples and in which informants (parents vs. youths) reported on youth problems. In the present study, we examined these three candidate moderators using a sample of exclusively clinic-referred youths (N=333, 34 % girls, aged 7–14,) and assessing youth problems through both parent- and youth-reports. Both family structure and youth gender emerged as robust moderators across parent and youth informants. Parent symptoms were associated with youth internalizing and externalizing problems in single-parent but not dual-parent homes; and parent symptoms were associated with youth internalizing problems among boys, but not girls. The moderator findings suggest that the risks associated with parent psychopathology may not be uniform but may depend, in part, on family structure and youth gender.

Keywords Parent psychopathology · Family structure · Gender · Moderation · Youth internalizing · Youth externalizing

Links between parental mental illness and adverse psychosocial outcomes for youth have been documented in numerous studies and reviews (Goodman 2007; Hammen 2009). Prospective, longitudinal studies have identified parents' psychiatric symptoms as risk factors for emotional and behavioral difficulties in youths (Anderson and Hammen 1993; Burstein et al. 2010; Weissman et al. 1997; Weissman et al. 2006; Williamson et al. 2004).[1] Because the association between parent symptoms and youth problems is not evident in all families, investigators have begun the search for factors that moderate the association (Burt et al. 2005; Suveg et al. 2011). In the present study, we sought to extend and refine that search by examining a promising candidate moderator that has received little attention to date, revisiting two previously studied candidate moderators that have generated conflicting findings, and using a sample and measurement model intended to enhance clinical relevance and the identification of robust moderators.

Across studies examining moderators of links between parent symptoms and youth problems, one factor that has received little direct attention is family structure (single- versus dual-parent households). This is surprising, as there are reasons to believe that single-parent households may be especially susceptible. Compared with youths raised by two parents in the same household, youths raised by a single parent tend to have more internalizing and externalizing

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[1] Several studies suggest that links between parent symptoms and youth problems are complex and bidirectional (e.g. Kim et al. 2009). Identification of causal pathways is beyond the scope of the present paper, which focuses instead on factors that moderate the strength of association. Identifying such moderators may help inform later investigations into causal patterns.

problems and suffer more social and academic impairment (Martins and Gaffan 2000; Olson et al. 2002). Single parents also tend to experience higher rates of stress and symptomatology than do parents in intact families, and their children may have less exposure to a healthy second caregiver (Connell and Goodman 2002). It is possible that these differences might be attributable to income, since research suggests that the proportion of youths living in poverty is considerably lower for youths in dual-parent families (11 %) than for youths in single-parent families (40 % for youths living with a divorced parent; 65 % for youths living with a never-married parent) (Dawson 1991). However, these differences tend to persist after accounting for socioeconomic proxies (Brooks-Gunn and Duncan 1997; Dodge et al. 1994). Further, while some research suggests that associations between youth problems and family structure are contingent upon ethnicity (Siegel et al. 1998), other studies have found that minority status does not consistently moderate this relationship (Wight et al. 2005). Further, single parents with significant psychiatric symptoms might have a limited capacity to monitor their youths' emotional and behavioral well-being. Due to an increased personal load of occupational and caregiving obligations, these parents might be less available to help their youths, and to notice and ameliorate their symptoms, in times of crisis. Accordingly, it is possible that youths of highly symptomatic parents living in single-parent households—compared with those in dual-parent households—might show relatively higher problem levels.

To our knowledge, previous studies have not systematically explored family structure as a moderator of associations between parent symptoms and youth problems. In their meta-analysis on moderators of this relation, Connell and Goodman (2002) found that samples composed of single-parent families yielded effects that were not significantly different from those samples composed mostly of dual-parent families. However, it appears that the heterogeneity of these samples may have obscured moderating effects: very few studies are based on predominantly single-parent families, and only some of the studies in the authors' meta-analysis controlled for potential confounds, such as ethnicity and family income. In fact, Connell and Goodman (2002) noted the need for studies systematically addressing differences between single- and dual-parent families in the strength of association between parent and youth symptomatology. Thus, a primary purpose of the present study was to formally test whether family structure moderates the association between parent symptoms and youth problems, controlling for effects of potential confounding variables (income, youth ethnicity).[2] Our test included two features that are not uniformly evident in the literature on parent-youth symptom associations, but which do appear to strengthen tests of moderation in this field. First, to make the test relevant to clinical levels of youth problem behavior, we used an exclusively clinical sample of youths. Focusing on youths experiencing problems warranting clinical referral might render findings directly applicable to treating and preventing problems in youths experiencing distress. Second, to reduce the risk that findings might reflect idiosyncrasies of one particular type of informant (e.g., parents alone, or youths alone), we tested for moderation using both parent- and youth-informant data. Many prior studies testing such moderators have relied on either parent *or* youth informants for youth symptoms, with some findings varying depending on informant type (Connell and Goodman 2002). Specifically, some studies relying on parent reports of youth problems have yielded larger effect sizes than those relying on youth report of youth problems. However, the size of these differences varied considerably across studies, and improved understandings of possible informant biases are needed.

Using these same two design features, we also tested two candidate moderators that *have* been explored in previous research, but about which existing evidence remains quite unclear: youth gender and youth age. On the gender front, researchers have hypothesized that boys may be more susceptible to family risk factors than girls during early and middle childhood, whereas girls may exhibit greater vulnerability during later childhood and adolescence (e.g., Cummings and Davies 1994a; Hops 1992, 1995; Petersen 1988). In support of this hypothesis, stronger links between parental and youth symptoms have been demonstrated for community samples of girls, compared with boys, during both later childhood (Goodman et al. 2011) and adolescence (Fergusson et al. 1995; Ge et al. 1995; Davies and Windle 1997; Cortes et al. 2006; Lewis et al. 2011). Although these findings suggest that girls may be more influenced by parent symptoms than boys, the findings are derived almost exclusively from non-clinical youth samples, and from designs that did not formally tests youth gender as a moderator. In their meta-analysis of relations between parental symptoms and youth problems, Connell and Goodman (2002) found negligible differences between effect sizes of parent symptoms on problems in boys and girls; this may have resulted from the fact that most studies relating symptoms in parents and youths have either focused exclusively on behavior problems in boys or grouped boys and girls together in analyses, limiting reliable assessments of gender differences (Connell and Goodman 2002). The present study was designed to help fill gaps in the literature, both by formally testing youth gender as a possible moderator and by examining youth internalizing and externalizing problems separately. Due to the mixed findings of previous research, the non-clinical samples employed, and the fact that most studies did not formally test moderation, there

[2] Notably, caregivers participating in this study were 90.7 % female. This gender imbalance may render findings more relevant to relations between mother symptoms and youth problems than between father symptoms and youth problems. However, for brevity, we will refer to participating caregivers as "parents" in this study.

did not appear to be a strong basis for specific predictions; but a primary research question was whether (and if so, in what direction) youth gender moderated the association between parent symptoms and youth problems.

A second potential moderator that has received attention in prior research is youth age, or developmental period. As outlined by Rothbaum and Weisz (1994), literature has posited two contrasting hypotheses regarding links between parent and youth symptoms. One hypothesis is that adolescents are more susceptible than children to the influence of parental symptoms. The stress of living with a symptomatic parent may hamper resolution of challenges faced by adolescents (e.g., independence from parents, establishment of intimate relationships), increasing risk for youth problems (Davies and Windle 1997). Indeed, some evidence has found links between parental symptoms and adolescent emotional and behavioral disturbances (Fergusson et al. 1995; Hammen et al. 2012; Hops 1992). A contrasting hypothesis is that parental symptoms pose a more significant risk for preadolescent youths. Indeed, some research has found that the strength of association between parent and youth symptoms is negatively correlated with youth age, perhaps because parents exert their greatest influence when their children are very young (Connell and Goodman 2002). Of course, differences in strength of association at different youth age levels could result from quite different models, including those positing youth effects on parents, and those positing third factors (e.g., shared genetic influence). An important step in evaluating various models related to age is determining whether age is in fact a robust moderator of the association between parent symptoms and youth problems; this is another objective of the present study.

To summarize, we conducted what is apparently the first direct test of family structure as a moderator and re-examined whether youth gender and youth age are in fact moderators. To provide clinically relevant evidence, we sampled only youths whose problems were significant enough to have generated clinic referral. And to reduce the risk that single-informant idiosyncrasies or bias regarding youth problems might influence findings, we included both youth and parent reports of youth problems, testing whether moderation effects were robust across both informants.

Method

Procedure

Data were from assessments used to determine eligibility for a treatment study (Weisz et al. 2012). The sample comprised youths who had sought outpatient care and had referral problems involving internalizing and/or externalizing problems. All research procedures for this trial were approved by the IRBs of Judge Baker Children's Center (an affiliate of Harvard Medical School) and the University of Hawaii at Manoa, and all participating parents and youths signed informed consent/assent documents approved by these institutional review boards.

The sample included 333 youths aged 7–14 (mean age=10.21 years; SD=1.75 years); 66 % were boys, 46.5 % Caucasian, 29.1 % multiethnic, 9.9 % African American, 7.5 % Latino or Latina, 3.3 % Asian, and 3.0 % identified as "other." There were significantly more Caucasian, African American, and Latino or Latina youths at Boston versus Hawaii sites, and there were significantly more Asian and Mixed youths at Hawaii versus Boston sites, all $p<0.05$. There were significantly more boys at Hawaii versus Boston sites, $t(330)=-2.60$, $p=0.010$. Annual family income was less than $40,000 for 54.9 % of the sample, $40,000–$59,000 for 13.5 % of the sample, $60,000–$79,000 for 11.1 % of the sample, and more than $80,000 for 15.6 % of the sample. There were no differences in family income in Hawaii versus Boston sites. In each family, the parent taking primary responsibility for the youth's clinical care (i.e., the "primary" parent) completed baseline assessments. Of these parents, 90.7 % were female. Specifically, 71.56 % were biological mothers; 6.56 %, biological fathers; 8.12 %, grandmothers; 0.31 %, grandfathers; 4.3 %, adoptive mothers; 0.31 %, adoptive fathers; 3.13 %, stepmothers; 0.31 %, stepfathers; 2.8 %, "other" female caretakers; and 0.31 %, "other" male caretakers. There were no significant family structure differences at Boston versus Hawaii sites.

Across parent informants, 52 % shared parenting responsibilities with another adult, and 48 % were single parents. In dual-parent households, the "non-primary" adult caregivers were 60.0 % biological parents, 16.3 % grandparents, 11.0 % stepparents, 3.7 % adoptive parents, and 14 % "other" (which included the primary parents' partners; cousins; uncles; and aunts). Independent-samples t-tests showed no significant differences in family structure or family income by youth gender or youth ethnicity. However, parents in single-parent families reported significantly lower incomes than parents in dual-parent families, $t(330)=-5.22$, $p<0.001$. Specifically, 13.9 % of dual-parent families had incomes of less than $19,000 per year, compared with 33.5 % of single-parent families.

Measures

Brief Symptom Inventory (Derogatis 1993) Referring parents completed this 53-item, self-report measure of adult symptomatology that generates scores on nine dimensions (e.g., Depression, Anxiety, Hostility) and a General Severity Index (GSI). Due to significant intercorrelations among BSI symptom subscales, research has suggested that the measure may be best used as a general indicator of symptomatology

(Boulet and Boss 1991); thus, we used the GSI to assess parental symptomatology. Test-retest reliabilities for the BSI have ranged from 0.68 to 0.91, and internal consistency alpha coefficients 0.71 to 0.85 (Derogatis and Melisaratos 1983). In the present study, alpha was 0.97.

Youth Self Report Form (YSR; Achenbach 1991) Youths also completed this 118-item measure assessing self-reported behavioral and emotional problems. In this study, the broadband Internalizing and Externalizing syndrome scales were used, which use items from the Anxious/Depressed, Social Withdrawal, and Somatic Complaints narrow-band scales, and the Delinquency and Aggression narrow-band scales, respectively. Achenbach (1991) has shown that the YSR and its subscales are reliable and valid measures of problematic behaviors among clinic-referred and nonreferred youths, and further research documents that the measure is reliable and valid for youths as young as 7 years old (Ebesutani et al. 2011; Yeh and Weisz 2001). All YSR syndrome scales have displayed excellent psychometric properties (Achenbach and Rescorla 2001). In the present study, alpha was 0.90 for the internalizing syndrome scale and 0.91 for the externalizing syndrome scale.

Child Behavior Checklist (CBCL; Achenbach and Rescorla 2001) Referring parents completed this measure, assessing the extent to which their youth has displayed certain symptoms within the past 6 months. The broadband CBCL Internalizing and Externalizing syndrome scales were used in the present study. The syndrome scales have demonstrated high test-retest reliability, intraclass correlations, content validity, and adequate inter-rater agreement, as evaluated with youths ranging from age 4 to 18 (Achenbach 1991). In this study, alphas were 0.89 and 0.91 for the Internalizing and Externalizing syndrome scales.

Results

Parent and Youth Symptom Correlations and T-Tests

Means and standard deviations for parent symptoms and CBCL and YSR scores are presented for the total sample in Table 1. The table shows positive and significant correlations between CBCL internalizing and externalizing, between YSR internalizing and externalizing scores, and (to a lesser degree) between GSI and CBCL, consistent with documented links between parent and youth symptom levels. However, YSR scores did not correlate significantly with GSI. Independent-samples t-tests revealed no significant differences in main study variables by gender, site (Hawaii versus Boston), family structure, or ethnicity. Younger youths reported higher YSR externalizing scores than older youths, $t(330)=2.10$, $p=0.04$, with no other significant differences in study variables by youth age. Parents in lower-income households reported higher GSI scores, $t(320)=-4.42$, $p<0.01$, and higher CBCL externalizing scores, $t(329)=-2.86$, $p<0.01$. Tests revealed no other significant differences on study variables by family income.

Moderation Analysis

Analyses to determine whether youth gender, youth age, and family structure moderated relations between parent symptoms and youth internalizing and externalizing problems followed procedures recommended by Hayes and Matthes (2009). Their SPSS macro (http://www.comm.ohiostate.edu/ahayes/SPSS%20programs/modprobe.htm) yields the significance of the change in R^2 produced by interactions between independent (Parent Symptoms) and moderator variables (Family Structure, Youth Gender, and Youth Age, respectively). This value serves as an index of whether each interaction significantly predicted relations between parent symptoms and youth problems (Howell 2002). The macro also allows examination of the link between parent symptoms and youth problems at low (−1 SD below the mean, or 0 for dichotomous moderators) and high (+1 SD above the mean, or 1 for dichotomous moderators) levels of the candidate moderator, thus replacing the Baron and Kenny (1986) approach. For all candidate moderators, we conducted parallel models for youth and parent reports of internalizing and externalizing problems. Results are described separately by informant type below.

Family Structure [3] The Parent Symptoms X Family Structure interaction produced a significant change in R^2 for youth-reported youth internalizing, $F(3, 313)=8.41$, $R^2=0.03$, $p<0.01$, and externalizing problems, $F(3, 313)=10.25$, $R^2=0.03$, $p<0.01$. Figure 1 illustrates the simple effects (regression lines) for youth-reported internalizing and externalizing problems, respectively. In single parent homes, youths whose parents had higher symptom levels reported higher levels of both internalizing, $t(313)=2.98$, $p<0.01$, and externalizing problems, $t(313)=3.09$, $p<0.01$, than youths whose

[3] To address potentially confounding effects of family income and ethnicity, we also tested the models presented including these factors as covariates. Income was measured in eight brackets of $20,000, from $0 to $140,000 or greater. Ethnicity was dummy-coded into seven separate dummy variables, each representing one ethnic category. Findings for all models (both parent- and youth- report) remained significant even after including income and ethnicity as a covariate (for youth-reported internalizing problems, $F(11, 311)=7.95$, $R^2=0.03$, $p<0.01$; for youth-reported externalizing problems, $F(11, 311)=10.07$, $R^2=0.03$, $p<0.01$; for parent-reported internalizing problems, $F(11,311)=5.84$, $R^2=0.02$, $p<0.05$; for parent-reported internalizing problems, $F(11, 311)=8.58$, $R^2=0.05$, $p<0.01$).

Table 1 Descriptive statistics and zero-order correlations of parent and youth symptom variables

	M	*SD*	(2)	(3)	(4)	(5)
(1) Parent symptoms	0.00	34.12	0.32**	0.32**	0.06	0.05
(2) CBCL internalizing	16.07	9.57	–	0.39**	0.15**	−0.02
(3) CBCL externalizing	16.44	10.50	–	–	0.02	0.37**
(4) YSR internalizing	13.98	9.35	–	–	–	0.57**
(5) YSR externalizing	10.38	8.38	–	–	–	–

As *Parent Symptoms* was centered prior to analyses, mean is 0.00 for this variable

*$p<0.01$

**$p<0.001$

parents had lower symptom levels. However, in dual-parent homes, level of parent symptoms was not significantly related to youth-reported internalizing or externalizing problems. To probe this interaction further, we performed independent samples t-tests on youth-reported symptom levels in youths with higher-symptom (+1 SD from mean or greater) and lower-symptom (-1 SD from mean or less) parents, respectively. Among youths with lower-symptom parents, those in single parent homes actually reported significantly lower levels of externalizing problems, $t(55)=-2.20$, $p=0.03$, than youths in dual parent homes, and there was no significant difference in youth-reported internalizing problems between single parent and dual parent homes. Among youths with higher-symptom parents, youths in single-parent homes reported significantly more internalizing problems than youths in dual-parent homes, $t(46)=2.18$, $p=0.03$. There was no difference between single parent and dual parent homes in the level of youth-reported externalizing problems.

The Parent Symptoms X Family Structure interaction also produced a significant change in R^2 for parent-reported youth internalizing, $F(3, 321)=5.94$, $R^2=0.02$, $p=0.02$, and externalizing problems, $F(3, 321)=5.98$, $R^2=0.04$, $p=0.01$. Figure 2 illustrates the simple effects for parent-reported internalizing and externalizing problems, respectively. In single parent homes, youths whose parents had higher symptom levels reported higher levels of both internalizing, $t(321)=4.41$, $p<0.0001$, and externalizing problems, $t(321)=3.94$, $p<0.001$, than youths whose parents had lower symptom levels. However, in dual-parent homes, level of parent symptoms was not significantly related to youth-reported internalizing or externalizing problems. To probe this interaction further, we performed independent samples t-tests on youth-reported symptom levels in youths with higher-symptom (+1 SD from mean or greater) and lower-symptom (−1 SD from mean or less) parents, respectively. Among youths with lower-symptom parents, parents in single parent homes reported marginally lower levels of youth externalizing problems, $t(55)=-2.04$, $p=0.05$, than parents in dual parent homes, and there was no significant difference in parent-reported internalizing problems between single parent and

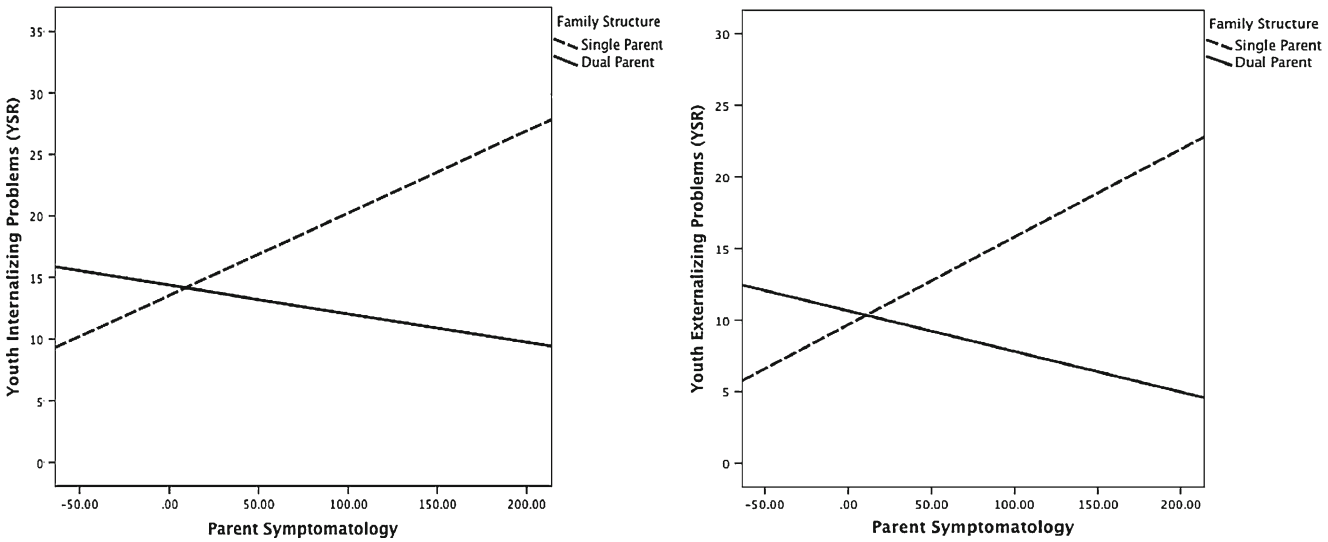

Fig. 1 Mean youth-reported youth internalizing (*left*) and externalizing problems (*right*) for youths in single and dual-parent homes as a function of parent symptoms

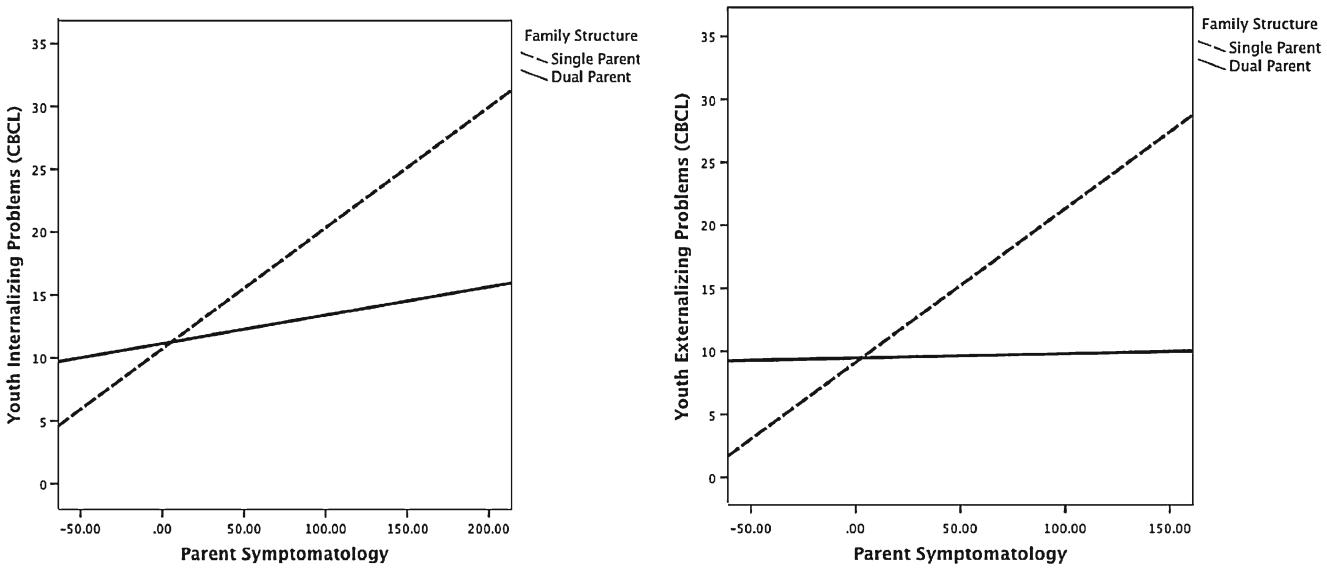

Fig. 2 Mean parent-reported internalizing (*left*) and externalizing problems (*right*) for youths in single- and dual-parent homes as a function of parent symptomatology

dual parent homes. Among youths with higher-symptom parents, parents in single-parent homes reported significantly more youth internalizing problems than parents in dual-parent homes, $t(44)=2.18$, $p=0.03$. There was no significant difference between single parent and dual parent homes in the level of parent-reported externalizing problems.

Youth Gender The Parent Symptoms X Youth Gender interaction produced a significant change in R^2 for youth-reported internalizing problems, $F(3, 315)=0.20$, $R^2=0.03$, $p<0.01$ but not youth-reported externalizing problems. Figure 3 shows the nature of the internalizing effect, using the method of simple slopes: boys with more symptomatic parents (+1 SD above mean parental GSI) showed significantly more internalizing problems than boys with less symptomatic parents (−1 SD below mean), $t(315)=2.41$, $p=0.02$, but parental symptoms did not predict internalizing problem levels in girls. To explore this effect further, we performed independent-samples t-tests comparing symptom levels in girls and boys with higher-symptom (+1 SD from mean or greater) and lower-symptom (−1 SD from mean or less) parents, respectively. Among youths with low-symptom parents, boys reported significantly fewer internalizing symptoms than girls, $t(55)=-2.15$, $p=0.04$, and there were no

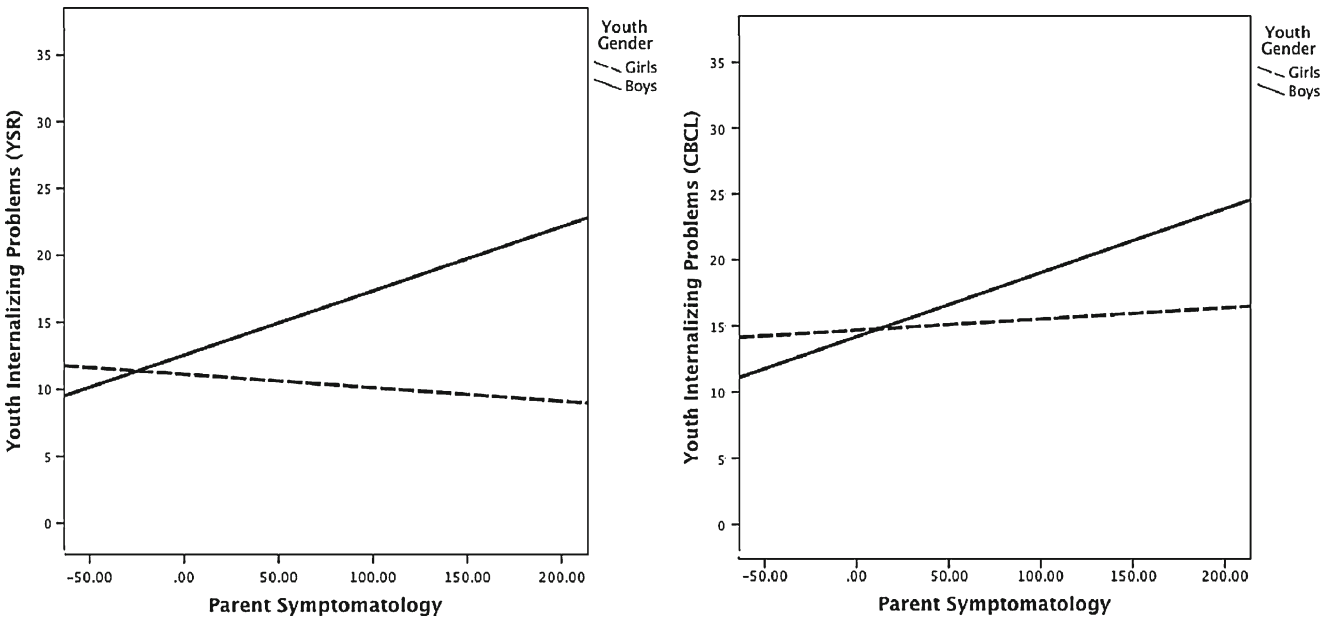

Fig. 3 Mean youth internalizing problems (youth-report, *left*; parent-report, *right*) for youths by gender as a function of parent symptomatology

significant differences in internalizing symptoms between boys and girls with high-symptom parents.

The Parent Symptoms X Youth Gender interaction also produced a significant change in R^2 parent-reported youth internalizing problems, $F(3,315)=4.62$, $R^2=0.01$, $p=0.03$, but not externalizing problems. Figure 3 shows the nature of the internalizing effect: boys with more symptomatic parents (+1 SD above mean parental GSI) showed significantly more internalizing problems than boys with less symptomatic parents (−1 SD below mean), $t(315)=4.32$, $p<0.001$; but parental symptoms did not predict internalizing problem levels in girls. To explore this effect further, we performed independent-samples t-tests comparing symptom levels in girls and boys with higher-symptom (+1 SD from mean or greater) and lower-symptom (−1 SD from mean or less) parents, respectively. These analyses revealed no significant differences in parent-reported internalizing problems for boys and girls with high- or low-symptom parents.[4]

Youth Age With age treated as a continuous variable, the Parent Symptoms X Youth Age interaction did not significantly predict youth- or parent-reported internalizing or externalizing problems in youths. Thus, moderating effects of youth age on relations between parent symptoms and youth problems were not supported in the present study.

Discussion

The current study was designed to advance prior work on relations between parent psychiatric symptoms and youth problems by formally testing family structure as a moderator, focusing on an exclusively clinical sample of youths, and using both parent- and youth-informant data on youth problems. We also tested moderating effects of youth gender and youth age, two candidate moderators about which prior findings have been quite mixed. Our findings offer support for family structure as a robust cross-informant moderator for both internalizing and externalizing problems, and youth gender as weaker moderator and only for internalizing problems, but do not show age to be a moderator of either internalizing or externalizing problems.

We hypothesized that family structure would moderate relations between parent and youth symptoms, such that youths in single-parent households would be more adversely affected by parent symptoms than youths in dual-parent households. In support of this hypothesis, we found that parents' symptom levels were significantly associated with both internalizing and externalizing problems in youths living in single- but not dual-parent homes. These effects remained robust across the separate parent and youth informant measures. However, a second, unanticipated finding also emerged: among youths with low-symptom parents, youths in single-parent homes showed *fewer* externalizing problems than youths in dual-parent homes.

These results both support and qualify prior findings indicating that youths with single parents may be at greater risk for emotional and behavioral symptoms (Martins and Gaffan 2000; Olson et al. 2002). That is, youths with single parents appear to be at greater risk for adverse outcomes *only* when parent symptoms are at a high level. In contrast, youths with single parents may be *protected* from some adverse outcomes (in this case, externalizing problems), compared with youth in dual-parent households, when their parent reports low symptoms. It is possible, for example, that youths with a low-symptom single parent may align themselves especially closely with the resilient, healthy adult role model. Youths in dual-parent households may experience such alignment to a lesser degree, simply because they are influenced by both parents in their household. Conversely, youths with a high-symptom, single parent may be at greater risk because they cannot align themselves with a healthier caregiver, while youths in dual-parent homes have an alternate parental role model readily available. Alternatively, single parents who are psychologically healthy might make efforts to be more mindful of their youths' well-being than adults with a parenting partner. Because such parents may be aware of risks associated with single parenting, they may more closely monitor youths' activities as is appropriate at given ages, or make special efforts to remain involved in such activities. To our knowledge, ours is the first study to directly test moderating effects of family structure on links between parent symptoms and youth problems. Thus, our findings help clarify the conditions under which parental symptomatology is, and is not, a risk factor for child internalizing and externalizing problems.

Results also supported youth gender as a modest but significant moderator of links between parent symptoms and youth problems. Specifically, we found that parents'

[4] While we lacked sufficient statistical power to assess moderating effects of parent gender, it remains theoretically plausible that moderating effects of family structure and youth gender might differ for a mixed-gender sample of caregivers versus a sample of exclusively female caregivers. To test this possibility within our sample, we re-tested all significant models excluding the 9.3 % of caregivers who were male. All significant findings for all models (both parent- and youth-report) identified in our initial analyses with the full sample remained significant, and no additional effects emerged as significant, after excluding male caregivers from analyses. For Family Structure x Parent Symptoms models: youth-reported internalizing problems, $F(3, 304)=5.36$, $R^2=0.02$, $p=0.02$; youth-reported externalizing problems, $F(3, 304)=6.58$, $R^2=0.02$, $p=0.01$; parent-reported internalizing problems, $F(3,304)=6.73$, $R^2=0.07$, $p<0.01$; parent-reported internalizing problems, $F(3, 304)=11.56$, $R^2=0.11$, $p<0.01$. For Youth Gender x Parent Symptoms models: youth-reported internalizing problems, $F(3, 301)=7.28$, $R^2=0.02$, $p<01$; youth-reported externalizing problems, $F(3, 301)=5.34$, $R^2=0.02$, $p=02$.

symptom levels were significantly related to internalizing problems in boys but not girls. This effect was significant using parent reports of youth symptoms and marginally significant using youth reports of their own symptoms. There are several potential explanations for these results. First, the mean age of this sample was 10.21 years, and the majority of youth had yet to reach adolescence. Some evidence has suggested that boys might be more susceptible to family-related risk factors than girls during childhood, with this pattern reversing during late childhood and early adolescence (Cummings and Davies 1994b; Hops 1992, 1995; Petersen 1988). Alternatively, some research has suggested that boys might require greater regulatory control during early childhood in order to develop adaptive emotion regulation skills: Weinberg et al. (1999) found that boys with a withdrawn, less expressive mother experienced more affective dysregulation than girls as early as infancy. Relatedly, in the present study, high-symptom parents might have lacked emotional resources to consistently regulate their children's behavior, placing boys at particular risk for internalizing problems during their pre-adolescent years. In contrast, girls with a high-symptom parent might show higher symptoms than boys with a high-symptom parent in a middle- or late-adolescent sample. Factors other than parent symptoms might also be more relevant to girls' internalizing problems, such as peer-related concerns or cognitive style. Future research exploring these possibilities is warranted.

It is notable that although youth gender moderated parent-youth symptom links for internalizing symptoms in youths, no such pattern emerged for externalizing symptoms. It is possible that parental struggles with psychiatric symptoms are especially likely to be associated with worry, sadness, and other forms of youth internalizing distress than with conduct problems and other forms of disruptive behavior. If so, this might be more readily detected in clinical samples, like the one used here, than in the community samples that have often been used in prior research on this topic. Further studies with clinical samples will be needed to assess how robust this particular finding is.

We found no evidence for a moderating effect of youth age on links between parent and youth symptoms. The lack of moderation might have resulted from the rather restricted age range in the present sample: 85.70 % of youths were between 8 and 12 years of age. Future studies might explore moderating effects of youth age in samples with a broader age range.

Our findings have several limitations. First, because data were collected at a single time-point, directionality of effects cannot be established. Although findings of this study are suggestive of possible moderating pathways, it is important to note that parents and youths influence one another in a bi-directional fashion, and other factors (e.g., shared genetic effects) may also influence links between the proposed moderators and outcome variables. Nonetheless, our findings are a useful step toward understanding moderators of links between parent symptoms and youth internalizing and externalizing problems—particularly among clinically-referred youths. Future longitudinal studies on youth gender, age, family structure, and other possible moderators may sharpen the picture of how parent symptoms influence and are influenced by youth problems over time.

Second, given that parents were the only informants of their psychological symptoms, the measurement of these variables is subject to concerns about self-report. Similarly, methods used to assess youth symptoms also relied on reports, both of parents and of youth. However, our use of youth *and* parent reports to measure youth internalizing and externalizing problems reduced the possibility of a shared method effect and decreased the likelihood that parents' own symptomatology influenced estimates of youth symptoms. In future studies, supplementing self-report measures with observational, diagnostic, or additional multi-informant data could help strengthen measurement of both parent and youth symptomatology.

Third, because the BSI has been shown to be most useful as a measure of global adult symptomatology (Boulet and Boss 1991), we did not assess relations of parent symptom clusters (e.g. internalizing, externalizing) to symptoms in offspring. Future studies might employ more comprehensive measures of parent symptomatology to parse specific patterns, and moderators of patterns, underlying the parent symptom-youth problem association.

Finally, it is notable that female parents or caretakers comprised over 90 % of the reporting parent sample. Thus, we did not have sufficient statistical power to explore potential moderating effects of parent gender. However, paternal participation is an ongoing issue in clinical research with youths and families. Future studies that include parents of both genders, and conduct separate analyses by parent gender, will help disambiguate their potential moderating effects on links between parent and youth mental health.

Despite these limitations, the present study has significant strengths and carries implications for our understanding of the association between parent symptoms and youth problems. First, our study appears to be the first to formally test family structure as a moderator of parent-youth symptom relations, and single- vs. dual-parent household emerged as a particularly potent moderator. Second, because most moderator studies in this field have relied on single informants to assess youth, they have raised concern about possible effects of informant bias; our use of both parent and youth problem reports helps address this concern and sheds light on the robustness of moderation effects. Finally, our use of an exclusively clinically-referred sample lends clinical relevance to our findings. Taken together, the findings suggest that parents' psychiatric symptoms are indeed associated

with youth problems, but only for a subset of youths defined in part by family structure and youth gender. Future research will be needed to assess the holding power of these findings across sample variations, and to explore competing causal explanations.

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