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Authors

Schleider, Jessica L
Ginsburg, Golda S
Keeton, Courtney P
[et al.](#)

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Parental psychopathology and treatment outcome for anxious youth: Roles of family functioning and caregiver strain

Jessica L. Schleider^{a,*}, Golda S. Ginsburg^b, Courtney P. Keeton^b, John R. Weisz^a, Boris Birmaher^c, Phillip C. Kendall^d, John Piacentini^e, Joel Sherrill^f, and John T. Walkup^g

^aDepartment of Psychology, Harvard University, 33 Kirkland Street, Cambridge, MA 02138, USA

^cPsychiatry and Behavioral Sciences, The Johns Hopkins University School of Medicine

^cDepartment of Psychiatry, University of Pittsburgh, and Western Psychiatric Institute & Clinic, University of Pittsburgh Medical Center

^dDepartment of Psychology, Temple University

^eDivision of Child and Adolescent Psychiatry, UCLA Semel Institute for Neuroscience and Human Behavior

^fDivision of Services and Intervention Research, National Institute of Mental Health

^gDepartment of Psychiatry, Weill Cornell Medical College, and Division of Child and Adolescent Psychiatry, New York-Presbyterian Hospital

Abstract

Objective—Research has examined the effects of parental psychopathology, family functioning, and caregiver strain on treatment response in anxious youths. Although these variables have shown individual links to youth treatment response, theoretical models for their combined effects remain unexplored. This study tested the hypothesis that improvements in family functioning and reductions in caregiver strain explained the effects of parental psychopathology on youth treatment outcome in an anxiety treatment trial.

Method—A multiple mediation technique was used to test the proposed model across independent evaluator (IE), parent, and youth informants in 488 youths, aged 7–17 years (50% female; mean age 10.7) meeting DSM-IV-TR criteria for social phobia, separation anxiety, and/or generalized anxiety disorder. Youths were randomized to receive 12 weeks of cognitive-behavioral treatment (Coping Cat), medication (sertraline), their combination, or a pill placebo. At pre- and post-treatment, parents completed self-report measures of global psychopathology symptoms, family functioning, and caregiver strain; parents, youths, and IEs rated youths' anxiety symptom severity.

Results—Changes in family functioning and caregiver strain jointly explained relations between parental psychopathology and reductions in youth anxiety. Specifically, across IE and parent informants, families with higher pre-treatment parental psychopathology showed more improvement in family functioning and caregiver strain, which in turn predicted greater youth

*Corresponding author: Tel: 1 917 439 1872; jschleider@fas.harvard.edu.

anxiety reductions. Further, higher pre-treatment parental psychopathology predicted greater caregiver strain reductions, and in turn, greater youth anxiety reductions, based on youths' reports of their own anxiety.

Conclusions—Findings suggest that improvements in family functioning and reductions in caregiver strain can influence treatment outcomes for anxious youths, especially among youths with more distressed parents.

Public health—Improvements in family functioning and caregiver strain can facilitate treatment outcomes for anxious youths, especially in families with more psychiatrically distressed parents.

Keywords

mediation; parental psychopathology; youth anxiety treatment; family functioning

Anxiety disorders are the most prevalent psychiatric conditions among youths (Costello, Egger, & Angold, 2005), predicting academic, interpersonal, and emotional difficulties (Piacentini, Peris, Bergman, Chang, & Jaffer, 2007). Fortunately, cognitive-behavioral and medication-based treatments can reduce anxiety symptoms and associated impairment (Silverman, Pina, & Viswesvaran, 2008; Ginsburg et al., 2011); however, some youths respond more favorably to these treatments than others (Kendall, 1994; Southam-Gerow, Kendall, & Weersing, 2001). Identification of predictor variables offers some insight into differential treatment responses and can inform refinements to extant treatments for specific subpopulations. For instance, family variables such as parental psychopathology and family dysfunction have emerged as predictors of poorer treatment response for anxious youths (Barrett et al 2005; Birmaher et al 2003; Ginsburg, Siqueland, Masia-Warner, & Hedke, 2004; Liber et al., 2008; Southam-Gerow et al 2001). An additional strategy for improving and streamlining treatments for subpopulations of youth involves identifying variables that influence the strength of association between pre-treatment factors and outcomes at post-treatment. When controlling for these variables weakens the direct association, the variables are called *mediators*; when controlling for these variables strengthens the direct association, the variables are called *suppressors* (Mackinnon, Krull & Lockwood, 2000; Rucker, Preacher, Tormala & Petty, 2011; Weisz, Ng, Rutt, Lau, & Masland, 2013). Identifying mediators and suppressors, henceforth referred to as *candidate explanatory variables*, can offer insight into mechanisms of change, or the nature of the relation between a predictor variable and the outcome. The present study used a multiple mediation technique, which tests for both suppression and mediation effects, to examine the nature of relations among three familial variables that have been linked to treatment response for anxious youth. Based on the literature, reviewed below, we hypothesized that improvements in family functioning, and reductions in caregiver strain across treatment conditions (cognitive-behavioral treatment, medication, their combination, or a pill placebo) would explain the relation between parental psychopathology and child treatment response. This question was tested using a sample of clinically-referred youths from the largest comparative treatment trial for pediatric anxiety disorders: the Child/Adolescent Anxiety Multimodal Treatment Study, or CAMS (Ginsburg et al., 2011; Walkup et al., 2008; Compton et al., in press; Piacentini et al., 2014).

Parental Psychopathology and Youth Treatment Response

Several studies have explored direct relations between parental psychopathology and youth anxiety treatment response. Cobham, Dadds, and Spence (1998) found that anxious youths with an anxious parent, compared to youths without an anxious parent, showed less improvement following group cognitive-behavioral therapy (CBT). Southam-Gerow and colleagues (2001) found that anxious youths with a depressed mother responded less favorably to individual CBT, compared to youths without a depressed mother. Similarly, lower parent self-reported symptoms of psychopathology predicted elimination of youth primary anxiety diagnosis as well as reduced symptom severity in an exposure-based treatment trial (Berman, Weems, Silverman, & Kurtines, 2000). Another study found that youths of mothers who had received treatment for clinical depression, but who had not received treatment themselves, showed decreases in anxiety symptoms one year later (Pilowsky et al., 2008).

However, these effects have not been fully consistent. For instance, parents' self-reported psychological symptoms failed to predict anxious youths' treatment outcome in three independent samples involving cognitive and behavioral treatments (Crawford & Manassis, 2001; Liber et al., 2008; Victor, Bernat, Bernstein, & Layne, 2007). Moreover, findings based on CAMS data found no relation between self-reported parental psychopathology and acute treatment outcomes (Compton et al, in press) or remission (Ginsburg et al., 2011) for anxious youth. Thus, evidence is mixed regarding effects of parental psychopathology on treatment outcomes for anxious youth.

Regardless of whether a direct effect persists across studies, parental psychopathology may influence youth treatment outcomes through mediating pathways. That is, parents' psychopathology may spur changes in other factors, such as family functioning or caregiver strain, which in turn affect youth treatment response. Indeed, the quantitative psychology literature suggests that there need not be a significant zero-order relation between independent and dependent variables for a theoretically sound mediation analysis (Zhao, Lynch, & Chen, 2009; Preacher & Hayes, 2008; MacKinnon, 2000). Thus, it remains important to parse whether parental psychopathology may influence youth anxiety treatment response through mediating factors, and if so, what the direction of the component effects may be.

Family Functioning and Youth Treatment Response

Family functioning is a complex, multidimensional construct, encompassing several conceptual domains. The study of family functioning in the context of youth psychiatric problems has largely relied on a framework outlined by Steinhauer and colleagues, labeled the *Process Model* (Skinner, Steinhauer, & Sitarenios, 2000; Steinhauer, 1987; Steinhauer, Santa-Barbara, & Skinner, 1984; see Skinner et al., 2000, for a review). The Process Model describes a conceptual framework for empirically assessing family functioning according to seven dimensions: task accomplishment (families' organizing to achieve tasks); role performance (families' allocation and enactment of responsibilities); communication (families' ability to achieve mutual understanding); affective expression (content, intensity,

and timing of feelings expressed among families); involvement (degree and quality of family members' interest in one another); control (family members influence over each other); and values and norms (e.g., scope allowed for family members to decide individual behaviors). The Brief Family Assessment Measure III (BFAM-III; Skinner, Steinhauer, & Santa-Barbara, 1995), which is used in the present study, was derived from the full Family Assessment Measure and assesses parents' perceived strengths and weaknesses in general family functioning. The total score represents an overall index of family functioning according to the domains assessed by the Process Model.

Similar to findings associated with parental psychopathology, links between family functioning and anxious youths' treatment response have been inconsistent. In a trial of 61 youths with diagnosed anxiety disorders, greater baseline family dysfunction (as measured by the B-FAM-III total score) predicted smaller clinician-rated symptom reductions across treatment (Crawford & Manassis, 2001). In another study, higher pre-treatment family cohesion (emotional bonding and connectedness among family members) predicted greater improvements in youth anxiety across treatment (Victor, Bernat, Bernstein, & Layne, 2007). However, another study based on CAMS data found that family functioning (i.e., B-FAM-III total score) did not predict youth anxiety treatment response (Compton et al., 2004). Further, in both the CAMS data (Keeton et al., 2013) and a separate trial (Crawford and Manassis, 2001), overall family functioning (in addition to youth anxiety) was found to improve with cognitive-behavioral and medication-based treatment modalities. Overall, evidence suggests that strong family functioning may facilitate treatment outcome, but additional research is needed to clarify the role of family functioning in the context of other salient factors.

Caregiver Strain and Youth Treatment Response

Caregiver strain refers to negative thoughts and feelings (e.g., stigma, guilt), as well as consequences (e.g. financial difficulties, household disruption) parents experience as a result of caring for a youth with emotional difficulties (Montgomery, Gonyea, & Hooyman, 1985; Platt, 1985). Decades of research suggest that caregivers of individuals experiencing mental illness experience caregiver strain due to their increased responsibilities (Clausen & Yarrow, 1955; Fisher, Benson, & Tessler, 1990; Grad & Sainsbury, 1968; Kreisman & Joy, 1974; Norbeck, Chafetz, Skodol-Wilson, & Weiss, 1991). One study using the Burden Assessment Scale (BAS; Reinhard, Gubman, Horwitz, & Minsky, 1994), the self-report measure used in the CAMS trial, has demonstrated that caregiver strain predicted unfavorable treatment outcome for clinically anxious youths, possibly by disrupting parents' capacity to engage positively with their youths (Crawford & Manassis, 2001). Studies using CAMS data corroborated this finding (Compton et al., 2004). CAMS data also demonstrated that, like family dysfunction, overall caregiver strain measured using the BAS improved over the course of cognitive-behavioral and medication-based youth anxiety treatments (Keeton et al., 2013).

Parental psychopathology, caregiver strain, and family functioning

Research suggests that parents higher in psychopathology tend to experience more severe caregiver strain and family dysfunction. For instance, mothers with depression have reported increased strain related to parenting responsibilities (Jackson & Haug, 2000; Sarason, Johnson, & Seigel, 1978). Further, parental history of mental health problems has strongly predicted parents' perception of burden related to their child's psychiatric symptomatology (Angold, Messer, Stangl, Farmer, Costello, & Burns, 1998). Over a ten-year period, parents with current or past depression (versus never-depressed parents) were more likely to experience an array of familial stressors related to poor family functioning: poor marital adjustment, low family cohesion, parental divorce, and affectionless control (i.e. low warmth and high protection from parents) (Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002). Research among families of anxious youth specifically has found positive associations between parental self-reported anxiety and depressive symptoms and general family dysfunction (Hughes, Hedtke, & Kendall, 2008). Therefore, parents experiencing psychopathology may be less well-equipped to cope with difficult events (e.g., a child's mental health problems), causing more strain and dysfunction in family interactions.

Effects of Parental psychopathology, Family Functioning, and Caregiver Strain on Youth Treatment Response

Despite data demonstrating bivariate relations among parental psychopathology, family functioning, caregiver strain, and treatment response for anxious youths, a theoretical model outlining the mechanisms by which these variables affect youth outcomes has yet to be tested. The current study tested whether the relation between lower parental psychopathology and improved youth anxiety treatment response was explained by improvements in family functioning and reductions in caregiver strain. We employed a multiple mediation technique to test parallel effects of these candidate explanatory variables. This model is based on the premise that parents with less psychopathology may be better-equipped emotionally to participate in treatment requirements, maintain positive or easily modify negative family interactions, and support their youth's efforts during treatment. Indeed, in a meta-analysis on predictors of parent training efficacy for youth behavioral problems, maternal psychopathology emerged as the most consistent predictor of poorer youth treatment response (Reyno & McGrath, 2006). The authors suggested that this finding likely reflected the high task demands involved in parent training. Specifically, successful outcomes required a high level of motivation, consistent implementation of behavior modification techniques, and changes in family interactions all of which were more challenging for parents with high levels of psychopathology. Others have also suggested that parents experiencing more psychopathology may be less emotionally equipped to work toward improved family functioning and may have more difficulty modifying family interactions during treatment (Southam-Gerow et al., 2001). In contrast, parents low in psychopathology may be able to make quicker, more sustained course corrections in family interactions (e.g. reducing accommodation) that reduce strain associated with their youth's disorder and complement treatment response.

Although the above model has intuitive appeal, alternative models are theoretically plausible (see Table 1). Correlational studies have established relations between family functioning, parental psychopathology, and caregiver strain; however, these factors likely have reciprocal influences (Cummings, Keller, & Davies, 2005). Therefore, it remains possible that higher quality baseline family functioning might predict greater improvements across treatment in parental psychopathology and caregiver strain (Model 1, Table 1). Parents in less dysfunctional homes might more readily experience reductions in psychopathology and strain across treatment; in turn, these reductions might boost youth anxiety improvements. Similarly, lower baseline caregiver strain might enable greater improvements in parental psychopathology and family functioning across treatment, thereby facilitating youth treatment response (Model 2, Table 1). Alternatively, given reciprocal links between parent improvements, including parent psychopathology, and youth improvements across youth treatment (Silverman et al., 2009), youth anxiety reductions might influence the relation between parental psychopathology and family functioning and/or caregiver strain (Models 3 and 4, Table 1). That is, decreases in youth anxiety symptoms may directly reduce parents' strain related to their youth's anxiety and improve family functioning. To determine the specificity of the proposed model, we tested these alternate models as part of the analyses.

For both proposed and alternate models, we examined a sample of clinically-referred youths with anxiety disorders enrolled in CAMS (Compton et al., in press; Ginsburg et al., 2011; Kendall et al., 2011; Piacentini et al., 2014; Walkup et al., 2008). CAMS enrolled 488 youth and compared the relative efficacy of cognitive-behavioral treatment (Coping Cat program), medication (sertraline), their combination (COMB), or a pill placebo (PBO) for pediatric anxiety disorders; all active treatments outperformed PBO and COMB led to the largest reductions in youth symptoms and diagnoses. Secondary studies using CAMS data found that lower caregiver strain (but not parental psychopathology or family functioning) predicted better youth outcomes (Compton et al., in press), and that both family functioning and caregiver strain significantly improved from pre- to post-treatment, across treatment conditions, including PBO (Keeton et al., 2013). This study builds on these findings, testing whether improvements in familial stressors might jointly explain relations between parental psychopathology and youth treatment response across treatment modalities. We also tested whether treatment condition moderated the strength of these effects. However, because changes in family functioning and caregiver strain may reasonably influence youth functioning regardless of what kind of treatment they receive, we did not expect the model to differ by treatment condition. Finally, to reduce the risk that single-informant idiosyncrasies regarding youth anxiety might affect findings, we conducted analyses separately for youth, parent, and independent evaluator (IE) reports of youth anxiety, testing whether the proposed model was robust across informants.

Methods

Procedure

Participants were part of the CAMS trial, conducted across six medical and academic institutions in the US. CAMS enrolled 488 youths (ages 7–17) who met DSM-IV-TR criteria for generalized anxiety disorder, social phobia, and/or separation anxiety disorder, and their

parents. Mean age was 10.69 years ($SD = 2.80$), and 74.2% were 7–12 years old; 49.6% of the participants were female and 78.9% were Caucasian. Most participants (74.5%) were of middle to high socioeconomic status, as indicated by a score of 40–66 on the Hollingshead four-factor index of social status (Hollingshead, 1971).

One parent of each youth completed pre- and post-treatment questionnaire batteries. Of these parents, 87.0% were mothers; 81.0% shared parenting responsibilities with another adult, and 19% were single parents. In dual-parent households, the “non-primary” caregivers were 91.14% biological parents, 6.29% stepparents, and 2.27% non-married partners.

Study procedures were approved by each site’s Institutional Review Board. Before completing study procedures, participants signed informed consent. Diagnostic eligibility was determined using the Anxiety Disorders Interview Schedule for Children and Parents (ADIS-C/P); participants completed questionnaires before being randomly assigned to 12 weeks of youth-focused treatment in one of four conditions. At post-treatment, diagnostic evaluations were repeated by an IE and youths and parents repeated the questionnaires. IEs were MA-level psychologists, social workers, a nurse practitioner, PhD psychologists and child psychiatrists, who were selected based on experience and predetermined background criteria. IEs were trained to reliability and engaged in regular supervision, both within and across sites (Kendall et al., 2010). Detailed demographic data and diagnostic characteristics are described in Kendall and colleagues (2010) and Walkup and colleagues (2008).

Measures

Youth Anxiety—Global severity of youth anxiety symptoms and impairment was rated by an IE at baseline and 12 weeks post-treatment using the one-item Clinical Global Impressions-Severity Scale (CGI-S; Guy, 1976). Scores range from 1 (*not at all ill*) to 7 (*extremely ill*). The CGI-S is a widely used measure of outcomes, especially in psychopharmacological pediatric clinical trials. The CGI-S is strongly related to self-report and clinician-administered measures of youth symptomatology and functional impairment (Zaider, Heimberg, Fresco, Schneier, & Liebowitz, 2003).

We also used the Pediatric Anxiety Rating Scale (PARS) (RUPP Anxiety Study Group, 2002) to assess youth treatment response. The PARS is an IE-rated 50-item anxiety symptom checklist and includes 6 anxiety severity/impairment items specifically addressing the combined symptoms of anxiety across disorders (e.g., SAD, GAD, SP). The same IE administered the CGI-S and the PARS to each youth. The PARS has excellent inter-rater reliability (> 0.97), as well as satisfactory convergent and divergent validity: PARS total scores have shown positive correlations with other measures of youth anxiety (i.e., the Screen for Child Anxiety Related Emotional Disorders; Birmaher et al., 1997) but not with youth depression measures (Children’s Depression Inventory; Kovacs, 1978) (Ginsburg, Keeton, Drazdowski, & Riddle, 2011). PARS scores have shown sensitivity to treatment, paralleling change in other measures of youth anxiety symptoms and global improvement (RUPP Anxiety Study Group, 2002). The PARS was used as the primary outcome measure in the main CAMS trial.

Additionally, we measured pre- and post-treatment youth anxiety using the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997), a 41-item youth- and parent- report instrument assessing youth anxiety symptoms in the past 3 months. Participants rate each item (e.g., “I worry/My child worries about sleeping alone”) on a three-point Likert scale from 0 (*Not True/Hardly Ever True*) to 2 (*Very True/Often True*). The SCARED includes subscales for panic/somatic, generalized anxiety, social phobia, separation anxiety, and school phobia, as well as a total score, with higher scores indicating more anxiety. Both the sub-scales and total score have sufficient reliability and have been shown to differentiate between youth anxiety, depressive and externalizing disorders and between different anxiety disorders (Birmaher et al. 1999). In this study, we used the total youth and parent SCARED scores to assess baseline and post-treatment youth anxiety. Alphas for the total scores were .93 for youths and .90 for parents at baseline, and .94 for youths and .93 for parents at post-treatment.

Parental psychopathology—Parents completed the Brief Symptom Inventory (BSI; Derogatis 1993), a 53-item self-report measure of distress associated with parental psychopathology. Items are measured on a 5-point Likert scale from 0 (*not at all*) to 4 (*extremely*); scale scores are calculated by taking the mean item rating. Scores are obtained on nine scales (e.g., Somatization, Obsessive-Compulsive). The BSI’s General Severity Index (GSI) is a weighted frequency score based on the sum of ratings the subject has assigned to each symptom. Due to significant intercorrelations among BSI symptom subscales, research suggests that the measure may be best used as a general distress indicator; convergent validity for the GSI as a measure of general psychopathology has been demonstrated through correlations with clinical scales on the Minnesota Multiphasic Personality Inventory and total scores on the SCL-R-90 (Derogatis, 1977), a well-validated measure of psychopathology in adults (Boulet & Boss, 1991; Derogatis, 1993). Thus, in this study, the GSI was used to assess general distress associated with symptoms of psychopathology. Derogatis and Melisaratos (1983) have reported both test-retest (across two weeks) and internal consistency reliabilities of the GSI, which ranged .68 to .91 and .71 to .90, respectively. In this study, alpha was .95 at pre- and post-treatment assessments.

Family Functioning—The Brief Family Assessment Measure-III (BFAM-III; Skinner, Steinhauer, & Santa-Barbara, 1995) is a 14-item parent-report questionnaire assessing perceptions of family functioning during the previous two weeks. This instrument was created to provide an operational definition and means of measuring the seven constructs in the Process Model of Family Functioning; it includes two items relating to each construct (Skinner et al., 2000). Items such as “We take the time to listen to each other” and “When things aren’t going well it takes too long to work them out” are scored on a 5-point scale. Items are summed to create a total score that is converted into a *T* score. Individuals with a psychologically ill family member have shown higher BFAM scores than individuals without a psychologically ill family member, demonstrating discriminant validity (Jacob, 1995). Further, strong links between MMPI special family scales and the BFAM support the BFAM’s construct validity (Bloomquist & Haris, 1984). Higher BFAM scores reflect greater perceived family dysfunction. In this study, alpha was .80 at pre-treatment and .87 at post-treatment.

Caregiver Strain—The 21-item Burden Assessment Scale (BAS; Reinhard, Gubman, Horwitz, & Minsky, 1994) measures caregiver strain associated with having a youth with a mental health disorder. Parents indicated the degree to which their youth’s anxiety disrupts aspects of family life, routines, and emotions (e.g., “impact on work,” “impact on family activities,” “how resentful did you feel”) over the past two weeks on a scale ranging from 1 (*not at all*) to 5 (*very much*). A higher score signifies greater burden. Content validity for the BAS has been demonstrated: caregivers for a relative with mental illness report higher BAS scores than caregivers for a relative without mental illness, and BAS scores for the former caregiver group decrease following treatment for their relative (Reinhard et al., 1994). Consistent with high internal consistency in initial studies (Reinhard et al., 1994), alpha for this sample was .91 at pretreatment and .93 at post-treatment.

CAMS Treatment Conditions

Participants received pharmacotherapy with sertraline (SRT); pharmacotherapy with a placebo drug (PBO); cognitive-behavioral therapy protocol (CBT) using the Coping Cat manual for children, and the developmental modification, the CAT Project, for adolescents (Kendall & Hedtke, 2006; Kendall, Choudhury, Hudson, & Webb, 2002); or a combination treatment (COMB) including all components from SRT and CBT. The SRT and PBO conditions were double-blinded, dosing was determined by a pharmacotherapist, and medication was dispensed by an investigational pharmacist. Acute treatments spanned a 12-week period. CBT involved twelve individual, youth-focused sessions and two parent sessions over the course of 12 weeks. The first six sessions focused on teaching the youth new skills (e.g., relaxation training, cognitive restructuring), and the second six offered the youth opportunities to practice anxiety management skills through graded exposures. Parent sessions focused on psychoeducation and supporting the youth; parental psychopathology and familial stressors were not directly addressed. See Compton and colleagues (2010) for more detailed descriptions of the treatment conditions.

Data analyses—We tested a multiple mediation model, which involves simultaneous indirect effects by multiple variables (Preacher & Hayes, 2008, p. 880). Preacher and Hayes recommended that testing a multiple mediation model involves (a) an analysis of the total indirect effect (the aggregate indirect effect of all the candidate explanatory variables under investigation) and (b) an analysis of specific indirect effects (the indirect effect of each specific candidate explanatory variable). Notably, suppressors and mediators are tested using the same statistical techniques (Rucker, Preacher, Tormala, & Petty, 2011). Therefore, using Preacher and Hayes’ multiple mediation technique would help identify whether this study’s candidate explanatory variables might be best described as *suppressors* or *mediators* within the model.

Present analyses used bias-corrected bootstrapping, a nonparametric sampling procedure, to test the significance of both specific and total indirect effects. Bootstrapping has the advantage of greater statistical power without assuming multivariate normality in the sampling distribution, lending itself to parsimonious analysis of multiple mediators or suppressors (Mallinckrodt, Abraham, Wei, & Russell, 2006; Preacher & Hayes, 2008). An SPSS macro designed for multiple mediation models tested the proposed model (Preacher &

Hayes, 2008). Bootstrap analyses use the obtained sample to generate multiple random samples with replacement that serve as the basis for repeatedly computing the statistic under investigation (Mallinckrodt et al., 2006). To test for indirect effects of candidate explanatory variables, parameter estimates of total and specific indirect effects are generated, along with their confidence intervals, using 1,000–20,000 random samples. In the present study, 5,000 resamples were specified, per Preacher and Hayes' (2008) recommendations. If the 95% bias-corrected confidence interval for the total indirect parameter estimate does not contain zero, then the total indirect effect can be considered statistically significant, demonstrating multiple mediation (Preacher & Hayes, 2008).

In the proposed model, parental psychopathology was specified as the independent variable, and post-treatment IE-, parent-, or youth-rated youth anxiety, as the dependent variable. Candidate explanatory variables were *z*-change scores between pre- and post-treatment family functioning and caregiver strain. Scores were calculated according to the formula: $(M_{\text{pre-tx}} - M_{\text{post-tx}}) / SD_{\text{pre-tx}}$. Due to established intercorrelations between race, socioeconomic status, and both parent and youth problems (Dawson, 1991; Siegel, Aneshensel, Taub, Cantwell, & Driscoll, 1998), we included parent race and family SES as covariates. We also included youth age as a covariate, as some studies have found differences in anxiety treatment response for older versus younger children (Southam-Gerow et al., 2001). To account for possible demographic differences across study sites, we also controlled for treatment site. Finally, we controlled for pre-treatment IE-rated, youth-rated, or parent-rated youth anxiety scores in order to investigate treatment-related changes in youth anxiety. Reported results include covariates in analyses. We used the same multiple mediation procedure to test the alternate models (Table 2). Finally, we used a moderated mediation technique to test whether the indirect effects differed by treatment condition. Moderated mediation occurs when the strength of an indirect effect depends on the level of a variable. In this study, moderated mediation would be expressed by significant interactions between treatment condition and the candidate explanatory variables (condition X improvements in family functioning/caregiver strain). We followed Preacher and Hayes' (2008) guidelines to carry out this test, using the same SPSS macro as for the main multiple mediation analyses.

A small amount of data was missing from the sample (less than 0.25%). To handle missing data, we used a sequential regression multivariate imputation algorithm in the SAS IVEware package, assuming data points were missing at random (Little & Rubin, 2002). Twenty imputed data sets were generated; results of multiple mediation analyses on each imputed data set were combined using Rubin's guidelines (Little & Rubin, 2002).

Results

Descriptives and Correlations

Means, standard deviations, and correlations for parental psychopathology, IE-, youth-, and parent-rated youth anxiety severity pre- and post-treatment, family functioning, and caregiver strain are presented for the total sample in Table 1. Greater improvements in caregiver strain correlated positively with higher pre-treatment parental psychopathology and lower post-treatment IE- and parent-rated (but not youth-rated) youth anxiety. Greater

improvements in family functioning correlated positively with higher parental psychopathology and lower post-treatment youth anxiety across informants. Youth- and parent-rated (but not IE-rated) post-treatment youth anxiety correlated negatively with pre-treatment parental psychopathology. Parents with more psychopathology at baseline reported greater improvements in caregiver strain, $t(486) = 2.72, p = .01$, and family functioning, $t(486) = 3.11, p < .01$, across all treatment conditions.

Proposed Model

IE-rated Youth Anxiety (CGI-S)—As shown in Figure 1, the test for multiple mediation predicting IE-rated youth anxiety severity based on the CGI-S revealed a non-significant direct effect of parental psychopathology on youth anxiety severity at post-treatment (path c). Because indirect effects can occur without a direct effect of the independent variable (IV) on the dependent variable (DV) (Preacher & Hayes, 2008; MacKinnon, 2000), reflecting patterns not evident through direct effects alone, we proceeded with planned analyses. After controlling for the combined effect of both candidate explanatory variables, the path (c') remained non-significant. Based on unstandardized regression coefficients, higher baseline parental psychopathology significantly predicted improvements in family functioning and reductions in caregiver strain across treatment, which both individually predicted lower post-treatment IE-rated youth anxiety severity. The indirect effect of parental psychopathology on post-treatment IE-rated youth anxiety severity was significant through improvements in family functioning, 95% CI: $(-.37, -.09)$, and through reductions in caregiver strain, 95% CI: $(-.14, -.02)$. Both confidence intervals suggest significant specific indirect effects. Specifically, higher baseline parental psychopathology predicted greater improvements in family functioning and greater reductions in caregiver strain, which in turn predicted lower post-treatment IE-rated youth anxiety.

The indirect effect of parental psychopathology on post-treatment IE-rated youth anxiety severity through *both* candidate explanatory variables had a bias-corrected, 95% confidence interval between $-.45$ and $-.15$, suggesting a significant indirect effect for the full model. That is, parental psychopathology predicted post-treatment IE-rated youth anxiety severity through improvements in family functioning *and* reductions in caregiver strain, assessed in parallel. The model's total indirect effect accounted for 23.93% of variance in post-treatment youth anxiety severity on the CGI-S, $R^2 = .24$, whereas only 9.41% of the model's total variance was explained by parental psychopathology and the covariates (baseline IE-rated youth anxiety severity, SES, youth age, treatment site, and race) alone, $R^2 = .09$. Thus, including the explanatory variables in the model explained an additional 14.52% of the total model variance (variance explained by total indirect effect - variance explained by independent variable and covariates alone). A contrast of the specific indirect effects revealed that reductions in caregiver strain across treatment had a greater indirect effect on post-treatment youth anxiety severity than did improvements in family functioning, 95% CI: $(-.30, -.01)$.

IE-rated Youth Anxiety (PARS)—To corroborate findings based on the CGI-S, we also tested this model predicting the IE-rated PARS. In this model, the indirect effect of parental psychopathology on post-treatment parent-rated youth anxiety severity was significant

through both improvements in family functioning, 95% CI: (-.67, -.08), and through reductions in caregiver strain, 95% CI: (-1.82, -.45). Further, the indirect effect of parental psychopathology on post-treatment IE-rated youth anxiety through both candidate explanatory variables had a bias-corrected, 95% confidence interval between -2.17 and -.65, suggesting a significant indirect effect for the full model. As in the CGI-S model, parental psychopathology predicted post-treatment IE-rated youth anxiety through improvements in family functioning *and* reductions in caregiver strain, assessed in parallel. The model's total indirect effect accounted for 28.47% of variance in post-treatment youth anxiety on the PARS, $R^2 = .28$, whereas only 11.64% of this variance was explained by parental psychopathology and the covariates (baseline IE-rated youth anxiety severity, SES, youth age, treatment site, and race) alone, $R^2 = .12$. Thus, including the explanatory variables in the model explained an additional 16.83% of the total model variance. A contrast of the specific indirect effects revealed that reductions in caregiver strain across treatment had a greater indirect effect on post-treatment youth anxiety severity than did improvements in family functioning, 95% CI: (-1.54, -.10).

Parent-rated Youth Anxiety—We next tested this model predicting parent-rated post-treatment youth anxiety severity (Figure 2), and results were quite similar to those based on IE-rated outcome. Baseline parental psychopathology significantly predicted improvements in family functioning and reductions in caregiver strain across treatment, which both individually predicted lower post-treatment parent-rated youth anxiety severity. The indirect effect of parental psychopathology on post-treatment parent-rated youth anxiety severity was significant through improvements in family functioning, 95% CI: (-1.11, -.17), and through reductions in caregiver strain, 95% CI: (-2.87 and -.51). Both confidence intervals suggest significant specific indirect effects. Specifically, higher baseline parental psychopathology predicted greater improvements in family functioning and greater reductions in caregiver strain, which in turn predicted lower post-treatment parent-rated youth anxiety.

Further, the indirect effect of parental psychopathology on post-treatment parent-rated youth anxiety severity through *both* candidate explanatory variables was significant, 95% CI: (-3.54 and -.97), suggesting a significant indirect effect for the full model. That is, parental psychopathology predicted post-treatment parent-rated youth anxiety severity through improvements in family functioning *and* reductions in caregiver strain, assessed in parallel. The model's total indirect effect accounted for 29.64% of the variance in post-treatment parent-rated youth anxiety severity, $R^2 = .30$, whereas only 13.33% of this variance was explained by parental psychopathology and the covariates (baseline parent-rated youth anxiety severity, SES, youth age, treatment site, and race) alone, $R^2 = .13$. Thus, including the explanatory variables in the model explained an additional 16.31% of the total model variance. A contrast of the specific indirect effects revealed that neither improvements in family functioning nor reductions in caregiver strain had a stronger indirect effect than the other on post-treatment youth anxiety severity, 95% CI: (-2.46, .01).

Youth-rated Youth Anxiety—Next, we tested the model predicting youth-rated post-treatment youth anxiety severity. Baseline parental psychopathology significantly predicted

improvements in both family functioning and caregiver strain across treatment; the latter significantly predicted lower post-treatment youth-rated youth anxiety, but the former did not. Because tests of indirect effects require a significant association between the candidate explanatory variable and the outcome variable (Preacher & Hayes, 2008), we tested only the specific indirect effect of parental psychopathology on post-treatment parent-rated youth anxiety severity through improvements in caregiver strain. This indirect was significant through improvements in caregiver strain, 95% CI: (-1.98, -.47). That is, higher baseline parental psychopathology predicted greater reductions in caregiver strain, which in turn predicted lower post-treatment youth-rated youth anxiety. The specific indirect effect through improvements in family functioning was not significant. Because we were unable to test whether improvements in family functioning account for the relation between parental psychopathology and post-treatment youth anxiety severity, we did not test the full proposed model using youth-reported outcomes.

Alternate Models and Moderation by Treatment Condition—We then tested four alternate models to assess the specificity of the predicted configuration of variables (summarized in Table 1). The total indirect effects for all of these models, across IE-, parent-, and youth-reports of youth anxiety severity, had 95% confidence intervals that included zero. Thus, the relation between parental psychopathology and post-treatment youth anxiety severity seemed uniquely explained through changes in family functioning and caregiver strain.

Finally, we tested whether the proposed model differed by treatment condition. Results of this analysis found no evidence for moderation of the total indirect effect by treatment condition, across the IE- and parent-report models. Additionally, when analyses were run separately for youths in each treatment condition, the total indirect effects for the proposed models were significant across all treatment conditions.

Discussion

This study assessed family functioning and caregiver strain as candidate explanatory variables between baseline parental psychopathology and post-treatment anxiety severity in clinically anxious youths. Consistent with hypotheses, results indicated that improvements in family functioning and reductions in caregiver strain led to lower post-treatment youth anxiety. However, in contrast to initial predictions, families in which parents reported higher psychological distress showed greater improvements in family functioning and reductions caregiver strain, which in turn was associated with larger reductions in youth anxiety from pre- to post-treatment. This pattern was consistent across IE and parent informants. In youth informants, improvements in caregiver strain but *not* in family functioning explained indirect relations between higher parental psychopathology and lower post-treatment youth anxiety.

Importantly, the proposed model was significant for the predicted ordering of variables only: *parental psychopathology* → *changes in family functioning/caregiver strain* → *youth treatment response*, not for alternative orderings that had some theoretical support (see Table 1) or reductions in youth anxiety leading to changes in family variables. Although

some evidence has suggested the bidirectional dynamics of change in youth anxiety treatment between parents and youths (Silverman et al., 2009), this study can speak to only one of these directions.

The partially unexpected finding regarding parental psychopathology might have emerged for several reasons. Parents who experience high levels of psychopathology may be more motivated to improve the familial environment, and psychologically distressed parents might have felt greater relief upon initiating treatment. Related, psychologically distressed parents may have had more “room to improve” with respect to their family functioning and caregiving strain. However, regression to the mean could not explain the *indirect* effects of parental psychopathology on post-treatment youth anxiety severity: across informants, youth anxiety severity did not differ by parental psychopathology. That is, in homes with more distressed parents, the relatively larger improvements in family functioning and reductions in caregiver strain benefited both parents (by improving the family environment) and youths (by facilitating reductions in anxiety). These findings fit with prior research from CAMS (Keeton et al., 2013) and separate trials (Crawford & Manassis, 2001; Victor, et al., 2007) suggesting that youth anxiety can confer “spillover” benefits for family members and that alleviation in familial stressors across treatment can improve youth outcomes. In the CAMS sample, improvements in familial factors might have helped parents better support their youths’ progress, thereby facilitating youth improvements.

Notably, across IE-, parent-, and youth-report models, the relation between pre-treatment parental psychopathology and child anxiety at post-treatment *grew stronger* after controlling for effects of candidate explanatory variable(s). In mediation, this relation is expected to *grow weaker* after accounting for these variables. When the present pattern emerges, and when the direct and indirect effects have opposite signs, the total effect is described as *suppression* (Mackinnon, Krull & Lockwood, 2000). Suppression occurs when a variable increases the predictive validity of another variable by its inclusion in a regression equation (Tzelgov & Henik, 1991). In this study, including improvements in familial stressors in the model clarified the role of parental psychopathology in youth anxiety treatment response: omitting improvements in family functioning and caregiver strain from the model *undermined* the effect of higher parental psychopathology on better youth treatment response, whereas accounting for them *revealed* this effect. The presence of suppression in these models reveals the complexity of links between parent psychopathology and youth anxiety treatment outcomes, which may be more than correlations alone can identify. In this study, we identified indirect pathways that may carry implications for clinical practice: higher parent psychopathology related to improvements in critical family processes, which in turn were associated with youth anxiety reductions. This pattern was robust across IE and parent informants; the same effect emerged for youth informants, but with improvements in caregiver strain as the only significant suppressor variable.

By suggesting particular mechanisms of change, present findings might inform clinical decision making in youth anxiety treatment. Specifically, explicitly targeting family dysfunction and caregiver strain in treatment may be especially helpful for youths with more psychologically distressed parents, for whom improvements in the family environment more strongly predicted reduced post-treatment anxiety severity. Indeed, the total indirect effect of

the model tested in this study accounted for close to a quarter of reductions in youth anxiety severity across all CAMS treatment conditions in parent and IE informants, with the changes in family functioning and caregiver strain on their own alone accounting for about 16%, despite the fact that none of these conditions targeted familial stressors. Interventions that *do* address these stressors might lead to even greater improvements in family environment, and in turn, more favorable outcomes for youths (see Manassis et al, in press). Further, that the model held across treatment conditions suggests the general relevance of familial stressors to treatment response in youths. Family functioning, caregiver strain, and parental psychopathology may be relevant to youth treatment outcomes across a range of intervention modalities.

This study has limitations that warrant mention and suggest future research. First, this study could not address all familial stressors relevant to youth treatment outcome. For example, negative parenting practices, such as psychological control and rejection, have shown prospective relations to anxiety and treatment outcome in youths (Schleider, Vélez, Krause, & Gillham, 2014). Indeed, improvements in parenting practices have been shown to influence youths' anxiety treatment response (Khanna & Kendall, 2009). Such improvements might be tested as explanatory variables in future studies. A second limitation, common in family-based clinical research, is that the majority of parent participants (87%) were mothers. Thus, we lacked sufficient statistical power to explore effects of parent gender on the mechanisms observed. Low paternal participation is an ongoing concern in intervention research with families (Phares, Lopez, Fields, Kamboukos, & Duhig, 2005). Future studies including large numbers of male and female caregivers may clarify potentially different links among mothers' and fathers' psychopathology, familial stressors, and youth treatment response. Additionally, the present study assessed *changes from pre- to post-treatment* in family functioning and caregiver strain. However, the strongest tests of explanatory variables involve interim assessment points: that is, measurement of these variables after measurement of the independent variable, but before measurement of the dependent variable. Because no interim assessments of the explanatory variables were available, we used change scores for family functioning and caregiver strain. This approach helped reduce the possibility that youth anxiety reductions might have driven changes in explanatory variables. Nonetheless, future studies might assess family functioning and caregiver strain at various points during treatment to more conclusively establish causal, explanatory mechanisms. Separately, because the BSI has been shown to be most useful as a measure of global distress associated with psychopathology (Boulet & Boss, 1991), we did not test effects of specific parent symptom clusters on youth outcomes. Further, the GSI correlates strongly with other self-report symptom scales, but little data is available on links between GSI scores and psychiatric diagnoses based on structured clinical interviews. Thus, as noted, GSI scores reflect general subjective distress rather than the presence of psychopathology. Future studies might employ comprehensive measures of parental psychopathology to test whether various parent symptoms, or the presence of certain disorders, differently influence youth treatment response. In addition, although parents' GSI scores ranged widely in the present sample, the average GSI score did not reflect greater distress compared to other community adult samples (Boulet & Boss, 1991; Derogatis, 1993). Thus, present findings may not extend to parent populations experiencing

higher mean distress. Finally, the sample was largely Caucasian and of middle-to-high SES, limiting generalizability of findings to other ethnic and socioeconomic groups.

The present study also has several strengths. First, while existing literature demonstrates individual effects of parental psychopathology, family dysfunction, and caregiver strain on youth treatment response, our study is the first, to our knowledge, to assess their joint influences on youth treatment response. Second, few studies on relations between parental psychopathology and youth treatment outcome have employed multiple mediation techniques to assess underlying mechanisms. Researchers have emphasized that effects of familial stressors on youth anxiety treatment response are likely to involve myriad factors (Ginsburg et al., 2002). Our findings suggest that multiple mediation is a useful tool for parsing these complex, interrelated processes. Third, our use of the large, clinically-referred CAMS sample renders the findings relevant to high-risk populations. Fourth, the fact that the same general pattern was evident in analyses for three separate and independent informants (parents, youths, and IEs) suggests that the pattern is reliable and robust. Overall, findings suggest that family functioning and caregiver strain can improve treatment outcomes for anxious youths, especially in families with more distressed parents. Further research should explore implications of these findings for personalized treatment protocols for youth.

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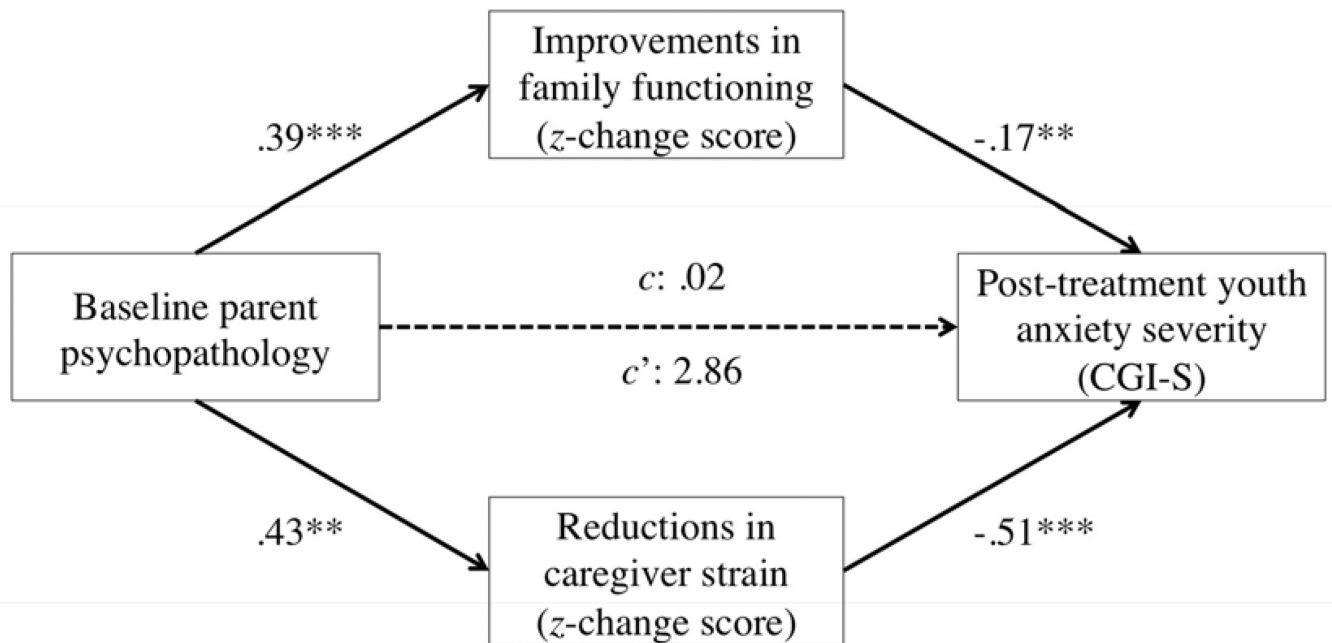


Figure 1. Multiple mediator model with unstandardized regression coefficients, predicting IE-rated post-treatment youth anxiety severity and controlling for pre-treatment youth anxiety severity. Parental psychopathology predicted post-treatment IE-rated youth anxiety severity through improvements in family functioning and reductions in caregiver strain, with their independent effects assessed in parallel. $*p < .05$, $**p < .01$, $***P < .001$.

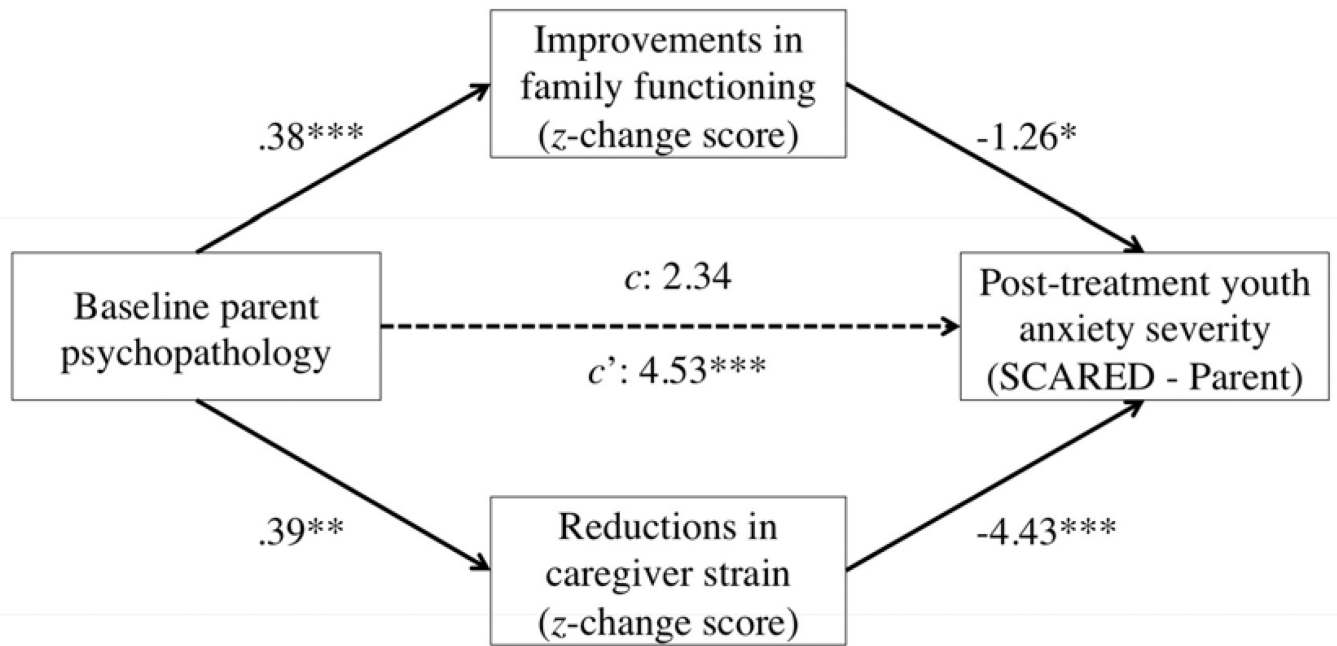


Figure 2. Multiple mediator model with unstandardized regression coefficients, predicting parent-rated post-treatment youth anxiety severity and controlling for pre-treatment youth anxiety severity. Parental psychopathology predicted post-treatment parent-rated youth anxiety severity through improvements in family functioning and reductions in caregiver strain, with their independent effects assessed in parallel, $*p < .05$, $**p < .01$, $***p < .001$.

Table 1

Summary of alternate models

	Independent Variable	Explanatory variables	Dependent Variable
Model 1	Baseline family functioning	1 Change in parental psychopathology 2 Change in caregiver strain	Post-treatment youth anxiety severity
Model 2	Baseline caregiver strain	1 Change in parental psychopathology 2 Change in family functioning	Post-treatment youth anxiety severity
Model 3	Baseline youth anxiety severity	1 Change in parental psychopathology 2 Change in caregiver strain	Post-treatment family functioning
Model 4	Baseline youth anxiety severity	1 Change in parental psychopathology 2 Change in family functioning	Post-treatment caregiver strain

Table 2

Correlations, means and standard deviations, all study variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
(1) Pre-tx youth anxiety severity: IE-report/PARS	--	.31***	.76***	.29***	.48***	.24***	.32***	.25***	.05	.14***	.09***
(2) Post-tx youth anxiety severity: IE-report/PARS	--	--	.23***	.86***	.14***	.63***	.11*	.43***	.01	-.36***	-.12***
(3) Pre-tx youth anxiety severity: IE-report/CGI-S	--	--	--	.27***	.42***	.20***	.27***	.19***	.03	.09*	.08
(4) Post-tx youth anxiety severity: IE-report/CGI-S	--	--	--	--	.11*	.60***	.09*	.45***	.015	-.13***	-.35***
(5) Pre-tx youth anxiety severity: Parent-report	--	--	--	--	--	.30***	.41***	.22***	.16***	.05	.16***
(6) Post-tx youth anxiety severity: Parent-report	--	--	--	--	--	--	.20***	.49***	.15***	-.12***	-.35***
(7) Pre-tx youth anxiety severity: Youth-report	--	--	--	--	--	--	--	.41***	.07	.02	.02
(8) Post-tx youth anxiety severity: Youth-report	--	--	--	--	--	--	--	--	.10*	-.04	-.20***
(9) Parent psychopathology	--	--	--	--	--	--	--	--	--	.17***	.17***
(10) Change in family functioning	--	--	--	--	--	--	--	--	--	--	.17***
(11) Change in caregiver strain	--	--	--	--	--	--	--	--	--	--	--
Mean	19.18	9.70	5.02	2.95	32.12	13.90	23.40	11.52	.48 (T=58)	.20	.67
SD	4.21	6.61	.72	1.45	12.83	11.55	15.09	11.62	.42 (T=66.12)	.96	1.03

* $p < .05$,** $p < .01$,*** $p < .001$