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The Interplay of Familial and Individual Risk in Predicting Trajectories of Clinical Improvements in Pediatric Anxiety Disorders

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Abstract

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Bioecological models of developmental psychopathology underscore the role of familial experiences of adversity and children's individual-level characteristics in heightening risk for pediatric anxiety through direct, combined, and interactive effects. To date, much of the existing research dedicated to pediatric anxiety disorders has largely been examined in bioecological models of diathesis-stress using community samples.

OBJECTIVE: This study extends our understanding of children's differential responsiveness to familial adversity by examining the diathesis-stress interaction of cumulative risk and children's individual-level vulnerabilities (negative affectivity and coping efficacy) within a clinic-referred treatment study for pediatric anxiety disorders.

METHOD: A cumulative risk index assessing exposure to familial adversity (e.g., socioeconomic status, parent psychiatric illness), and self-reported measures of children's negative affectivity and coping efficacy were each measured at the intake of a randomized controlled clinical trial for the treatment of pediatric anxiety disorders (N = 488; ages 7–17). Trajectories of interviewer-rated anxiety symptoms were assessed across 12-weeks of treatment at baseline, 4, 8 and 12 weeks.

RESULTS: Consistent with models of temperamental risk for mental health problems, negative affectivity predicted higher anxiety symptoms at intake. A significant diathesis-stress interaction between cumulative risk and coping efficacy emerged, as high risk and perceptions of lower coping efficacy attenuated declines in anxiety across 12-weeks. These patterns did not differ across treatment conditions.

CONCLUSION: The results indicate that for youth experiencing high levels of stress, additional treatment efforts targeting familial stressors and coping efficacy may be important in maximizing treatment outcomes.

Keywords

Cumulative Risk; Pediatric Anxiety; Coping Efficacy

Chronic and adverse familial environments deviate from normative and expected environments and are characterized by disruption, danger, and stress (Felitti & Anda, 1997; Gest, Reed & Masten, 1999; McLaughlin, 2016). The impact of adverse family environments is not benign, as frequent and repeated stress tax developing biobehavioral stress response systems and may exacerbate vulnerability to mental health conditions, including anxiety disorders (Loman & Gunnar, 2011; Luthar, Crossman, & Small, 2015). Individuals' vulnerability to the impact of adversity (e.g., adverse family environments), in which some children are more susceptible to the negative effects of chronic exposure to adverse experiences, has been traditionally quantified using diathesis-stress models (Boyce & Ellis, 2005), which may facilitate our understanding of which individuals flourish or falter when exposed to stressors such as familial socioeconomic strain or parent mental health problems. These nuanced models are particularly relevant for clarifying what factors and processes may predict the maintenance of pediatric anxiety disorders, which are among the most prevalent and impairing mental health diagnosis in children and adolescents (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Thus, examining these patterns of environment-individual interactions in clinical samples may clarify whether trajectories of risk in a clinic-referred sample mirrors those observed in community settings.

Adverse familial environments increase risk for detrimental and lasting psychological effects, predicting psychopathology and disability well into adulthood (Brooks-Gunn & Duncan, 1997; Evans, 2003; Kohn, Dohrenwend, Mirotznik, & Dohrenwend, 1998; Wadsworth & Achenbach, 2005). Children raised in low socioeconomic status (SES) households are at increased risk for mental health problems (Wadsworth & Achenbach, 2005), with a two-fold increase in psychiatric diagnoses among low versus the highest SES groups (Kohn et al., 1998). In addition, existing research supports the importance of low family income and associated risk factors including low parental education and single parent status as risk factors for higher rates of childhood psychopathology (Hetherington et al., 1998) and pediatric internalizing disorders more specifically (Xue et al., 2005). Further, parent mental health problems increase risk directly and indirectly for children's mental health problems. The impact of parent mental health problems on risk for pediatric anxiety is purported to operate through biological, behavioral, and environmental channels (Goodman & Gotlib, 1999) including the transmission of a genetic predispositions for psychopathology, exposure to impairments in parental functioning, and detriments in caregiving behaviors (van der Bruggen, Stams, & Bogels, 2008). However, to date the impact of these family risk factors, as measured using a cumulative risk index, has only been examined in pediatric community samples. Therefore, it is also important to consider the influence of family adversity on clinically elevated anxiety symptoms, within the context of a treatment-referred sample. Drawing from the adult literature, research demonstrates that socioeconomically distressed adults with panic disorder demonstrate poorer treatment outcomes, indicating that socioeconomic adversity may interfere with treatment (Roy-Byrne et al., 2006). However, less is known about how specific exposure to familial adversity in childhood may impact the course of treatment for children with diagnosed anxiety disorders. Therefore, this study examined a treatment-referred sample of youth enrolled in a clinical trial for the treatment of pediatric anxiety.

Because familial adversity factors (i.e., socioeconomic strain, parent psychopathology, etc.) often co-exist, amplify in effect, and appear to carry a bioecological burden, indices such as Adverse Childhood Experiences Study (ACES; Felitti et al., 1998; Luthar et al., 2015) tabulate the accumulation of stressful childhood experiences that foster risk for psychiatric symptoms and disability in adulthood. In childhood cumulative risk indices are parallel methods for quantifying exposure to adversity factors by modeling aggregated exposure to adverse familial contexts (Conger & Elder, 1994; Evans, 2003). Specifically, cumulative risk models reflect the amount of exposure to familial stress across adverse contexts including socioeconomic stress, low parent education, parent mental health problems, family composition, and exposure to environmental risk (Evans & Whipple, 2013). Moreover, cumulative risk predicts increased risk for adjustment problems and psychopathology across childhood and into adulthood (Evans & Cassells, 2014). However, few studies have tested cumulative risk in predicting pediatric anxiety (see Evans & Cassells, 2015 for review) as an outcome, let alone how children experiencing cumulative risk may respond during treatment. Rather, these variables have been examined as separate risk factors for the development of internalizing disorders in community samples. This study will build on existing models of developmental psychopathology to test the effect of cumulative family risk on trajectories of anxiety symptoms in a clinic-referred sample during the course of treatment.

Children's Differential Responding to Family Adversity

Concurrently, individual-level vulnerabilities including negative affectivity and coping efficacy may heighten risk for pediatric anxiety disorders, particularly in response to experiences of adversity and stress (Muris & Ollendick, 2005; Watson & Clark, 1984). More specifically, negative affectivity reflects a susceptibility to react to stress with a high degree of distress and may render individuals more sensitive to the effect of negative experiences (Krueger & Markon, 2006; Watson, Gamez, & Simms, 2005). Similarly, perceived inability to cope in the face of stressors increases risk for childhood psychopathology more generally (Compas et al., 2001; Lengua et al., 1999; Zimmer-Gembeck & Skinner, 2011) and pediatric anxiety more specifically (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008; Muris & van der Heide, 2006). Therefore, individual differences in psychosocial characteristics may be important in predicting the trajectory of anxiety symptoms over the course of treatment for children in high risk families.

Negative affectivity has been defined as individual differences in emotional arousal and expression in childhood and may be conceptualized within a temperament framework (Rothbart & Bates, 2006). Temperamental characteristics reflect a predisposed range of emotional and behavioral responses and engage in a reciprocal interaction in shaping and being influenced by development (Gray, 1991). Further, temperament encompasses dispositional tendencies under relevant environmental conditions (e.g., a fear response to novelty) and need not be expressed continuously. Temperamental reactivity includes individual differences in reactions to stimuli (internal and external) including variation in onset, intensity, and duration of responses (Rothbart, 1989; Rothbart & Bates, 2006) and has been defined as emotionality with a focus on negative affectivity or negative emotionality (Rothbart, Ahadi, Hershey, & Fisher, 2001). Negative affectivity is a non-specific risk factor often conceptualized as a key underlying component of internalizing disorders. Based on the seminal work of Watson and Clark, the tripartite model of internalizing disorders supports negative affectivity as a common substrate of anxiety and depressive disorders (Clark & Watson, 1991). Further, research evidence linking negative emotionality in childhood and anxiety disorders is quite robust (see Garber & Weersing, 2010 for a review) highlighting a temperamental risk factor that may exacerbate risk for pediatric anxiety. Concurrently, emerging research in adulthood suggests that temperamental negative affectivity is an important individual characteristic in clinic-referred samples, as adults with high negative affect exhibited better responses to medication treatment with drugs targeting serotonin transmission (Gerra et al., 2014). While less is known about how negative affectivity may relate to anxiety symptoms across time in clinic-referred pediatric samples, theoretical frameworks for cognitive behavioral therapies (CBT) for anxiety disorders suggest that treatment components targeting arousal (i.e., relaxation) and behavioral responses (e.g., graduated exposure) may address core substrates related to negative affectivity. Thus, examining baseline negative affectivity may be important in predicting treatment response and tailoring intervention methods to address this core vulnerability for internalizing disorders.

Additionally, anxious children tend to overestimate the likelihood and severity of threatening situations and underestimate their ability to effectively cope in these situations (Beck, 2005;

Bögels, & Zigterman, 2000). This tendency for lower coping efficacy increases vulnerability for perceived deficits in one's ability to cope with current, past, or future stressors and has been found to mediate children's use of active coping strategies (Sandler, Tein, Mehta, Wolchik, & Ayers, 2000). Coping efficacy differs from coping strategies and behaviors, which are defined as thoughts and behaviors that are intended to reduce negative affect (including stress, anxiety, and depression; Compas et al., 2001; Lazarus & Folkman, 1984). Efficacious coping strategies may include cognitive (e.g., problem solving, rumination, wishful thinking), behavioral (e.g., avoidance, escape), and physiological (e.g., relaxation) strategies, and can all be effective in the face of stress (Compas et al., 2001; Kaplow, Gipson, Horwitz, Burch, & King, 2014; Prins & Ollendick 2003). Notably, children with anxiety disorders engage in less efficacious coping and report perceptions of reduced coping efficacy, particularly during anxiety-provoking situations (Hogendoom et al., 2014; Kendall et al., 2016; Lau et al., 2010; Mathews, Koehn, Abtahi, & Kerns, 2016; Prins & Ollendick, 2003). Within the context of treatment studies, CBT has been found to lead to positive changes in coping and positive thoughts in anxious children (Chu & Harrison, 2007; Kendall, Hudson, Flannery, & Suveg, 2008; Prins & Ollendick, 2003). Still, it is important to consider factors that may hinder treatment benefits. In the context of other treatment studies, less efficacious coping predicts longer time to recovery from depression regardless of type of intervention (Rohde, Seeley, Kaufman, Clarke, & Stice, 2008). Conversely, actual or perceived use of ineffective coping strategies may be problematic while efficacious coping may mitigate the impact of chronic stress, parental depression, exposure to violence, and socioeconomic disadvantage (Conger & Conger, 2002; Evans & Kim, 2013; Langrock et al., 2002; Mohammad, Shapiro, Wainwright, & Carter, 2015; Wadsworth & Compas, 2002).

Given the importance of coping in predicting response to treatment, this study examined how youth's baseline coping efficacy may serve as a resource in the face of ongoing stress (Sandler et al., 2000). We are not aware of prior work that has tested the interplay of children's coping efficacy and experiences of family cumulative risk in predicting the trajectory of anxiety symptoms in the context of treatment studies or clinic-referred samples. Because coping efficacy is important in predicting treatment response (see Kendall et al., 2016), interventions may be refined by understanding the interaction of familial risk factors and children's coping efficacy in predicting the trajectory of anxiety symptoms within treatment settings.

Present Study

The present study extends existing bioecological models of pediatric anxiety disorders and complements existing intervention research by examining the diathesis-stress link between experiences of familial adversity and children's individual-level characteristics in predicting the course of anxiety symptoms across a 12-week randomized clinical trial for pediatric anxiety disorders. Testing how children's pre-treatment negative emotionality and coping efficacy moderate responses to familial cumulative risk in the context of a clinic-referred sample for pediatric anxiety disorders, will facilitate our understanding of children and adolescents' susceptibility to stress. This may be particularly important in the context of treatment studies, as clarification of individual and contextual factors that predict intervention response is important for tailoring treatment strategies and increasing efficacy

(Sheeber & McDevitt, 1997). It was predicted that cumulative risk would reduce treatment response, as demonstrated by an attenuated slope in anxiety across treatment for children in high risk families. In addition, we expected that children's negative affectivity and anxious coping would exacerbate responses to cumulative risk, and in turn hinder improvements in symptoms. Lastly, we explored whether the findings differed by treatment condition, as parental factors may differentially impact children's anxiety symptoms in across treatment conditions (e.g., Gonzalez et al., 2014).

Method

Participants

Participants included 488 youth ages 7 to 17 (mean age 10.7; SD = 2.8 years) recruited as part of the Child and Adolescent Multimodal Treatment Study (CAMS) across six sites nationwide (for details see Compton et al., 2010 for design, Kendall et al., 2010 for sample characteristics, and Walkup et al., 2008 for primary intervention outcomes) and their primary parent or caregiver. Briefly, the study included an even number of boys and girls (49.6% female). Seventy-nine percent of the sample identified as Caucasian, with 9% of participants identifying as African American, 3% as Asian or Pacific Islander, 1% as American Indian, and 8% as Other. Twelve percent of the sample identified as Hispanic, and 21.1% of families were considered low socioeconomic according to the Hollingshead socioeconomic status index (occupation + education; Hollingshead, 1957; score of 1–3 on a 5-point scale). Most participants (86%) were living in two parent households headed by biological, adoptive, or step parents. All participants met DSM-IV criteria for a primary diagnosis of Social Phobia, Generalized Anxiety Disorder, and/or Separation Anxiety. Of the 488 youth randomized to treatment, 11 (2.3%) stopped treatment but were included in the assessments (treatment withdrawals); 46 (9.4%) stopped both treatment and assessments (study withdrawals; Walkup et al., 2008).

Procedures

All participants and at least one parent/legal guardian provided informed consent/assent. The institutional review board at each site approved and monitored the protocol. Safety monitoring was performed quarterly by a NIMH Data Safety and Monitoring Board. Participants were randomized to twelve weeks of cognitive behavior therapy (CBT; n=139), sertraline (SRT; n=133), combination CBT/SRT (COMB; n=140), or pill placebo (PBO; n=76). SRT and PBO were double-masked conditions, while COMB and CBT were masked to independent evaluators (IEs) but not to patients and therapists. Participants in the placebo condition were retained for all analyses, as the aim of the current study was to extend diathesis-stress models of cumulative risk to youth referred for treatment of pediatric anxiety disorders. Therefore, some participants did not receive active treatment during the 12-week study timeframe. All youth were assessed by IEs masked to study condition at weeks 0 (baseline), 4, 8, and 12 (post-acute treatment). Evaluations included collection of demographic data, anxiety and comorbid symptomatology, and psychosocial functioning measured via IEs and parent- and child-report. Efforts were made to maintain IE continuity over time and treatment masking was rigorously enforced.

Measures

Cumulative Risk.—All cumulative risk factors were measured at the baseline assessment, included seven risk factors measured at the start of the study assessing a variety of domains, including: socioeconomic (parental education, parental employment status, single parent, and nontraditional households), parental (being born to an adolescent parent, current and historical parental psychopathology) and developmental (low birth weight) risk. These factors were selected, as they represent domains of risk commonly included in cumulative risk indices. To create a risk index dichotomous risk factors (i.e., low parental education, parental unemployment, single parent status, being born to an adolescent parent, and low birth weight) were scored as 0=not present or 1=present. In addition, exposure to familial psychiatric illness was aggregated across diagnoses, with higher scores reflecting increased burden and exposure to mental illness (Range = 0 to 5). Lastly, continuous risk factors (e.g., parental psychopathology symptoms) were converted into proportions of the total possible score, rescaling each variable from 0 to 1 to facilitate equal weighting across all risk indicators. The total cumulative risk index was the sum of all component factors. Cumulative risk scores ranged from 0 to 10.13 with a mean of 1.3.

Low parental education was defined as having one parent with less than a high school education (n = 29, 6.1%). Overall, the sample was relatively well-educated with the modal level of parental education at or above a college degree. Parental employment status was classified as a risk factor if the head of household (n = 28, 5.9%) or spouse (n = 78, 21.4%) were unemployed at the baseline of the study. Lastly, single-parent status and nontraditional households (e.g., living with a non-parental family member or non-relative) were relatively infrequent housing arrangements (n = 70, 14.4%) and therefore combined into a single risk indicator. Given the study design, information about current familial income was not available. As noted above, Hollingshead socioeconomic indicators were available for all participants. However, because variables used in the Hollingshead index overlap with the risk factors outline above (i.e., employment status), the measure was not included as an indicator of socioeconomic risk to prevent redundancy.

Risk factors capturing parental risk included being born to an adolescent parent, which was defined as being born to a mother age 19 or younger (n = 15, 3.1%). In addition, we examined parental mental health, both current and historical. The *Brief Symptom Inventory* (BSI; Derogatis & Melisaratos; 1983), a 53-item self-report measure of psychopathology and was used to indicate current mental health symptoms for the parent participating in the study. For the present analyses, the Global Severity Index (BSI–GSI) provided a single composite score of current symptoms of somatization, obsessive–compulsive disorder, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychosis. Higher scores suggest greater severity of psychological distress. Internal consistency was .95 at pre- and post-treatment assessments. Participating parents also provided information on current and historical psychiatric diagnoses for the two relatives closest in relation to the participant and were scored as 1 = yes, 0 = no. Because a higher number of psychiatric diagnoses may reflect additional exposure to risk, the number of psychiatric diagnoses were summed across relatives for use in the cumulative risk index (M = .33 diagnoses, Range = 0 to 5).

We further included low birth weight as a potential proxy indicator for in utero exposure to risk and lack of prenatal care. Low birth weight (i.e., weighing less than 5.5 lbs at birth) is a multi-determined risk factor that may reflect access to prenatal care, maternal nutrition, pregnancy complications, and genetic or congenital risk. Further, low birth weight predicts risk for developmental, health, and psychiatric symptoms across the lifespan (da Silva, 2010; Kramer, 1987). Seven percent of children were reported to have weighed less than 5.5 pounds at birth and were therefore indicated as developmentally at risk.

Negative Affectivity.—At baseline, children's temperamental negative affect was assessed using *Negative Affect Self-Statement Questionnaire* (NASSQ; Ronan, Kendall, & Rowe, 1994; Sood & Kendall, 2007). The NASSQ is a 70-item child report measure of specific self-statements (rated on a 5-point scale) associated with depression, anxiety, and negative affect. For this study, only the 10-item Negative Affect subscale was used. The scale was developed to assess the overall level of negative affect common to depression and anxiety. Items on the Negative Affect scale include, "Just my luck – it went wrong", "Why did it have to happen to me?", and "All good things will happen". Youth rated how often each statement came to mind in the past week on a scale ranging from *1=not at all to 5=all of the time*. The questionnaire consists of separate items for younger and older children and, for its initial and cross-validation samples, possesses strong internal consistency. In this study, the NASSQ was only administered to children ages 11–18, resulting in a subsample for the study analyses (n = 222).

Coping Efficacy.—At baseline, children's perceived ability to cope with anxiety-provoking situations were assessed using the Coping Questionnaire, Child version (CQ-C; Kendall, 1994; Kendall et al., 2016). The CQ-C is a 7-item instrument that assesses children's perceptions about managing personally identified anxiety-provoking situations. Prior to completing the measure, trained experimenters explained the task and offered sample items. Once the child understands the measure, the examiner selects three anxiety-provoking scenarios from the CQ-C battery (e.g., giving a speech) that align with the child's self-identified fears during a prior clinical interview. Children were then asked to rate themselves on how well they would be able to cope (i.e., help themselves feel "less upset") during each hypothetical situation using a response range from "not at all able to help myself" (1) to "completely able to help myself feel comfortable" (7). All children in the sample completed the CQ-C. Therefore, analyses including children's coping as a predictor used the entire sample (N = 488).

Anxiety Symptoms.—The *Pediatric Anxiety Rating Scale* (PARS; RUPP Anxiety Group, 2002) is an interviewer-rated measure of anxiety severity that integrates youth and parent reports of youth anxiety. The PARS was employed as the primary continuous outcome measure and was completed by masked independent evaluators at baseline, 4-, 8-, and 12-weeks. Total scores were obtained by summing six items assessing anxiety severity, frequency, distress, avoidance, and interference over the previous week. Total scores range from 0–30, with scores above 13 indicating clinically meaningful anxiety. The PARS has acceptable internal consistency ($\alpha = 0.64$), strong inter-rater reliability ($r = 0.97$), and moderate retest reliability ($r = 0.55$). In the current sample, the PARS demonstrated

acceptable internal reliability at each time point (Baseline α = .77; Week 4 α = .81; Week 8 α = .82; Week 12 α = .87).

Covariates.—Research has established gender differences in the prevalence of anxiety symptoms emerging as early as school age (Costello, Compton, Keller, & Angold, 2003). In addition, developmental differences exist in the emergence and severity of anxiety, as well as a high co-occurrence between anxiety and depression symptoms in childhood (Garber & Weersing, 2010). Therefore, child sex, child age, and baseline child depression symptoms were examined as potential covariates prior to testing the study hypotheses. Baseline children's *depression symptoms* were assessed using the Mood and Feelings Questionnaire, child-report (MFQ-C; Wood, Kroll, Moore, & Harrington, 1995). The MFQ measures depression symptoms across the previous two weeks on 33 youth-reported items assessing depressive symptomatology in children and adolescents. In this sample, the MFQ-C had excellent internal consistency with Cronbach α = .92. Although the rate of attrition was low (see above; n = 57), premature termination was included as a covariate to account for the potential effect of attrition on the study findings.

Analytic Plan

Analyses used the CAMS intent-to-treat sample (N=488) with established multiple imputation procedures for missing data (see Walkup et al., 2008). Because this study represents the first examination of these cumulative risk x individual risk interactions in youth seeking treatment for pediatric anxiety disorders, we considered both the role of treatment modality (CBT only, medication only, combined CBT and medication, and placebo) and inclusion in the study (i.e., all participants regardless of treatment modality) for all analyses. However, we also explored whether treatment modality affected the pattern of findings, using multiple group analysis. Families were included if data was available from at least one time point and missing data was estimated using Full Information Maximum Likelihood Estimation (FIMLE), which simultaneously utilizes all available estimations of means and intercepts, covariances and path coefficients to calculate parameter estimates (Arbuckle, 1996). Using Latent Growth Curve Modeling (LGCM), initial unconditioned growth models were specified to examine trajectories of changes in anxiety across the active treatment period (baseline to week 12) and tested using MPlus (Version 7; Muthén & Muthén, 2012). After evaluating variability in initial anxiety levels and changes across treatment, conditioned growth models to test how exposure to cumulative risk, negative affectivity, coping efficacy, and their interaction (cumulative risk x negative affectivity; cumulative risk x coping efficacy) at baseline predicted initial levels and changes in anxiety symptoms across active treatment (baseline to week 12) were estimated. Because LGCM considers change over time, all interaction models tested 3-way interactions (cumulative risk by negative affectivity/coping efficacy by time). Significant interactions were probed using methods consistent with the recommendations of Preacher, Curran, and Bauer (2006). Comparison of the results across treatment modalities were conducted using multiple group analysis and estimated in MPlus (Muthén & Muthén, 2012). A series of nested multi-group models were specified with four identified groups (1) CBT + medication (2) medication only, (3) CBT only, and (4) placebo. A Satorra-Bentler chi-square difference test was used to examine equivalency across models (Satorra & Bentler, 2010). In the first multi-group

model, all variable paths were constrained to be equivalent across groups. A subsequent model allowed all paths to vary by treatment condition.

Results

Correlations among Study Variables

Descriptive statistics and correlations among all variables are presented in Tables 1 and 2. Baseline cumulative risk at intake was not related to changes in symptoms across treatment. However at intake, youth perceptions of lower coping efficacy was associated with higher cumulative risk. In addition, baseline negative affectivity and coping efficacy were each related with symptoms across treatment, indicating they may be plausible predictors of changes in anxiety. As expected modest and negative associations among symptoms were observed, suggesting that youth reported declines in anxiety across 12-weeks.

Tests of Changes in Symptoms Across Treatment

Consistent with the assumptions of LGCM, growth factors in child anxiety symptoms were first examined without predictors to examine variability in levels and changes in independent evaluator ratings of child anxiety across 12 weeks. Using FIMLE in Mplus Version 7 (Muthén & Muthén, 2012), unconditioned growth models were specified in which factor loadings were set to define the intercept as levels of symptoms at baseline and the slope as linear change across weeks 4, 8, and 12. The model indicated that on average, symptoms decreased (Intercept $b = 19.078$, $p < .001$; Slope $b = -3.645$, $p < .001$; χ^2 (2) = 7.679, $p = .02$, CFI = .99, RMSEA = .08, $p = .17$) across the 12-week active treatment period, for all participants. Still, there was significant variability in intercept ($b = 8.41$, $p = .03$) and the slope of anxiety ($b = 2.94$, $p < .001$), indicating variability in the rate of change across active treatment and highlighting the potential for exogenous predictors in explaining children's treatment-related symptom trajectories.

Covariates as Predictors of Treatment Response

To retain power for the main study hypotheses, a covariate model was tested to evaluate whether child sex, child age, baseline depression symptoms, and attrition (i.e., premature termination from treatment) predicted initial levels or changes in anxiety. A conditioned latent growth model was estimated in which all covariates were entered as simultaneous predictors of initial levels and changes in evaluator-rated anxiety symptoms. The model provided adequate fit, χ^2 (10) = 21.17, $p = .02$, CFI = .98, RMSEA = .05, $p = .51$, and demonstrated that child age, depression symptoms, and premature termination from the study predicted initial levels of anxiety, but not changes across 12-weeks (age $b_{int} = .16$, $p = .02$, $b_{slo} = .03$, $p = .44$; depression $b_{int} = .06$, $p < .001$, $b_{slo} = -.01$, $p = .26$; premature termination $b_{int} = 2.01$, $p < .03$, $b_{slo} = .63$, $p = .25$). Based on model results, these covariates were retained for subsequent tests of the hypotheses. Child sex was dropped from additional analyses as it did not predict initial levels or changes in anxiety ($b_{int} = -.39$, $p < .28$, $b_{slo} = .34$, $p = .13$).

The Interaction of Cumulative and Individual Risk

To test whether the effect of cumulative risk would be moderated by children's negative affectivity and/or coping efficacy two latent growth curve models were specified in which cumulative risk, children's baseline negative affectivity or coping efficacy, and the multiplicative of these variables were entered as predictors of initial levels and changes in anxiety symptoms from baseline to week 12. Family cumulative risk and each child characteristic (negative affectivity and coping) were separately mean centered and then used to create the interaction terms (Curran, Bauer & Willoughby, 2004). The results are presented in Table 3.

Cumulative Risk and Negative Affectivity.—Children's self-reported negative affectivity, but not cumulative risk, predicted initial levels of anxiety symptoms (see Table 3; Model Fit, χ^2 (15) = 36.82, $p < .01$, CFI = .96, RMSEA = .08, $p = .06$). This direct effect was above the variance explained by child age, premature termination from the study, and children's baseline depression symptoms at intake. Conversely, the main effects for negative affectivity, familial cumulative risk, and the interaction of these terms was not significant in predicting the slope of anxiety symptoms across 12-weeks.

Cumulative Risk and Coping Efficacy.—With regard to children's coping efficacy (see Table 3; Model Fit, χ^2 (14) = 28.61, $p = .01$, CFI = .98, RMSEA = .05, $p = .57$), lower coping efficacy was associated with higher anxiety symptoms at baseline and weaker declines in evaluator-rated symptoms across the 12-week acute treatment phase of the study. However, this main effect was subsumed within a significant interaction between cumulative risk and children's coping efficacy. As shown in Figure 2, youth experiencing either high levels of familial cumulative risk or lower coping efficacy exhibited attenuated declines in anxiety symptoms (High CR x High Coping Efficacy, $b = -4.00$, $p < .0001$; Low CR x Low Coping Efficacy $b = -3.10$, $p < .001$; High CR x Lower Coping Efficacy, $b = -3.96$, $p < .001$). Conversely, anxiety symptoms decreased faster across treatment for youth exposed to low risk contexts and those that reported higher coping efficacy ($b = -4.36$, $p < .001$).

Comparison Across Treatment Conditions—To test whether the pattern of findings varied across treatment conditions, multi-group latent growth curve models were tested in a nested fashion as described above. In the first model, all paths were constrained to be equivalent across treatment modalities.

Cumulative Risk x Negative Affectivity: First we tested a restricted model of the interaction of cumulative risk and negative affectivity, which resulted in a reduction to model fit (χ^2 (108) = 173.43, $p < .001$, RMSEA = .10, $p < .001$, CFI = .84) from the single group analyses reported above. Secondly, an unrestricted model in which paths were free to vary across groups was estimated. The unrestricted model demonstrated a similar fit (χ^2 (76) = 130.66, $p < .001$, RMSEA = .11, $p < .001$, CFI = .86). To test for multi-group equivalence, a Satorra-Bentler chi-square difference test was conducted and yielded a non-significant result (χ^2 (32) = 41.70, $p = .18$).

Cumulative Risk x Coping Efficacy: In a parallel fashion, we first tested a restricted model of the interaction of cumulative risk and coping efficacy, which again resulted in a reduced model fit (χ^2 (86) = 149.81, $p < .001$, RMSEA = .08, $p = .02$, CFI = .91) from the larger analysis presented above. Secondly, an unrestricted model in which paths were free to vary across groups was estimated. The unrestricted model also demonstrated a similar model fit (χ^2 (64) = 118.21, $p < .001$, RMSEA = .08, $p < .01$, CFI = .93). To test for multi-group equivalence, a Satorra-Bentler chi-square difference test was conducted and yielded a non-significant result (χ^2 (22) = 32.12, $p = .08$). In both sets of analyses, the restricted models were retained suggesting that the pattern of findings did not differ across treatment assignment.

Discussion

The present findings support the unique contributions of familial and individual-level vulnerabilities in predicting changes in anxiety symptoms for youth seeking treatment for clinically impairing anxiety. The diathesis-stress interaction between cumulative risk and coping efficacy indicated that high risk and perceptions of lower coping efficacy attenuated declines in anxiety across treatment. Thus, the results indicate that tailoring anxiety interventions to target familial stressors and coping efficacy may be important in maximizing treatment outcomes. The findings extend prior models of differential responsiveness by applying a diathesis-stress framework to predicting symptom changes within a clinic-referred sample. This more nuanced approach deconstructs children's rate of improvement in symptoms as well as identifying which factors may shape the course of symptom decline.

We examined the interaction of familial adversity, as measured by cumulative risk, and two individual vulnerabilities for pediatric anxiety: negative affectivity and coping efficacy in predicting the decline of anxiety symptoms across a 12-week double-masked clinical trial. Previous research has demonstrated that the accumulation of familial adversity factors such as socioeconomic risk and parent psychiatric history increase risk for pediatric psychopathology, perhaps by taxing stress response systems with repeated exposure to harmful and negative environments (Loman & Gunnar, 2011). Further, the impact of cumulative risk and stress may be compounded by individual vulnerabilities (Rothbart & Bates, 2006). This study presents the first investigation, to our knowledge, into the joint impact of cumulative risk and children's characteristics in shaping changes in anxiety symptoms in a treatment seeking sample, thereby extending models of developmental psychopathology into clinical care settings.

In an effort to improve and tailor treatment approaches based on patient and familial characteristics, we tested a differential responsiveness model of cumulative risk and children's negative affectivity and coping efficacy in predicting rates of symptom improvements. This approach tested a diathesis-stress model of cumulative risk and children's characteristics. The results demonstrated that while the accumulation of high familial risk did not directly predict improvements in functioning, when considered in combination with individual vulnerabilities for anxiety disorders more nuanced patterns of symptom decline emerged. Thus, it appears that clinicians and researchers should consider

the importance of children's characteristics (negative affectivity and perceived coping efficacy) as important predictors and qualifiers in formulating and tailoring treatment of pediatric anxiety disorders.

In line with expectations, children reporting higher negative affectivity were also more anxious at the start of the study. This finding replicates prior work as high emotionality and reactivity represented direct temperamental vulnerabilities for pediatric psychopathology (Nigg, 2006). Notably, the effect was above the association of children's depression symptoms, suggesting some unique association between high negative affectivity and children's anxiety symptoms. With regard to other direct effect associations, cumulative risk did not directly predict children's anxiety at intake or across the study. These findings suggest that in a clinic-referred sample, the accumulation of adverse family factors may not directly affect children's baseline anxiety. Perhaps because the relative variability of cumulative risk in this sample was low. However, because broader research in community samples demonstrates the deleterious impact of familial adversity in heightening risk for psychopathology across development (Evans & Cassells, 2014) further investigation is warranted to better understand how clinicians and providers may need to consider the impact of cumulative risk in shaping the course of treatment for clinic-referred youth. Future research may benefit from considering that heightened family stress may predict higher rates of comorbid diagnoses or exposure to trauma, which may complicate and potentially hinder treatment planning and progress.

In our test of the interaction between cumulative risk and children's perceived coping efficacy, a diathesis-stress interaction was supported, suggesting that the combined effect of familial adversity and inability to cope were important joint predictors of treatment response. More specifically, children's endorsement of poor coping efficacy in stressful or anxiety-provoking settings predicted greater impairment (i.e., higher symptoms) at the start of treatment and slower declines in symptoms across 12-weeks. However, this effect was subsumed within an interaction with cumulative risk. The pattern of findings was such that for children experiencing higher levels of family risk, lower coping efficacy hindered declines in symptoms. Thus, children who tended to perceive themselves as less able to cope with anxiety might be more susceptible to chronic exposure to familial risk factors. Further, the accumulation of risk may tax children's coping capacity by repeatedly calling for youth to employ automatic coping strategies in response to experiences of socioeconomic risk, parent mental health problems, and associated factors. In turn, exposure to broader family-level risk factors and stressors and the reinforcement of anxious coping strategies may hinder the children's perceived ability to adaptively cope (Sandler et al., 2000). This parallels findings from depression research in which ineffective coping strategies hindered treatment response for adolescents (Rohde et al., 2008). In addition, previous research with this sample demonstrated that when parents report high levels of anxiety, children receiving medication treatment exhibited faster treatment responses (Gonzalez et al., 2014). However, this study did not support differences in the rate of treatment response across intervention types. Instead, it appears important to directly target familial stressors and coping efficacy as an adjunct to treatment.

This study includes several notable strengths including the use of a large, double masked clinical trial across multiple sites, and a rigorous study design. In addition, cumulative risk was modeled from a number of risk factors including socioeconomic, parental, and child-level variables. This approach allowed for a comprehensive assessment of risk. Further, masked interviewer-ratings were used to measure children's anxiety symptoms across the study reducing bias and minimizing effects due to shared variance from reporters. This study also included several limitations that cannot be overlooked. These include the reduced sample size for models examining negative affectivity, as the measure used was not available for children under the age of 11. Our assessment of coping was limited to children's perceived coping efficacy and may have differed if actual coping strategies in times of stress were examined. Additionally, these results cannot speak to cumulative risk diathesis-stress interactions in the context of response to treatment for pediatric anxiety, as 16% of participants were randomized to placebo care. However, our initial findings suggest that the inclusion of participants in the placebo condition did not inflate the results, as the pattern of findings did not differ by treatment condition. This study represents an important initial step in testing the impact of cumulative risk by individual risk interactions in youth referred for clinically elevated anxiety. Still, the range of endorsed adversity factors were restricted by the use of a treatment-seeking sample. Thus, additional work is needed to understand how these patterns impact children's clinical outcomes in samples with greater diversity in experience, background, and familial risk. Lastly, the measurement of cumulative risk was limited to the available risk factors measured in the CAMS study and did not include direct measures of socioeconomic status including family income or household density, which may limit the accuracy of our cumulative risk measure.

Nonetheless, this study presents a novel approach to modeling factors important in predicting how children may respond to treatment and enhances our clinical approaches by facilitating the tailoring of treatments. More specifically, for children high in negative affectivity the findings demonstrate that a focus on reactivity and emotionality may be particularly important in alleviating symptoms. Conversely, for children with lower coping efficacy the role of familial risk and exposure to stress may be important factors to emphasize during treatment. Thus, the current study supports the unique contributions of familial and individual-level vulnerabilities in predicting treatment-related symptom changes for youth with diagnosed anxiety disorders. Further, this research extends prior models of differential responsiveness by applying a diathesis-stress framework to predicting symptom changes in a clinic-referred sample. This more nuanced approach deconstructs children's rate of improvement in symptoms and identifying which factors may shape the course of symptom decline. Together these findings allow researchers and clinicians to better assess and understand the combination of factors that may affect treatment, thereby aiding in treatment planning and structure.

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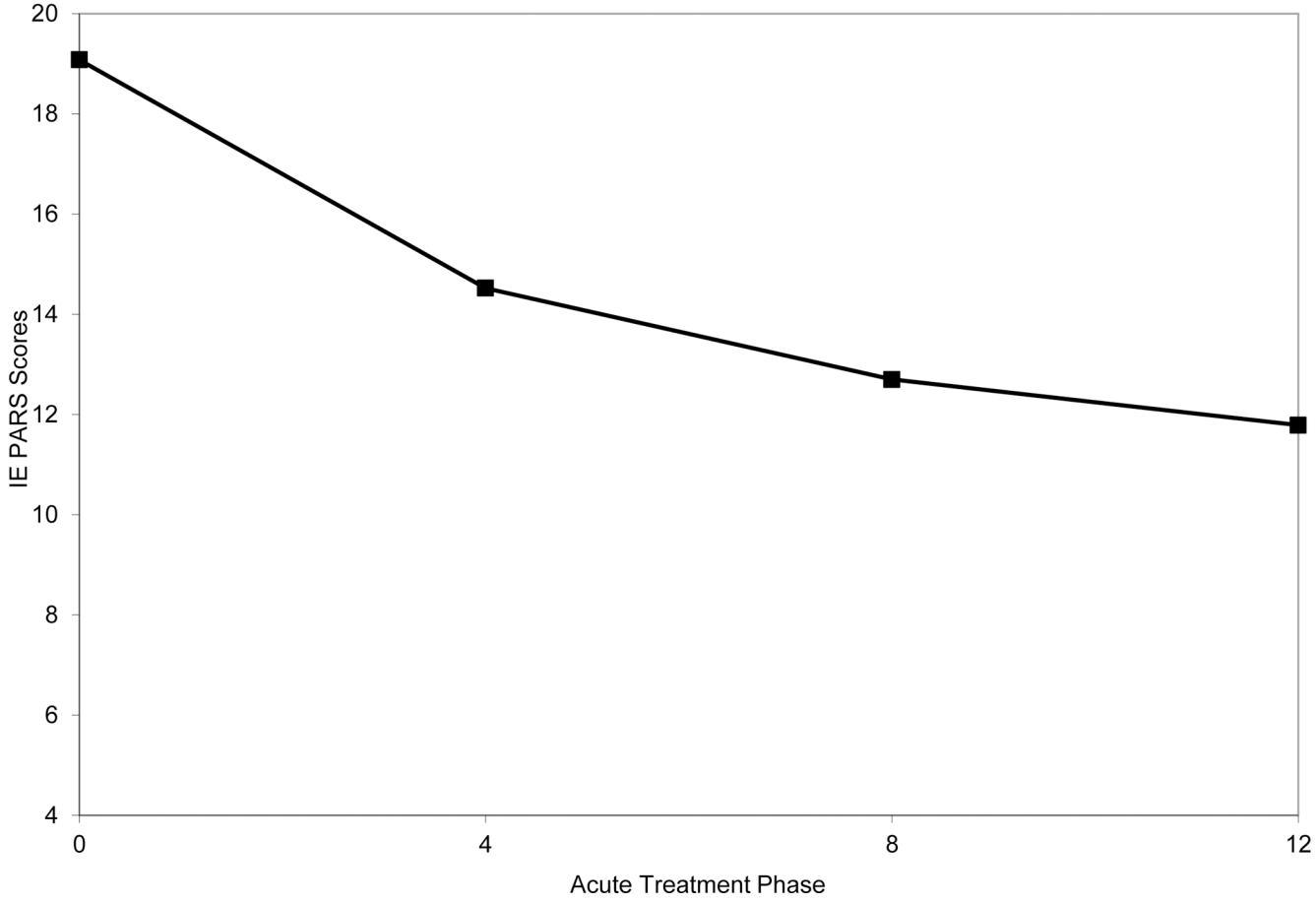

Figure 1.

A plot of the unconditioned change in anxiety symptoms across the acute treatment phase (12-weeks) for the intent to treat sample. Anxiety symptoms significantly decreased across treatment, however there was variability in the rate at which participants improved.

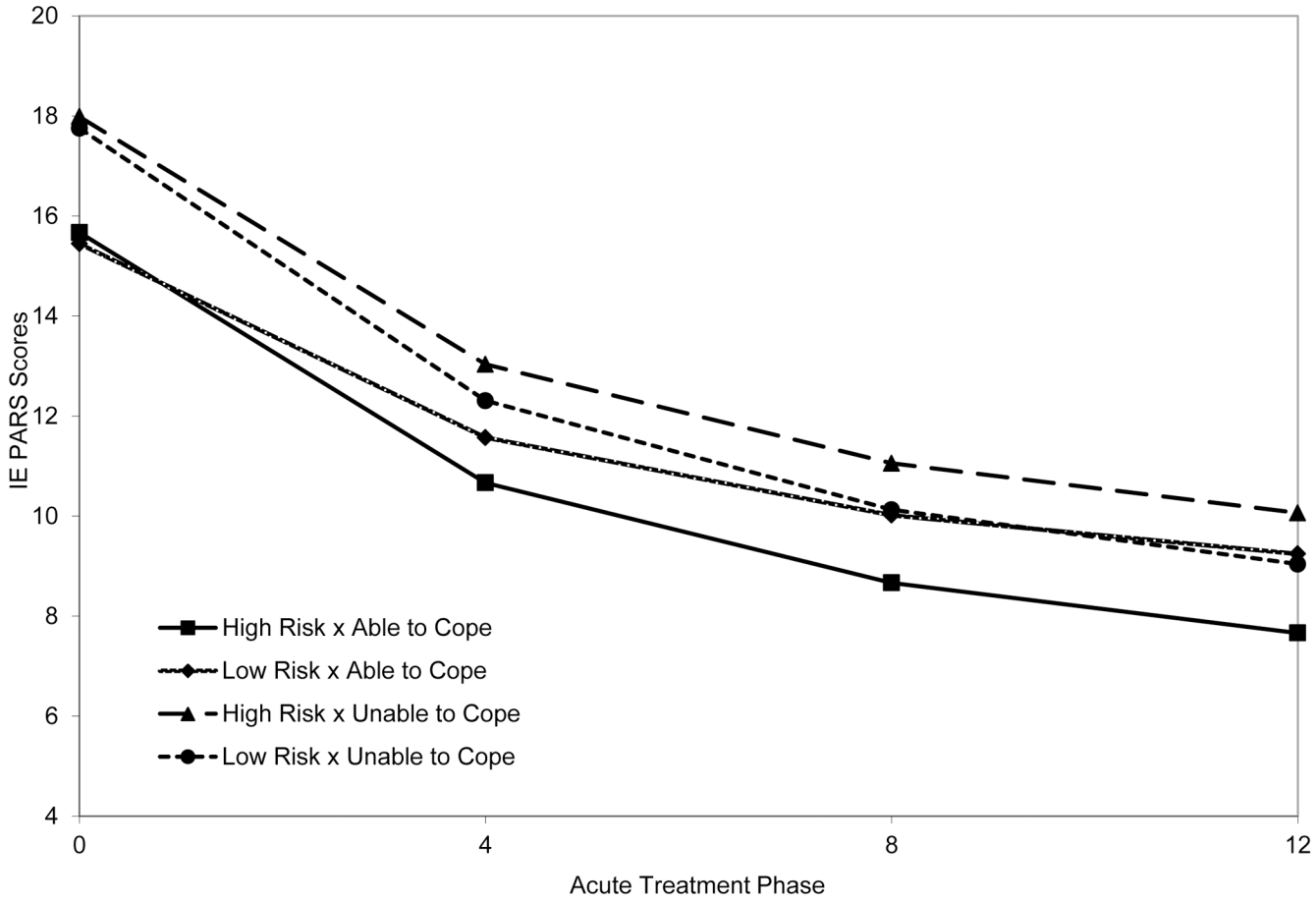

Figure 2.

A plot at 1 *SD* above and below the mean of cumulative risk and children's coping order to probe the observed interaction. The X-axis denotes the time in treatment across 12 weeks.

Table 1

Descriptive Statistics

Variable	*M*	*SD*	Range
Time 1 (Baseline)			
Child Age	10.69 yrs	2.81	7.00 – 17.00
Child Depression Symptoms	17.77	11.83	0.00 – 59
Cumulative Risk	1.33	1.47	0.00 – 10.13
Negative Affectivity	21.07	8.06	10.00 – 49.00
Coping Efficacy	3.21	1.39	1.00 – 7.00
Child Anxiety Symptoms	19.16	4.19	7.00 – 30.00
Time 2 (Week 4)			
Child Anxiety Symptoms	14.67	5.49	0.00 – 29.00
Time 3 (Week 8)			
Child Anxiety Symptoms	11.70	5.94	0.00 – 25.00
Time 4 (Week 12)			
Child Anxiety Symptoms	9.49	6.59	0.00 – 27.00

Table 2

Correlations among Study Variables

	2	3	4	5	6	7	8	9	10
1. Child Age	.16**	.009	−.026	.26**	−.04	.11*	.18*	.12*	.10*
2. Child Depression T1	--	.03	.08	.84**	−.24**	.19**	.09	.09	.07
3. Attrition		--	−.01	−.01	−.001	.09	.17*	.09	.12*
4. Cumulative Risk T1			--	.04	.098*	.00	−.03	−.007	−.06
5. Negative Affectivity T1				--	0.20*	.29**	.16*	.13	.11
6. Coping Efficacy T1					--	−.31**	−.17**	−.08	−.08
7. Child Anxiety T1						--	.47**	.37**	.34**
8. Child Anxiety T2							--	.66**	.495**
9. Child Anxiety T3								--	.67**
10. Child Anxiety T4									--

Table 3

Parameters from Conditioned Growth Models Testing Trajectories of Anxiety Symptoms

	Anxiety Growth Factors					
	Intercept Factor			Slope Factor		
	b	SE	β	b	SE	β
Negative Affectivity Model						
Child Age	.07	.14	.04	.03	.10	.03
Child Depression Symptoms T1	−.05	.04	−.19	.02	.03	.11
Attrition	−.61	1.38	−.04	1.38	.94	.13
Cumulative Risk	−.35†	.19	−.15	−.06	.13	−.04
Negative Affectivity	.20***	.06	.50	−.05	.04	−.20
Cumulative Risk x Negative Affectivity	.00	.03	−.01	−.01	.02	−.05
Coping Model						
Child Age	.15	.06	.14	.03	.04	.05
Child Depression Symptoms T1	.04*	.02	.14	−.01	.01	−.04
Attrition	1.997*	.87	.13	.81	.55	.09
Cumulative Risk	.08	.12	.04	−.09	.08	−.07
Coping Efficacy	−.83***	.13	−.38	.22**	.08	.17
Cumulative Risk x Coping Efficacy	−.001	.09	.00	−1.6**	.06	−.18

Note. The Negative Affect scale was only given to children 11 and older. Therefore, the sample size for the Negative Affect model was reduced to N = 222. The Anxious Coping Model used all participants from the intent to treat sample and included N = 488 participants.

† $p = .06$

* $p < .05$

** $p < .01$

*** $p < .001$