Child Youth Care Forum
https://doi.org/10.1007/s10566-017-9432-z

ORIGINAL PAPER

Family and Parent Predictors of Anxiety Disorder Onset in Offspring of Anxious Parents

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Abstract
Background Offspring of anxious parents are at increased risk for developing anxiety disorders. There is a need to identify which youth are at greatest risk for disorder onset in this population.
Objective This study prospectively examined several theory-based family and parent characteristics (e.g., family conflict, parental over-control, parental psychopathology) as predictors of anxiety disorder onset in children whose parents were clinically anxious.
Methods Families were enrolled in a randomized controlled trial evaluating a family-based preventative intervention, relative to an information monitoring control condition, for offspring of anxious parents (N = 136; child mean age 8.69 years; 55% female; 85% Caucasian). Family and parent measures were collected using multiple informants and an observational task at baseline, post-intervention, and at a 6 and 12 month follow-up. Child anxiety disorder diagnosis was determined by independent evaluators using the Anxiety Disorders Interview Schedule for Children.
Results Results indicated that none of the baseline family or parent variables examined predicted the onset of an anxiety disorder in children over the 1 year follow-up period.
Conclusions Findings raise questions about the short-term risk associated with family and parent factors in anxiety disorder development in this high risk population.

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Published online: 20 December 2017

Keywords Parental anxiety · Child anxiety · Prevention · Parenting · Family factors

Introduction

Pediatric anxiety disorders are among the most common psychiatric illnesses and are associated with numerous deleterious short and long-term consequences (Bittner et al. 2007; Costello et al. 2003). Etiological models of pediatric anxiety disorders implicate both genetic and environmental factors. With respect to genetic factors, meta-analyses of family aggregate studies conclude that offspring of anxious parents are approximately four times more likely to have an anxiety disorder relative to their peers whose parents do not have a psychiatric disorder (Micco et al. 2009). Despite the elevated risk, a large portion of these high risk youth do not develop anxiety disorders and data from twin studies suggest that only a modest amount ($\leq$ 30%) of the variance in anxiety disorders is attributed to genetic loading (Eley et al. 2015) leaving a large proportion of unexplained variance that is likely explained by environmental influences.

Parent and family characteristics are key environmental factors that appear to be associated with the development and maintenance of child anxiety. While findings from this literature are fraught with inconsistencies due to a host of methodological variations (see McLeod et al. 2007 for a meta-analysis) there is evidence that: (1) family environments, (2) specific parenting behaviors, and (3) parental distress associated with psychiatric symptoms are linked to elevated child anxiety symptoms. Specifically, data on family environments indicate that families characterized as high in conflict (Bögels and Brechman-Toussaint 2006) and external locus of control (Becker et al. 2010) have been associated with higher levels of child anxiety and/or the presence of child anxiety disorders. Similarly, several specific parenting behaviors, including parental over-control and overprotection, modeling of anxious behaviors, and reinforcing dependency or accommodation of child anxiety (i.e., allowing the child to avoid anxiety provoking situations) have all been associated with elevated child anxiety (see McLeod et al. 2007). Finally, higher levels of parent psychopathology (e.g., higher levels of global distress) have been associated with higher child anxiety (see Drake and Ginsburg 2012 for review).

Theoretically, these dimensions of family environments and parenting behaviors have been hypothesized to increase child anxiety through a number of potential pathways (Schleider and Weisz 2017). For instance, family environments and parenting behaviors characterized by greater control (and less granting of autonomy) over children's choices, decisions, and behaviors are theorized to undermine youths perceptions of self-competence, heighten their external locus of control, and restrict their ability to develop mastery skills that might reduce anxiety (Affrunti and Ginsburg 2012; Lebowitz et al. 2013). Consistent with social learning theory, parents' modeling of anxiety and their own expressions of distress are theorized to increase children's anxious cognitions and behaviors and ineffective coping (Burstein and Ginsburg 2010; de Rosnay et al. 2006; Gerull and Rapee 2002; Schleider et al. 2014).

Taken together, while there is evidence that these family environments and parenting behaviors are linked to elevated child anxiety symptoms and disorders, few of these factors have been examined prospectively to determine their unique contributions to the development of anxiety disorders in high risk offspring. Understanding whether and which of these factors increases the risk of youth developing disorders could refine current preventive interventions and improve lifelong outcomes for these at risk youth. To address

these issues, this study used a multi-informant and multi-method approach to examine these three theory-based domains of family and parent predictors: (1) family environment (conflict and external locus of control); (2) parenting behaviors (over-control/over protection, modeling of anxiety, reinforcement of child dependency), and (3) parental psychological distress. These proposed predictors of child *anxiety disorder onset* were assessed in the context of a randomized controlled trial evaluating the efficacy of the *Coping and Promoting Strength* (CAPS) intervention compared to an information-monitoring control condition (IM; see Ginsburg et al. 2015 for study details and CONSORT diagram). CAPS is an eight session family-based psychosocial preventive intervention based on cognitive behavioral strategies and designed to prevent the onset of anxiety disorders in the offspring of anxious parents. The intervention was not designed to treat parents' anxiety. Based on the extant literature it was hypothesized that anxiety disorder onset would be predicted by: (1) family environments high in conflict and external locus of control, (2) parental behaviors high in over-control and overprotection, modeling of anxious behaviors, and reinforcement of dependency, and (3) higher levels of parent psychopathology (i.e., global distress).

Methods

Participants

Participants were 136 parent–child dyads (79% mothers). All parents (mean age 40.79 years; $SD = 4.99$) were diagnosed with a DSM-IV anxiety disorder using the Anxiety Disorders Interview Schedule (Brown et al. 1994). The most common primary anxiety disorder of parents was generalized anxiety disorder (GAD; 69.12%). 63.97% of parents had a comorbid anxiety disorder. The most common secondary diagnoses were: social anxiety disorder (11.76%) and panic disorder (12.50%).

Youth participants did not meet criteria for a DSM-IV anxiety disorder at baseline. Children (55.88% female) were 6–13 years old ($M = 8.69$ years; $SD = 1.80$). The majority of youth (> 75%) were White, from two parent, upper income, and college educated families (Ginsburg et al. 2015 for full description of sample).

Procedures

Prior to data collection, the study was approved by the university's Institutional Review Board and written informed consent was obtained from all participants. Anxious parents were recruited via radio and print advertising, referrals from mental health providers, and word of mouth to participate in a study examining the impact of an anxiety prevention program for their children. Interested families completed a phone screen and if they appeared eligible, were invited to complete an in-person baseline evaluation. Eligible families (i.e., those with a parent meeting diagnostic criteria for a current primary anxiety disorder and a child who did not meet criteria for a current anxiety disorder) based on the baseline evaluation, were randomized (1:1) to CAPS ($n = 70$) or IM ($n = 66$). Follow-up evaluations (including the diagnostic interviews) were scheduled to occur 8 weeks post-baseline as well as at 6- and 12-month follow-ups. Children were assisted with completing the measures below by a trained research assistant as needed. Details of the CAPS

intervention and study methods and primary outcomes are in Ginsburg (2009) and Ginsburg et al. (2015) respectively.

Measures

Primary Outcome

The *Anxiety Disorders Interview Schedule for DSM-IV Child Version* (ADIS-C; Silverman and Albano 1996), the widely used gold standard diagnostic interview for anxiety disorders in youth, was used to determine anxiety disorder onset. The ADIC-C assesses a broad range of anxiety, mood, and externalizing disorders and was administered by trained and masked evaluators who assigned a Clinical Severity Rating (CSR) for each disorder (range = 0–8; a 4 is required to make a diagnosis). In this study, both child and parent were interviewed separately and a composite CSR was assigned by the independent evaluators. The CSR composite score was used as a covariate in study analyses to control for baseline child anxiety symptom severity. The presence of any anxiety diagnosis (yes/no) over the 1 year study period (i.e., across the three follow-up evaluations) was used as the outcome variable; inter rater agreement on a randomly selected 25% of ADIS-C administrations was 97%.

Family Environment Measures

The *Self-Report Measure of Family Functioning Scale* (MFFS; *Conflict and Locus of Control subscales;* Bloom 1985; Bloom and Naar 1994), completed by parents, was used to assess these two theoretically informed aspects of family functioning. Five items per subscale were rated on a four point Likert scale (1 = very untrue for my family to 4 = very true for my family); internal consistency at baseline was .62 and .66 on the Conflict and Locus of Control subscales respectively.

Children's Perception of Inter-parental Conflict Scale (CPIC: Grych et al. 1992) was completed by children to capture child's perception of family conflict. Two scales, each consisting of 6 items, assessed (1) conflict frequency (e.g., I often see my parents arguing) and (2) perceived threat of conflict (e.g., I am afraid something bad will happen to me when my parents argue) associated with inter-parental conflict and items were rated as "True," "Sort of True," or "False." Lower scores reflect higher perceived frequency of inter-parental conflict or higher perceived threat; internal consistencies in the current study for the frequency and threat subscales at baseline were .80 and .83, respectively.

Parenting Behavior Measures

Egna Minnen Betraffande Uppfostran (EMBU-C/P; Muris et al. 2003 Swedish for "My memories of upbringing") completed by children was used to assess two specific anxiety enhancing parenting behaviors: overprotection/control (e.g., *I want to decide how my child should be dressed or how he/she should look*) and anxious rearing (e.g., *I worry about what my child is doing after school*, *I worry about my child getting into trouble*). Each subscale has 10 items that are answered on a 4-point Likert scale (1 = No, 2 = Yes, but seldom, 3 = Yes, often, 4 = Yes, most of the time). Each subscale scores range from 10 to 40, with a higher score indicating a stronger endorsement of that parenting style. Internal consistencies were .63 for overprotection/control and .69 for anxious rearing.

Etch-A-Sketch observational paradigm, a video-recorded parent–child task that requires the dyad to work as a team in order to copy 3 designs that increase in complexity, was used to measure parental overcontrol. Overcontrol was coded by independent evaluators using a 5 point scale (0 = behavior not present, 1 = very rarely present/up to 25% of time, 2 = behavior present a little/26–50% of time and/or of mild severity, 3 = behavior present some/51–75% of the time and/or of moderate severity, 4 = behavior present most of time/76% or more of time and/or of marked severity). Inter-rater reliability has been demonstrated with these tasks and coding manual in previous studies (Drake and Ginsburg 2011; Ginsburg et al. 2004).

The Child Development Questionnaire—Reinforcement of Dependency scale (CDQ; Zabin and Melamed 1980) was modified by the first author and completed by parents to assess parental reinforcement of dependency (i.e., allowing child to avoid potentially anxiety provoking situations). This measure was selected because of its similarity to the construct of parental accommodation of child anxiety which has been documented to be associated with higher child anxiety (Lebowitz et al. 2013). Parents rated 14 items using a 5-point Likert-type scale (1 = never, 2 = rarely, 3 = sometimes, 4 = often, and 5 = always). An example item from this subscale was: "If I took my child to get a haircut and s/he absolutely refused to sit on the chair because s/he was frightened, I would most likely take child home immediately." Internal consistency at baseline for this subscale was .68; higher scores reflect higher parental reinforcement of dependency/accommodation of child avoidance.

The *Learning History Questionnaire-Revised* (LHQ-R), completed by parents is an abbreviated version of Ehlers' Learning History Questionnaire (Ehlers 1993; see Watt et al. 1998) and was used to assess parental modeling of anxious behavior (e.g., avoiding tasks due to anxiety). The LHQ-R consists of 17 items (e.g., "Do you stay home from work or cut back on household chores due to anxiety?"), each rated on a 4 point scale ranging from 0 (Never) to 3 (Often). Higher scores reflect higher levels of anxious avoidance; internal consistency for the Total score was .83 in the current sample at baseline.

Parental Psychopathology Measure

The Brief Symptom Inventory (Derogatis and Melisaratos 1983), completed by parents, is a widely used 53-item measure that was used to assess general distress associated with symptoms of psychopathology (e.g., depression, hostility). In the current study, the Global Severity Index, representing global distress (baseline internal consistency was .95) was used.

Data Analyses Table 1 presents descriptive statistics (i.e., frequencies, means and standard deviations) for all family predictors and covariates, and Table 2 presents correlations among all study variables and covariates. Hierarchical logistic regressions were conducted to test whether any of the theory-driven family and parent factors at baseline predicted child anxiety disorder onset during the 12-month follow-up period. Covariates in Step 1 of regressions included family income, child age and gender, current enrollment of parent in mental health treatment, parent gender, baseline child anxiety symptom severity, and CAPS or IM intervention condition. We also controlled for youth race/ethnicity. Because over 85% of the participating children were Caucasian/non-Hispanic, we included a binary "Caucasian/Non-Hispanic versus Non-Caucasian and/or Hispanic" variable for this purpose. Family and parent predictors were added in Step 2, one at a time. If multiple predictors emerged as significant, we planned to test their relative effects through an additional regression including all significant predictors.

Table 1 Descriptive statistics: familial predictor variables, covariates, and demographic sample characteristics

	M (SD)	% missing	Skewness statistic (SE)	Kurtosis statistic (SE)
Family predictors				
Etch-A-Sketch task: overcontrol	1.33 (1.14)	14.0	.60 (.22)	− .39 (.44)
EMBU—anxious rearing	24.53 (4.84)	6.6	.39 (.22)	.08 (.43)
EMBU—parental overprotection	25.12 (4.49)	6.6	.67 (.22)	.41 (.43)
BSI—Global Severity Index	.99 (.60)	1.5	1.04 (.21)	1.35 (.42)
CDQ—reinforce dependence	39.23 (6.62)	2.9	− .11 (.21)	.36 (.42)
MFFS—conflict	2.22 (.50)	1.5	.03 (.21)	− .25 (.42)
MFFS—external locus of control	1.75 (.48)	1.5	.46 (.21)	− .04 (.42)
LHQ—anxious modeling	1.37 (.52)	2.2	.29 (.21)	− .32 (.42)
CPIC—frequency of conflict	13.80 (2.96)	5.1	− .70 (.21)	− .25 (.42)
CPIC—perceived threat	14.65 (3.33)	5.1	− .87 (.21)	− .20 (.42)

	M (SD)	% missing
Demographics and covariates		
Family income (based on brackets numbered 1 through 8, each spanning $10 K)	8.41 (1.47)	0
Child age	8.69 (1.80)	0
Baseline child anxiety symptom severity (sum total Clinical Severity Rating score across anxiety disorder domains)	7.90 (4.42)	0

	% of total sample	% missing
CAPS Intervention Group (vs. control)	51.50	0
Child gender: female (vs. male)	55.88	0
Child race: Caucasian (vs. non-Caucasian)	84.55	0
Parent gender: female (vs. male)	78.67	0
Parent currently receiving anxiety treatment	64.71	0
Child met criteria for anxiety disorder during study period (vs. never met anxiety disorder criteria)	16.17	0

Analyses included all randomized children regardless of subsequent study withdrawal. As such, we addressed missing data in two ways. First, due to participant dropout prior to the 12-month assessment, data regarding whether children developed an anxiety disorder during the study period was incomplete for 15 of 136 initially enrolled children. For these children, the last observation carry-forward was used to estimate whether or not that child developed an anxiety disorder during the study, based on the latest assessment s/he had completed (either post-treatment or 6-month follow-up). Second, there was some subject-level missing data for all baseline parent and family variables (see Table 1 for details). To address missing data in candidate predictors, we employed multiple imputation in SPSS Version 24 to generate 20 imputed datasets. SPSS utilizes a Markov chain Monte Carlo algorithm known as fully conditional specification, or chained equations imputation, which

Table 2 Zero-order/point biserial correlations among all study variables from the original non-imputed dataset

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
1. CAPS intervention group (vs. control)	.12	.09	− .04	− .15	.04	− .04	− .05	− .01	.06	.02	.02	.02	.02	− .02	.03	− .01	.33*	− .02
2. Family income (based on brackets numbered 1–8, each spanning $10 K)		.01	.01	− .13	− .02	− .04	.11	− .18*	− .10	− .19*	− .05	.04	− .16	− .16	.28**	.04	− .07	.09
3. Child age		–	− .09	.03	.05	.04	.04	− .20*	− .19*	.14	− .05	.04	.18*	.17*	.03	− .09	.03	.03
4. Child race (Caucasian vs. non-Caucasian)			–	.08	− .13	− .13	.05	.02	− .08	.12	.01	− .04	− .13	.01	.07	.05	− .07	− .08
5. Female child gender				–	.15	− .15	.09	− .07	.01	.00	.07	.07	− .10	− .02	− .02	− .00	.07	− .13
6. Female parent gender					–	− .04	− .13	.13	.02	.14	.20*	.11	− .08	.04	.04	− .08	− .02	.00
7. Parent currently receiving anxiety treatment						–	.07	− .18*	− .21*	.16	.14	.10	.11	.33**	− .24**	− .05	− .01	.05
8. Etch-A-Sketch—overcontrol							–	− .07	.10	− .06	− .01	− .12	.06	− .08	− .01	-.01	.00	.06
9. EMBU—anxious rearing								–	.60**	− .02	− .07	− .08	− .09	− .11	− .04	− .20*	.04	.26**

Table 2 continued

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
10. EMBU—parental overprotection									–	.09	− .07	.05	.01	− .10	− .06	− .09	.09	.12
11. BSI—Global Severity Index										–	.16	.21*	.42**	.57**	− .21*	− .24**	− .01	.11
12. CDQ—reinforce dependence											–	.18*	− .14	.13	− .02	− .12	.01	− .03
13. MFFS—conflict												–	.41**	.17	− .26**	− .03	.09	.01
14. MFFS—external locus of control													–	.28**	− .28**	− .23*	.13	.10
15. LHQ—anxious modeling														–	− .22*	− .13	− .11	.04
16. CPIC—frequency of conflict															–	.28**	− .01	− .06
17. CPIC—perceived threat																–	− .01	− .27**
18. Did child meet anxiety disorder criteria? (across entire study period; 1 = yes, 0 = no)																	–	.12

Table 2 continued

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
19. Baseline child anxiety severity																		–

* $p < 0.05$; ** $p < 0.01$

imputes incomplete variables one at a time through linear regression, using the nonmissing variable from one step as a predictor in all subsequent steps. Candidate predictors and covariates were included as predictors for each imputation procedure. Regression results reported below reflect pooled estimates based on the 20 imputed datasets, although results based on original and imputed data did not significantly differ.

Results

Descriptive Statistics

Table 1 presents descriptive statistics for all study variables, including covariates; Table 2 presents correlations among these variables. Several parent and family variables were significantly correlated with one another in anticipated directions (see Table 2): for example, higher parental psychopathology, lower parental locus of control, greater parental modeling of anxious behavior, and higher child-reported inter-parental conflict and perceived threat were all significantly associated with one another. No parent/family variables correlated with one another above $r = .60$, mitigating potential multicollinearity concerns. Child gender was not associated with any parent or family variable. Older child age correlated with stronger parental external locus of control, as well as lower child-reported "anxious rearing" behaviors. No parent or family variables were significantly correlated with anxiety disorder onset over the course of the study period.

Predictors of Disorder Onset

Table 3 presents results from the logistic regression analysis testing the baseline family and parent variables as independent predictor of child anxiety disorder onset during the study period, one at a time above and beyond the covariates. Specifically, we examined: (1) family environments high in conflict (as measured by the CPIC and MFFS conflict scales) and external locus of control (as measured by the MFFS), (2) parental behaviors high in over-control (as measured by the Etch A Sketch behavioral observation and EMBU) and anxious rearing/overprotection, modeling of anxious behaviors (as measured by EMBU and LHQ respectively), and reinforcement of dependency (as measured by the CDQ), and (3) higher levels of parent psychopathology (as measured by the BSI). After controlling for the covariates, none of the parent-reported, child-reported, or observational parent/family variables significantly predicted child anxiety disorder onset over the course of the study period.

Discussion

Previous research has demonstrated that offspring of anxious parents are at increased risk for developing anxiety disorders (Micco et al. 2009). Few studies have prospectively examined potential parental mechanisms of transmission associated with child anxiety disorder onset. In the current study, several theory-based family and parent factors were examined, using multiple informants and an observational task, as predictors of child anxiety disorder onset over a 1-year period in a high-risk sample of offspring of anxious parents. In contrast to our hypotheses and extant literature, none of the examined parent or

Table 3 Results of logistic regressions testing individual family/parent variables as predictors of child anxiety disorder onset during 12 month follow-up period, based on pooled estimates from 20 imputed datasets

Step and variable	*b*	*SE*	Wald statistic	Odds ratio (95% confidence interval)	χ^2	Cox and Snell ΔR^2
Step 1					χ^2 (8) = 22.59**	.20**
Intervention group	− 2.49	.73	11.51	.08 (.02, .35)**		
Annual family income	− .35	.19	3.30	.77 (.51, 1.15)		
Child age	.03	.15	.04	1.03(.76, 1.40)		
Child female gender	− .91	.56	2.65	.40 (.13, 1.20)		
Child race (Caucasian vs. non-Caucasian)	.20	.81	.06	.82 (.25, 2.69)		
Baseline child anxiety severity (total CSR rating)	− .34	.64	.28	.71 (.20, 2.52)		
Parent female gender	.12	.72	.03	.89 (.22, 3.63)		
Parent currently receiving anxiety treatment	− .20	.58	.12	.82 (.26, 2.58)		
Step 2 (each variable entered in individual regressions)						
Family environment						
MFFS Scale—parent external locus of control	1.11	.64	2.95	3.04 (.85, 10.79)	χ^2 (1) = 3.08	.02
MFFS Scale—parental conflict	.74	.66	1.26	2.10 (.58, 7.62)	χ^2 (1) = 2.37	.02
CPIC—frequency of conflict (child-report)	.02	.11	.04	1.02 (.82, 1.27)	χ^2 (1) = .04	.00
CPIC—perceived threat (child-report)	.09	.12	.72	1.10 (.88, 1.38)	χ^2 (1) = .76	.01
Parenting behaviors						
Etch-a-Sketch task—overcontrol	− .04	.24	.02	.96 (.60, 1.54)	χ^2 (1) = .02	.00
EMBU—anxious rearing	− .01	.07	.27	.99 (.86, 1.14)	χ^2 (1) = .03	.00
EMBU—parental overprotection	.04	.07	.40	1.04 (.92, 1.19)	χ^2 (1) = .39	.00
LHQ—parental anxious modeling	− .72	.57	1.60	.49 (.16, 1.49)	χ^2 (1) = 1.66	.01
CDQ—reinforcement of dependency	− .002	.04	.001	.99 (.92, 1.09)	χ^2 (1) = .00	.00
Parent psychological distress						
BSI—Global Severity Index	− .21	.45	.22	.81 (.34, 1.95)	χ^2 (1) = .23	.00

* $p < 0.05$; ** $p < 0.01$

family variables predicted child anxiety disorder onset. Specifically, family environment (i.e., high conflict and high parental perceptions of external locus on control), anxiety-enhancing parenting behaviors (i.e., reinforcement of dependency, anxious rearing, modeling of anxiety, and overcontrol) and parental psychopathology (global distress) did not predict child anxiety disorder onset. Similarly, our control variables (including parent treatment for their own anxiety) did not predict child anxiety disorder onset, with the exception receiving the active CAPS intervention, which lowered the odds of a child developing an anxiety disorder relative to the control condition.

Several interpretations of these findings are plausible. It may be that the specific parent and family factors selected for this study do not influence the onset of anxiety disorders, although they have been found to be associated with pediatric anxiety using cross sectional methods (see McLeod et al. 2007). Unmeasured and unexamined parent/family factors, such as inconsistent parenting or insecure attachment, parental depression or substance use, or even type of parental anxiety disorder (e.g., panic or social anxiety) may be significant risk factors for child anxiety disorder onset. It is also possible that family/parenting factors do not exert an *independent effect* on child anxiety disorder development but rather interact with other factors such as peer victimization or child temperament. Findings from a recent study support this interpretation. Using this same dataset, Schleider, Ginsburg and Drake (2017) found that children's perceived peer victimization at baseline predicted increased anxiety severity at a 1 year follow-up among children with highly or moderately anxious parents, but not among children with low-anxiety parents (Schleider et al. 2017). It is also plausible that family and parental factors may play a role in *maintaining* anxiety, rather than *causing* anxiety disorder onset, as most studies linking family factors and anxiety have been cross sectional and focused on clinically anxious youth. Family/parent factors may also predict specific domains of anxiety disorders, such as separation or social anxiety disorder—rather than "any" disorder as examined in this study. The low frequencies of specific disorders precluded testing this hypothesis in this sample (e.g., three children met criteria for social anxiety disorder at any assessment point). The timing or frequency of measuring family/parent factors may have also influenced the findings. Specifically, perhaps assessing parent/family variables at more than one time point is needed, ensuring a stable impact of family/parenting characteristics, to identify a clinically meaningful predictor of anxiety disorder onset.

Methodological factors may also explain the current null findings—the sample size of youth who developed an anxiety disorder was relatively small (n = 22) and, as in many studies examining predictors of child anxiety disorders, statistical power may have been inadequate to detect small effects. Related, the range of scores on some of measures assessing family and parent factors were skewed, the internal consistency of some measures were low, and the confidence intervals were wide, which may also have reduced statistical power and/or led to non-statistically significant findings. To assess the possibility that the study was underpowered, we conducted a power analysis for multivariate logistic regression using guidelines established by Lipsey (1990) and G*Power 3.1.7 (Faul et al. 2013) to determine the sample sizes necessary to detect small (OR = 1.3), medium (OR = 1.72), and large (OR = 2.48) effect sizes. Setting alpha at 0.05 and power at 0.80, and using a two-tailed significance test, sample sizes of N = 655, N = 166, and N = 70 were deemed necessary to detect small, medium, and large effects, respectively. Thus, the present study was only powered to detect medium-to-large effects, and the impact of parenting and other family factors has been found to be small (McLeod et al. 2007). Other study limitations should be noted: the sample of anxious parents in this study were

homogenous with respect to racial composition, level of education, marital status, and income and may limit the generalizability of findings.

Summary and Conclusions

Studies examining the relation between family factors and child anxiety in high risk families have been inconsistent and largely cross sectional (Wood et al. 2003), and more systematic longitudinal research is needed to explicate the mechanisms of anxiety transmission. Of particular importance is the need to accumulate large data sets to test hypotheses that will identify family and parent factors that are linked to the transmission of anxiety from parent to child. Taken together, these findings appear to be good news for anxious parents and can reassure them that their own anxiety/distress or parenting may not independently lead to child anxiety disorder onset—a least over a one year period.

Acknowledgements This study was supported by a grant from the National Institute of Mental Health (R01 MH077312) awarded to Golda S. Ginsburg.

Compliance with Ethical Standards

Conflict of interest None of the authors have potential conflicts of interest to report.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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