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Parent and Child Anxiety Sensitivity: Relationship to Children's Experimental Pain Responsivity

Jennie C. I. Tsao,* Qian Lu,* Cynthia D. Myers,† Su C. Kim,* Norman Turk,* and Lonnie K. Zeltzer*

*Pediatric Pain Program, Departments of Pediatrics, Anesthesiology, Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, University of California, Los Angeles.
†Department of Interdisciplinary Oncology, Moffitt Cancer Center and Research Institute, Tampa, Florida.

Abstract: **Anxiety sensitivity (AS) or fear of anxiety sensations has been linked to childhood learning history for somatic symptoms, suggesting that parental AS may impact children's responses to pain. Using structural equation modeling, we tested a conceptual model in which parent AS predicted child AS, which in turn predicted a hypothesized latent construct consisting of children's pain intensity ratings for 3 laboratory pain tasks (cold pressor, thermal heat, and pressure). This conceptual model was tested in 211 nonclinical parent-child pairs (104 girls, 107 boys; mean age 12.4 years; 178 mothers, 33 fathers). Our model was supported in girls only, indicating that the sex of the child moderated the hypothesized relationships. Thus, parent AS was related to child laboratory pain intensity via its contribution to child AS in girls but not in boys. In girls, 42% of the effect of parent AS on laboratory pain intensity was explained via child AS. In boys, there was no clear link between parent AS and child AS, although child AS was predictive of experimental pain intensity across sex. Our results are consistent with the notion that parent AS may operate via healthy girls' own fear of anxiety symptoms to influence their responses to laboratory pain stimuli.**

Perspective: ***The present study highlights sex differences in the links among parent and child anxiety sensitivity (fear of anxiety sensations) and children's experimental pain responses. Among girls, childhood learning history related to somatic symptoms may be a particularly salient factor in the development of anxiety sensitivity and pain responsivity.***

Key words: *Anxiety sensitivity, laboratory pain, children, adolescents, parent, sex differences.*

Anxiety sensitivity (AS) refers to the tendency to interpret anxiety sensations (eg, rapid heartbeat) as leading to harmful consequences.[21,26] In adult studies, AS has emerged as an important correlate of chronic pain[1,20] as well as a salient predictor of experimental pain responses.[12-14] The general finding for adult laboratory studies is that for women high AS is associated with increased experimental pain intensity, but AS shows no relationship with pain threshold or tolerance for either men or women. In children, links between AS and pain symptoms have recently been reported. In one study, pediatric patients with unexplained chest pain evidenced higher AS levels than controls.[15] Although an initial investigation conducted in our laboratory using standard linear regression techniques revealed only weak associations between AS and experimental pain response in children,[29] more recent work in a larger sample using complex modeling has shown a robust link between AS and pain-related anticipatory anxiety which in turn strongly predicts laboratory pain intensity (Tsao et al[31]). These findings and those of others[22] support the notion that AS may be conceptualized as the propensity to perceive any source of arousal as threatening.[22,34,35] Thus, AS may amplify the experience of bodily symptoms related to a wide range of somatic events, including the experience of pain.

Despite a paucity of data on the developmental origins of AS, retrospective studies have found that childhood

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Address reprint requests to Jennie C. I. Tsao, Pediatric Pain Program, Department of Pediatrics, David Geffen School of Medicine at UCLA, 10940 Wilshire Blvd, Ste 1450, Los Angeles, California 90024. E-mail: jtsao@mednet.ucla.edu

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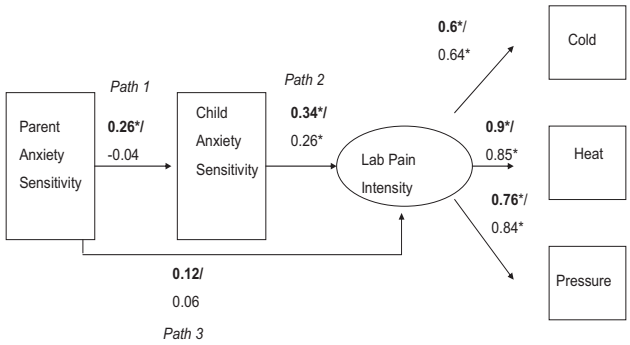

Note: Path coefficients (standardized coefficients) and factor loadings are depicted for girls/boys. Path coefficients are bolded for girls.

* p<0.05

Figure 1. Path model with standardized path coefficients and factor loadings for girls and boys. Path coefficients for girls are in bold. *$P < .05$.

learning history related to somatic symptoms, including pain, demonstrates significant associations with high AS in adults.[25,34,35] Muris et al[19] reported that parental transmission of the idea that somatic symptoms, including pain symptoms, may be dangerous was significantly linked with elevated AS in healthy adolescents. These data suggest that parents' perceptions of the danger of somatic symptoms may influence their children's perceptions of such symptoms. Children's own AS may then in turn amplify the intensity of experienced somatic sensations. Despite the likely role of parental AS beliefs in indirectly influencing children's response to somatic events, no studies to date have examined the relationships among parent AS (PAS), child AS (CAS), and children's response to controlled pain stimuli administered in the laboratory.

Therefore, the purpose of the present study was to investigate these associations in a healthy sample using structural equation modeling (SEM). Structural equation modeling allows the examination of both direct and indirect effects as well as the relationships between observed variables and latent variables to reduce measurement errors. The hypothesized model (Fig 1) posited that PAS is related to CAS (path 1) and that CAS in turn is predictive of the latent factor laboratory pain intensity (path 2) composed of pain intensity ratings for 3 standardized pain tasks (cold pressor, pressure, and heat). Thus, PAS was expected to show an indirect relationship with child laboratory pain intensity via its contribution to CAS. We further hypothesized that the sex of the child would moderate the indirect relationship between PAS and pain intensity through CAS. In a prior study using standard linear regression techniques in this sample, we found a significant association between parent and child AS in girls but not in boys[30]; however we did not explicitly examine the relationship between PAS and a latent factor representing child laboratory pain response, nor the direct and indirect links among the variables of interest in our earlier work. In the present study, pain intensity was chosen as the index of pain reactivity, because in previous research AS has not demonstrated an association with experimental pain threshold or tolerance in adults[13] or children.[29]

Materials and Methods

Participants

There were 244 healthy child and adolescent participants (124 female, 50.8%) with a mean age of 12.7 years (SD 3.11, range 8-18). The ethnic composition of the child/adolescent sample was: 40.2% caucasian, 13.9% African American, 9.8% Asian American, 23.8% Hispanic, 12.3% other. Parent socioeconomic status[7] was: unskilled workers 3.7%, semiskilled workers 4.1%, clerical/sales 11.9%, technical 41.8%, professional 34.8%. Participants were recruited from a major urban area through mass mailing, posted advertisements, and classroom presentations. The source of the mailing list was a database of consumers with children between 8 and 17 years old residing in Los Angeles County as well as a mailing list of UCLA staff and faculty members. Study advertisements were posted in locations where parents and children would be expected to encounter them, such as community centers, libraries, public businesses, youth clubs and organizations, hospitals/clinics, and private medical offices in Los Angeles County. Study flyers were also posted in and around the UCLA campus and hospital. The study was presented to middle and high school students during a science classroom assembly. The mailings and advertisements were widely targeted across sites with varying ethnicities and income levels, because one of the goals of subject recruitment was to enhance the enrollment of children from low-income and minority neighborhoods.

Eligibility was confirmed by telephone. Individuals reporting on-going acute or chronic illness or use of prescription medications that would affect study measures were excluded. Parents and participants were told that the purpose of the study was to examine the roles of gender and puberty in the ways healthy children respond to laboratory pain tasks. Written informed consent forms were completed by parents, and children provided written assent. This study was approved by the UCLA Institutional Review Board. Participants received a $30 video store gift certificate and a T-shirt for their participation.

The majority of parent participants were mothers (n = 188; 77.05 %), followed by fathers (n = 35; 14.34%), legal guardians (n = 2; 0.82%), and others (n = 10; 4.1%); 9 participants (3.69%) did not report their familial status. The "other" category consisted of an aunt, a grandmother, a stepmother, a brother, and sisters. Data regarding ethnicity of parent participants was not available. Because the focus of the present study was on parent-child relationships, data for legal guardians and "other" participants were excluded from the analyses. Twelve cases with incomplete data on either the questionnaires or the laboratory pain measures along with

the participants who did not report familial status were excluded. The final sample consisted of 211 parent-child pairs. Most of these parent-child pairs (n = 207) have been studied in another report on parent-child AS relationships.[30]

Procedure

Details of the study from which the current data are drawn are described elsewhere.[29] Briefly, on the day of the laboratory session, parents and child participants were escorted to separate rooms. Children first completed questionnaires, administered by an experimenter in a quiet room adjacent to the laboratory. Child participants were then led into the laboratory where they were instructed on the use of the visual analog scale (VAS) for rating pain (described below) and then exposed to the 3 pain tasks, counterbalanced across participants (see below). Parent participants completed the questionnaires either at home or during their child's laboratory session. Parents were not with their child while he or she completed questionnaires, nor were parents present in the laboratory during administration of the pain tasks.

Cold Pressor Task

Participants underwent 2 trials of 10°C water using a commercial ice chest measuring 38 cm wide, 71 cm long, and 35 cm deep. A plastic mesh screen separated crushed ice from a plastic large-hole mesh armrest in the cold water. Water was circulated through the ice by a pump to prevent local warming about the hand. In the first trial, participants placed the nondominant hand in cold water to a depth of 2 inches above the wrist for as long as they could, with an uninformed ceiling of 3 minutes. In trial 2, participants were instructed to keep the dominant hand in the water for 1 minute. Data from this trial are presented elsewhere.[28]

Pressure Task

The Ugo Basile Analgesy-Meter 37215 (Ugo Basile Biological Research Apparatus, Comerio, Italy) was used to administer focal pressure through a lucite point approximately 1.5 mm in diameter to the second dorsal phalanx of the middle finger or index finger of each hand. Four trials at 2 levels of pressure (322.5 g and 465 g) were run with an uninformed ceiling of 3 minutes.

Thermal Task

The Ugo Basile 7360 Unit was used to administer a total of 4 trials of 2 infrared intensities (15 and 20) of radiant heat 2 inches proximal to the wrist and 3 inches distal to the elbow on both volar forearms, with an uninformed ceiling of 20 seconds. Thermal pain tolerance was electronically measured with an accuracy of .1 second.

Between each trial, there was a 1-minute intertrial interval. For the thermal and pressure tasks, the presentation order (setting, site) was counterbalanced across participants. Before the start of each trial, subjects were informed that they would experience moderate sensation, which may eventually be perceived as pain. They were instructed to continue with the task for as long as they could, although they were also told, "you are free to withdraw from the (cold water, heat, or pressure) at any time."

Measures

Child Questionnaire

The Childhood Anxiety Sensitivity Index (CASI)[23] is an 18-item scale that measures the tendency to view anxiety-related bodily sensations as dangerous. Sample items include "When my stomach hurts, I worry that I might really be sick" and "It scares me when my heart beats fast." Items are scored on a 3-point scale (none, some, a lot); total scores are calculated by summing all items. The CASI has demonstrated high internal consistency (α = .87) and adequate test-retest reliability (range .62-.78 over 2 weeks).[23] The CASI correlates well with measures of trait anxiety (r = .55-.69) but also accounts for variance in fear not attributable to trait anxiety measures.[36]

Parent Questionnaire

The Anxiety Sensitivity Index (ASI)[21] is a 16-item measure of the tendency to interpret physical sensations as harmful, asked participants to indicate on a 5-point scale (very little, a little, some, much, very much) the degree to which each statement was true for them. The ASI has been shown to have adequate internal consistency (α = .82)[27] and high test-retest reliability (.71 over 3 years).[17]

Child Pain Task Measure

Pain intensity ratings. Immediately after each trial, participants were asked to rate the level of pain experienced during the task. Participants used a vertical sliding VAS, anchored with 0 at the bottom indicating the least amount and 10 at the top indicating the greatest amount, in response to the instruction to rate "at its worst, how much pain did you feel" during the task. The scale also had color cues, graded from white at the bottom to dark red at the top, as well as a neutral face at the bottom and a negative facial expression at the top. The VAS has been established and widely used as a valid and reliable measure of pain intensity with children in clinical and experimental studies.[18,32]

Data Analysis

Structural Equation Modeling Overview

EQS program version 6.1[3] was used to test the hypothesized model using standard maximum likelihood (ML) estimation. The goal of SEM is to compare a covariance matrix generated from a particular sample with a covariance matrix generated by a hypothesized model. For samples of n $\leq$ 250, Hu and Bentler[8] recommended combinational rules to evaluate model fit, with a value of .95 or above for the comparative fit index (CFI) and a value of or below .09 for the stan-

Table 1. Bivariate Correlations Among Parent Anxiety Sensitivity (PAS), Child Anxiety Sensitivity (CAS), and Pain Intensity in the Total Sample and in Boys and Girls

	PAS	CAS	Cold Pain Intensity	Heat Pain Intensity	Pressure Pain Intensity
Total sample					
PAS					
CAS	0.12				
Cold pain intensity	0.16*	0.18**			
Heat pain intensity	0.10	0.29**	0.53***		
Pressure pain intensity	0.08	0.23**	0.50***	0.69***	
Boys and girls separately					
PAS		−0.04	0.13	0.02	0.03
CAS	**0.26****		0.01	0.18	0.12
Cold pain intensity	**0.20***	**0.29*****		0.44***	0.52***
Heat pain intensity	**0.18**	**0.37*****	**0.61*****		0.71***
Pressure pain intensity	**0.12**	**0.31*****	**0.47*****	0.69***	

NOTE. Correlations for girls are in bold.

*$P < .05$.

**$P < .01$.

***$P < .001$.

dardized root mean-square residual (SRMR) indicating good fit. Hu and Bentler also recommended values of at least .95 for the nonnormed fit index (NNFI), and .06 or below for the root mean-square error of approximation (RMSEA). Maximum likelihood estimation assumes multivariate normality. For all the study variables, univariate skewness and kurtosis were fairly normal. Multivariate kurtosis Mardia's coefficient was 1.89 and normalized estimate 1.63, suggesting that the multivariate distributions were normal. Therefore, the ML method was used to estimate all models. Maximum likelihood robust estimator was also used to confirm the results obtained in nonrobust methods.

To evaluate model fit across multiple groups, SEM analyzes parameters simultaneously to determine which of several models best reproduces the sample data in each group. Starting with a baseline unrestricted model, increasingly restrictive hypotheses may be evaluated by constraining certain key parameters to equality across groups. The difference between 2 chi-squared values for nested models is distributed as chi-squared values and their degrees of freedom. Parsimonious explanations are preferred. The goal is to not degrade the models by constraining parameters across the groups; therefore, a nonsignificant chi-squared is preferred. If there is no significant difference in chi-squared values between the models, the more constrained model is considered superior. If there is significant difference in chi-squared values between models, the less constrained model is considered to fit significantly better than the more constrained model.

Results

Descriptive Statistics

Pain intensity ratings for the thermal and pressure tasks were highly correlated across the 4 trials within each task (r = .53-.89; $P < .001$). Therefore, these data were averaged across trials yielding a mean thermal intensity rating and a mean pressure intensity rating. Bivariate correlations among the measured variables in the total sample and in boys and girls separately are provided in Table 1. Girls and boys did not differ on CAS, PAS, or pain intensity for any task. Although the impact of child age on the hypothesized relationships was initially considered for inclusion in our models, bivariate correlations among the measured variables did not change substantially even after controlling for child age. Thus for the sake of parsimony, child age was not included in the proposed models.

Structural Equation Modeling and Multiple Group Comparison

Structural equation modeling was initially performed in the total sample to determine the adequacy of the hypothesized factor structure and hypothesized relationships among variables. The model posited that the independent variable, parent anxiety sensitivity (PAS), would predict child anxiety sensitivity (CAS), which in turn would predict pain response (latent construct of pain intensity). This latent construct of pain intensity was hypothesized to comprise pain intensity ratings for the 3 laboratory tasks. The SEM fit the data well: χ^2 (4, N = 211) = 3.96; $P = 0.41$; CFI = 1.00; NNFI = 1.00; SRMR = 0.025; RMSEA = 0.00. As expected, pain ratings for the 3 tasks reflected an underlying latent construct of pain intensity. The path coefficients were: cold pain intensity (standardized coefficient = .62), heat pain intensity (standardized coefficient = .87), and pressure pain intensity (standardized coefficient = .79). However, PAS did not significantly predict pain intensity or CAS, although CAS did predict pain intensity (r = 0.31; $P < .05$).

To test the hypothesis that sex moderated the relation-

Table 2. Summary of CFA and Path Analysis Fit Indices for Multiple-Group Model Comparisons

Model	χ^2	*df*	*P value*	*CFI*	*NNFI*	*SRMR*	*RMSEA*
A. All free	5.64	8	0.6	1.00	1.03	0.03	0.00
B. FL	9.08	10	0.5	1.00	1.07	0.05	0.00
C1. FL and Cov (PAS, CAS)	14.84	11	0.18	0.99	0.97	0.07	0.00
C2: FL and Cov (CAS, pain)	11.14	11	0.43	1.00	1.00	0.07	0.00
C3. FL, Cov (PAS, CAS), and Cov (CAS, pain)	16.92	12	0.15	0.98	0.97	0.09	0.04
B-A difference	3.44	2	>0.1				
C1-B difference	5.14	1	<0.025				
C2-B difference	2.06	1	>0.1				
C2-A difference	5.50	3	>0.1				
C3-C2 difference	5.78	1	<0.025				

Abbreviations: CFI, comparative fit index; NNFI, nonnormed fit index; SRMR, standardized root mean-square residual; RMSEA, root mean-square error of approximation; *df:* degree of freedom; FL, factor loadings constrained to equality across boys and girls; FL and Cov, factor loading and covariance constrained to equality across boys and girls; Cov (PAS, CAS), covariance between parent anxiety sensitivity and child anxiety sensitivity; Cov (CAS, pain), covariance between child anxiety sensitivity and pain; B-A model difference, $\chi^2_{\text{model B}} - \chi^2_{\text{model A}}$.

ships among PAS, CAS, and pain intensity, a series of analyses were conducted to determine whether girls and boys differed significantly on: 1) the factor loadings for the latent pain intensity construct, 2) the paths between PAS and CAS, and 3) the path between CAS and the latent pain intensity construct. We first tested invariance of factor loadings to examine whether the measurement model was equally valid among boys and girls. A summary of these model fit indices and nested difference χ^2 tests is given in Table 2.

Invariance Across Latent Factor Loadings

In SEM, the measurement invariance test uses a chi-squared difference test to assess whether a set of indicators reflects a latent variable equally well across groups in a given sample. The constrained model is one in which factor loadings are specified to be equal across groups. If there is no significant difference between the constrained and the nonconstrained models, the constrained model is considered superior and it can be concluded that the indicators are valid across groups. Two sets of structural models examined invariance across factor loadings. The first model tested (model A) allowed all factor loadings and path coefficients to be freely estimated for boys and girls separately. The second model (model B) constrained the 3 factor loadings on the pain intensity latent factor to equality for both girls and boys. Both model A and model B produced good fit of data (Table 2). The chi-squared difference test comparing model A and model B was not significant ($\chi^2_{\text{B-A model difference}} = 3.44$; $df = 2$; $P > .1$), thus model B, the more restricted model, was confirmed, indicating that the factor loadings for boys and girls were the same.

Invariance Across Path Coefficients

In SEM, the invariance across path coefficients test uses chi-squared difference tests to assess whether path coefficients reflect relationships between variables equally well across groups in a given sample. In the constrained model, path coefficients are specified to be equal across groups. If there is no significant difference between the constrained and the nonconstrained models, the constrained model is considered superior and it can be concluded that the path coefficients are valid across groups. If there is a significant difference between the 2 models, the less constrained model is considered superior and it can be concluded that the path coefficients are different across groups.

We thus examined whether the path coefficients between PAS and CAS differed for girls and boys. Model C1 tested this notion by constraining all factor loadings and the path coefficient between PAS and CAS to equality across groups. This model fit the data reasonably well, but chi-squared difference test indicated that model C1 provided a significantly worse fit to the data compared to model B which constrained only the factor loadings to equality ($\chi^2 = 5.14$; $df = 1$; $P < .025$). Thus, model C1, the more restricted model, was disconfirmed, suggesting that path coefficient between PAS and CAS was different for girls and boys. Model A was a less constrained model than model B, so further comparison between C1 and A was not necessary.

We then examined whether the path coefficients between CAS and pain intensity differed for girls and boys. Model C2 tested this notion by constraining all factor loadings and the path coefficient between CAS and pain intensity to equality. This model fit the data well, and there was no significant difference in model fit between model C2 and model B ($\chi^2 = 2.065$; $df = 1$; $P > .1$). Therefore the more restricted model C2 was confirmed, suggesting that the path coefficient between CAS and pain intensity did not differ between girls and boys. In addition, there was no significant difference in model fit between model C2 and model A ($\chi^2 = 5.5$; $df = 3$; $P > .1$), indicating that both the factor loadings and path coefficient between CAS and pain intensity were the same in girls and boys.

Finally, we examined whether there was a more parsimonious model that would fit the data better than model C2. In a hypothesized model C3, all the path coefficients and all the factor loadings were constrained to

equality for girls and boys. Model C3 fit indices were good, but model C3 provided significantly worse fit than model C2 ($\chi^2 = 5.78$; $df = 1$; $P < .025$). Therefore, model C2 represented the most parsimonious model as well as the best model fit among the models tested.

Analysis of model C2 also revealed that CAS mediated the relationship between PAS and pain intensity, and this mediational relationship was modified by child sex. Standardized parameter estimates for this final model (C2) are shown in Fig 1. In girls, PAS significantly predicted CAS which in turn predicted pain intensity. In boys, PAS did not predict CAS, although CAS did significantly predict pain intensity. Lagrangian multiplier test indicated that the correlation between PAS and CAS differed significantly by sex ($\chi^2 = 6.461$; $P = .011$) and that the correlation between CAS and pain intensity did not differ by sex ($\chi^2 = 2.309$; $P = 1.29$). These results suggested that CAS mediated the relationship between PAS and pain intensity in girls only.

Further Mediation Analysis

Recent methodologic work has provided statistical techniques that build on the Baron and Kenny[2] model but provide a more precise picture of mediation, including the provision of a statistical test for the mediation path (ie, the Sobel test).[16] Using the Sobel test, we found that the mediation path was significantly different from zero in girls. The formula for the Sobel test is:

$$t_{ab} = \frac{a \cdot b}{Se_{ab}}$$

where

$$Se_{ab} = \sqrt{(a^2 \cdot Se_{b^2}) + (b^2 \cdot Se_{a^2})}$$

In this formula, a refers to the unstandardized regression coefficients between the independent variable and the mediator, and b refers to the unstandardized regression coefficients between the mediator and the outcome variable, with Se_a and Se_b referring to the standard errors of these coefficients. Using this equation, t_{ab} was calculated ($t_{ab} = 2.13$; $df = 232$; $P < .05$), indicating that the mediation path was significantly different from zero.

In addition, we calculated the amount of explained variance accounted for by the mediation which was computed simply as:

$$R^2 = \frac{a \cdot b}{(a \cdot b) + c'}$$

where a and b are the unstandardized path coefficients and c is the unstandardized path between the independent variable and the outcome variable in the full model. Thus, we calculated $R^2 = 42\%$, indicating that 42% of the effect of PAS on pain intensity was accounted for by mediation through CAS.

Discussion

We tested a conceptual model positing that PAS would predict CAS, which would in turn predict a latent factor representing children's pain intensity in response to laboratory pain tasks involving cold, pressure, and heat stimuli (Fig 1). The proposed model was not confirmed in the total sample; the path coefficient between PAS and CAS was not significant (r = .12) (Table 1). However, further analyses examining the moderating effect of sex on the hypothesized relationships revealed that in girls PAS showed a significant association with CAS, which in turn predicted the latent factor pain intensity. Mediation analysis revealed that 42% of the effect of PAS on pain intensity was explained via its effects on CAS in girls. These findings are consistent with our prior work using standard multiple regression analysis that revealed a significant link between parent and child AS in girls but not in boys.[30] However, the present study extended these earlier findings by testing the indirect relationship between PAS and child laboratory pain response through CAS; moreover, the current analyses were able to confirm the existence of a child pain intensity latent factor composed of pain responses to the 3 laboratory tasks. One possible reason for the lack of an association between parent and child AS in our sample of boys is that such relationships may be found primarily in clinical samples of boys. In girls, on the other hand, our results suggest that even in healthy samples, the association between parent and child AS holds. Despite the lack of a clear relationship between parent and child AS in boys, CAS was found to be significantly related to pain intensity in the total sample.

Our findings agree with prior research reporting significant associations between parental anxiety and child distress during painful medical procedures,[9,11] although those studies did not examine sex-dependent effects. Whereas both of the earlier studies found that parent anxiety related to upcoming procedures (ie, anticipatory or state anxiety) predicted child distress, only 1[11] reported that parent trait anxiety was related to child distress. Previously, we found that symptoms of anxiety and depression in parents did not evidence significant relationships with CAS after PAS was taken into account.[30] Therefore, we did not include these more general measures of negative affect in the current study. Taken together, our results suggest that parent dispositional factors specifically related to AS, rather than general negative affect, are associated with AS among healthy girls; AS in turn influences how these girls respond to painful stimulation.

The current results support the possibility of sex differences in the transmission of AS from parent to child. It has previously been reported that AS is heritable in women only, with genetic factors accounting for 37% to 48% of the variance in AS among women but environmental factors accounting for all of the variance in men.[10] Several pathways have been posited for the development of AS, including temperamental factors (eg, behavioral inhibition), insecure attachment,[37] and social learning (eg, instruction by parents).[24] Few empirical studies, however, have specifically tested these potential pathways and additional longitudinal research is warranted. Our findings suggest that the en-

vironmental factors influencing the development of boys' AS may not stem directly from parent's own fear of somatic symptoms but derive from other, unknown sources. In girls, however, the direct association between parent and child AS supports the possibility that, in tandem with genetic effects, environmental influences related to PAS may help shape the development of their daughters' AS.

Recent work has revealed that maternal verbal and nonverbal behavior[4,6] directly affect experimental pain responsivity in healthy children. In a study by Chambers et al,[4] mothers' behavior during the cold pressor task influenced daughters' but not sons' pain intensity during a subsequent cold pressor trial. The authors noted that girls may have been more aware of and reactive to their mother's behavior during the task than boys—an explanation consistent with research suggesting that girls are more sensitive to parents' behavior regarding pain symptoms than boys.[33] The authors further speculated that parent behaviors may function primarily as signs of parental anxiety or concern which precipitate children's behavioral distress. Thus, it may be that girls are more sensitive to behaviors reflecting parental anxiety concerning somatic events, compared to boys. This heightened sensitivity in girls might then lead to their own increased tendency to interpret somatic sensations as dangerous, resulting in elevated reactivity to pain.

It should be noted that our sample consisted of mostly mothers and thus, it is possible that the results may partially reflect a stronger bond among same-sex mother-daughter pairs versus cross-sex mother-son pairs. The work by Chambers et al[4] similarly suggests a more robust mother-daughter relationship in pain-related behavior compared to mothers and sons; fathers were not included in their study. Unfortunately, owing to the small number of fathers in the current sample, we were unable to test whether our findings could be attributed to same-sex effects. Moreover, prior research on childhood learning history has not distinguished between the potential influence of mothers and fathers on their sons' and daughters' AS. Assuming a 2-parent family, it is conceivable that such influences may differ and additional studies should include both fathers and mothers to examine possible interaction effects between the sex of the parent and the sex of the child.

The notion that parental AS may influence children's pain responses is consistent with the conceptualization of AS reflecting beliefs about the dangerousness of somatic symptoms in general, rather than anxiety symptoms per se.[5] As mentioned previously, recent investigations have shown that parental informational transmission related to somatic symptoms, including pain, are associated with high AS,[25,34,35] indicating that elevated AS may arise from learning to catastrophize about somatic symptoms in general.[34] One area for future study is the extent to which AS leads to increased pain sensitivity to acute procedural pain. Additional studies might examine how parental transmission of AS may contribute to the development of chronic pain in certain vulnerable children. In light of the known adult female predominance in anxiety and chronic pain disorders, further research may also focus on the possibility that high AS in girls may be a pathway by which such conditions develop and whether there may be certain developmentally sensitive periods (eg, preadolescence) during which parental AS exerts maximal influence on girls' AS which may then lead to increased risk for psychopathology in later adolescence or early adulthood.

Several limitations of the current findings should be mentioned. The present data are cross-sectional and therefore conclusions regarding causality cannot be inferred. Although the current results suggest that our hypothesized model fit the data closely in girls, this does not rule out other possible causal models. Our models focused specifically on child-reported pain intensity and did not include a behavioral measure of pain tolerance. Previous work has indicated that AS does not show a strong relationship with tolerance for experimental pain tasks in adults[13] or children.[29] Future studies should examine models incorporating parent-child psychologic factors that predict behavioral aspects of pain response (eg, threshold and/or tolerance). Our analyses revealed a statistically significant indirect relationship between PAS and girls' laboratory pain response via girls' own AS; however, the clinical significance of these findings is less clear. Finally, our study does not allow statements regarding mechanisms by which PAS impacts girls' AS or by which CAS might influence pain response. Future studies may examine how these pathways operate.

In sum, our findings support the notion that parents' tendency to interpret anxiety symptoms as dangerous may play a salient role in how healthy girls respond to painful stimuli by influencing girls' own fear of anxiety symptoms. However, parents' concerns about anxiety symptoms did not show a similar link to their sons' own fears. Nevertheless, the potential role of AS in shaping children's response to somatic symptoms was supported by our findings that AS in both boys and girls were predictive of their perceived pain intensity experienced across an array of painful stimuli. The present study was conducted in a healthy sample and the generalizability of our results to clinical samples remains unclear. Nevertheless, 1 clinical implication of the current findings is that both parents' and children's fear of anxiety symptoms may be important targets for evaluation as part of a comprehensive assessment approach in families presenting with pediatric pain problems. Moreover, the possibility that amelioration of AS in parents and their children may lead to reductions in children's pain response to acute pain stimuli such as that encountered during routine medical procedures should be explored. Potential sex differences in how such interventions might be delivered should also be considered. Thus, it may be that for girls, optimal effects are found when modification of AS is directed at both parents and children, whereas for boys, it may be more appropriate and cost-effective to focus such interventions on children only.

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