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Anxiety Sensitivity and Fear of Pain in Pediatric Headache Patients

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Abstract

Background—Recent research suggests that anxiety sensitivity (AS) may be a critical factor in the maintenance of chronic pain. Converging lines of evidence also propose a relationship between anxiety sensitivity and fear of pain that may result from interoceptive fear conditioning in interoceptively-biased individuals. While some anxiety sensitivity and fear of pain research has been done in adults, literature exploring this relationship is sparse in clinical pediatric populations.

Methods—This study investigated the hypotheses that fear of pain mediates the relationships between AS and disability as well as AS and somatization in children and adolescents with chronic headache pain. Mediation models were investigated using bootstrap regression analyses.

Results—Results indicate that the anxiety sensitivity-disability relationship is mediated by fear of pain while anxiety sensitivity seems to contribute both directly and indirectly to somatization.

Conclusion—These results provide evidence for the pivotal role of anxiety sensitivity in the pediatric chronic pain model. The findings of this study further emphasize the application of the fear-avoidance model in children, and provide new evidence for the critical role of anxiety sensitivity in a pediatric headache population.

Anxiety sensitivity (AS), which refers to distress related to the potential sensations and consequences associated with anxiety (Asmundson & Taylor, 1996), including heightened interoceptive awareness may be a precursor to the development of pain-related fear where physiological cues are interpreted as precursors to aversive pain-related outcomes. AS has consistently been associated with a greater fear of pain in adult headache (Asmundson *et al.*, 1999), chronic musculoskeletal pain populations, and non-clinical samples (Asmundson *et al.*, 1999; Greenberg and Burns, 2003; Ocanes *et al.*, 2010), linking these overlapping but distinct constructs. Due to this robust association in adult headache populations and the absence of a similarly comprehensive pediatric model, pediatric headache presents as an

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Author Contribution

Both authors contributed significantly to this work, with Laura Simons performing statistical analysis, editing, and approving the manuscript and Stefanie Cappucci drafting, editing, and approving the manuscript. Both authors discussed the results and commented on the manuscript.

appropriate genesis point for the development of this link in pediatric chronic pain. This relationship may result from high levels of interoceptive fear conditioning, such that chronic pain patients with heightened AS may be increasingly associating interoceptive cues with painful experiences (De Peuter *et al.*, 2011). The fear avoidance model, in fact, cites interoceptive fear conditioning as a potential cause for the acquisition of pain-related fear in chronic pain patients (Vlaeyen and Linton, 2012).

Because fear of pain plays such a pivotal role in the maintenance of chronic pain in children, (Asmundson *et al.*, 2012; Simons and Kaczynski, 2012), it is useful to understand any predictors of fear of pain that may render them more susceptible to chronic pain development. Initial inquiries into AS in healthy children and adolescents suggest a relationship between anxiety sensitivity and fear of pain (Muris *et al.*, 2001a; Muris *et al.*, 2001b). These results were confirmed in a small sample of children with chronic pain, though the authors were unable to substantiate an association between AS and disability, likely due the sample size ($n=21$) (Martin *et al.*, 2007).

Relatedly, somatic complaints, enigmatic physiological or bodily symptoms, are also common in highly anxious children (Beidel *et al.*, 1991). Heightened interoceptive awareness may be related to an intensified somatic experience. Given the evidence for the links between AS and somatization (Mahrer *et al.*, 2012), somatization and disability (Harris *et al.*, 2009), and AS and FOP (Muris *et al.*, 2001b) we are also interested in determining if the impact of AS on somatic complaints is FOP mediated.

In the current study, we examined the relationship between AS and fear of pain and their influence on pain-related outcomes (Figure 1). Specifically we are interested in testing whether fear of pain mediates the relationship between anxiety sensitivity and pain related disability, and between anxiety sensitivity and somatic complaints in a large pediatric headache sample.

Methods

Participants

Participants were recruited from the Boston Children's Hospital Pediatric Headache Program during their initial headache clinic appointments. The sample consists of 177 children and adolescents ages 8 to 17 (mean 13.64 ± 2.58 years) who presented with chronic headaches, a basic reading ability, and the capacity to understand questionnaire items. A variety of headache etiologies presented within the cohort including migraine ($n=38$), tension-type headache ($n=36$), both tension and migraine ($n=39$), daily persistent headache ($n=22$), and post concussive headaches ($n=10$), among others (e.g., occipital neuralgia; $n=29$). Participants were generally female (73%) Caucasians (93.8%) with an average pain rating of 5.98 ($SD = 1.62$). Average duration of chronic headaches upon evaluation was 2.79 years ($SD = 3.08$).

Measures and Procedures

Informed consent was obtained from all participants under an institutional review board approved protocol. Pain ratings and all demographic and medical information were extracted

from the medical record. The Functional Disability Inventory (FDI) (Walker and Greene, 1991) and Child somatization Inventory (CSI) (Walker *et al.*, 1991) as measures of functional outcomes were completed as part of the standard clinic evaluation. In addition to these measures, patients were given packets specific to this study that included the Fear of Pain Questionnaire (FOPQ-C) (Simons *et al.*, 2011) and the Child Anxiety Sensitivity Index (CASI) (Silverman, 1991). These measures were all collected on the same day.

Pain Intensity

During an interview with a psychologist, patients were asked to provide their typical pain rating on the standard (Varni *et al.*, 1987) 11-point scale with responses ranging from 0 (no pain) to 10 (most pain possible).

Disability

The Functional Disability Inventory (FDI) is a 15-item scale assessing a child's perception of their activity limitations over the last two weeks. Each item is scored on a scale of 0 ("no trouble") to 4 ("impossible"), and the total scores are computed by summing the items. Higher scores are indicative of greater disability. Internal consistency in the current sample was .90.

Somatization

The Child somatization inventory (CSI) is a 35 item self-report measure that surveys a child's somatic symptoms that are unrelated to any particular illness. Each item is scored based on the frequency the patient experiences each symptom (ie. "Weakness", "dizziness") from 0 ("not at all") to 4 ("a whole lot"). Items are summed to get total score. Higher scores signify higher levels of somatization. The CSI had strong internal consistency in the current sample ($\alpha=.91$).

Pain-related Fear

The Fear of Pain Questionnaire (FOPQ-C) is a 24 item measure assessing self-report of child pain related fears and avoidance behaviors. This study solely utilized the 13-item Fear of Pain subscale, as the Avoidance of Activities subscale was not of interest in the mediation model. Each item (ie "I worry when I am in pain", "Feelings of pain are scary to me") is scored from 0 ("strongly disagree") to 4 ("strongly agree"). Total scores are calculated by summing items. Higher scores indicate higher levels of pain-related fear. The FOPQ-C fear subscale had high internal consistency ($\alpha=.85$) in the current sample.

Anxiety Sensitivity

The Children's Anxiety Sensitivity Index (CASI) is an 18-item self report scale that measures anxiety sensitivity in children and adolescents. Patients rate how negatively they view anxiety symptoms (ie. "It scares me when my heart beats fast", "Unusual feelings in my body scare me") on a scale of 1 ("none") to 3 ("a lot"). There are three subscales in the CASI pertaining to physical, psychological, and social concerns. Total CASI score results from summing all items where higher scores are indicative of higher levels of AS. The CASI had strong internal consistency ($\alpha=.88$) in the current sample.

Statistical Analyses

Data were analyzed using IBM SPSS Statistics (version 21). Descriptive statistics including the means and standard deviations were conducted. Two-tailed Pearson product-moment correlations were used to examine the relationships between all variables except for gender and headache diagnosis that was examined using one-way ANOVA.

Regression analyses using Preacher and Hayes' bootstrap script (Preacher and Hayes, 2008) ($n=5000$ bootstrap samples) were utilized to test the mediation model and assess any indirect effects. The Bias Corrected and Accelerated confidence intervals (BCACI) were set at 99%. Age, average pain, and gender were used as control variables. As seen in Figure 1, mediation is assessed with this method by comparing the total effect (weight c) of an independent variable (IV) on a dependent variable (DV), which is composed of a direct effect (weight c') of the IV on the DV and an indirect effect (weight $a \times b$) of the IV on the DV through a proposed mediation variable (M). Weight a signifies the effect of the IV on the M, whereas weight b signifies the effect of the M on the DV. The indirect effect is calculated from the unstandardized regression weights of paths a and b . If there is a significant amount of variance between the IV and the DV explained by the proposed mediator, the indirect effect is significant (Baron and Kenny, 1986). Bootstrapping produces sampling distributions of the indirect effects by sampling (in this case, $n=5000$) from the data set and calculating the indirect effects present in the resamples. This process produces point estimates that are considered significant if the BCACIs (set at 99%) do not include zero (Xinshu Zhao, 2010).

Initially, the regression tested whether FOP (M) mediates the relationship between AS (IV) and disability (DV). The bootstrapping analysis was repeated to see if FOP (M) mediates the relationship between AS (IV) and somatization (DV). Given the cross-sectional nature of the data, we tested the rigor of our hypothesized pathway with an alternative model with AS serving as the mediator for both outcomes.

Results

Descriptive and Preliminary Correlation Analyses

Table 1 shows descriptive statistics for all study measures. When compared to a healthy sample of children ($n=244$) (Tsao *et al.*, 2009) both AS and somatization were significantly higher ($t=2.96$, $p < .005$; $t=7.03$, $p < .0001$, respectively). For fear of pain, 28% had high levels of fear (FOPQ ≥ 51), 29% had moderate levels (FOPQ =34 to 50), and 42% had low levels of fear (FOPQ ≤ 33) (Simons *et al.*, 2011). Almost half of patients reported moderate disability (48%; FDI =13 to 29), with over a third reporting mild disability (37%; FDI ≤ 12) and a small minority reporting severe disability (15%; FDI ≥ 30) (Kashikar-Zuck *et al.*, 2011). Pain intensity was related to FOP, somatization, and disability, but not AS. Age did not significantly correlate with FOP, somatization, disability, or AS. Using one-way ANOVAs for each variable of interest (FOP, somatization, disability, AS), no differences were observed across gender or pain diagnoses. The correlation analysis age was not significant, nor was the ANOVA results for gender or pain diagnosis. With pain intensity significant and age and gender approaching significance, we included these variables as

covariates in the bootstrapping regression analyses. Significant positive correlations were present between all proposed independent, dependent and mediator variables.

Mediation Models

The bootstrap results for the mediation of the effect of anxiety sensitivity on pain-related disability by fear of pain are shown in Table 2. The total effect of AS on disability was significant (path c, $\beta = .55$, $p < .001$). The relationship between AS and fear of pain was significant (path a, $\beta = .1.00$, $p < .001$), as was the relationship between FOP and disability (path b, $\beta = .39$, $p < .001$). Once fear of pain was accounted for, there was no longer a significant relationship between anxiety sensitivity and disability. The bootstrapping results confirmed the indirect effect of anxiety sensitivity on disability fully explained through fear of pain (BCAC = .15–.66, 99% Confidence Interval [CI]).

The results of the bootstrap analysis of the mediation effect of fear of pain on the anxiety sensitivity – somatization relationship are displayed in Table 3. The direct effect of AS on somatization was significant (path c, $\beta = .93$, $p < .001$). Both the effect of AS on FOP (path a, $\beta = .1.00$, $p < .001$), and the effect of FOP on somatization (path b, $\beta = .44$, $p = .001$) were also significant. With fear of pain in the model AS continued to be a significant predictor of somatization (path c', $\beta = .49$, $p < .05$). Bootstrapped results confirmed an indirect effect of anxiety sensitivity on somatization partially explained through fear of pain (BCAC = .09–.79, 99% CI).

Given the cross-sectional nature of the data we tested the rigor of our hypothesized pathway with an alternative model with AS serving as the mediator for both outcomes. For functional disability, there was no direct effect of anxiety sensitivity on functional disability (path b, $\beta = .16$, $p = .23$) with no evidence of mediation (BCAC = -.06–.24, 99% Confidence Interval [CI]). For somatic symptoms, there was a direct effect of anxiety sensitivity on somatization (path b, $\beta = .67$, $p < .01$) with evidence of partial mediation (BCAC = .01–.51, 99% Confidence Interval [CI]), with fear of pain continuing to be a significant predictor of somatization (path c', $\beta = .44$, $p < .01$). This is consistent with our partial mediation finding above that that anxiety sensitivity has a unique relationship to somatization, beyond fear of pain.

Discussion

We tested the hypotheses that fear of pain mediates the relationship both between anxiety sensitivity and disability, and anxiety sensitivity and somatization. Results supported the first hypothesis, but it appears as though there is both a direct and indirect effect between AS and somatization. The results of this study extend the current state of the literature by affirming the critical role of anxiety sensitivity in the maintenance of pediatric chronic pain. While there has been significant investigation into the role of anxiety sensitivity in adults (Ocanes *et al.*, 2010) and healthy children (Muris *et al.*, 2001b; Tsao *et al.*, 2006; Tsao *et al.*, 2009), this is the first study to our knowledge that explores the relationship between anxiety sensitivity and fear of pain in a pediatric headache population. These results support the literature (Zvolensky *et al.*, 2001; Martin *et al.*, 2007; Simons and Kaczynski, 2012) citing fear of pain as a major factor contributing to the maintenance and exacerbation of

chronic pain and its associated negative outcomes. According to the findings of this study, anxiety sensitivity may be a component central to the maintenance of pediatric chronic pain and the development of fear of pain. While it is not apparent from these results whether AS or FOP may be a better target for early intervention and treatment of disability, given that AS is a trait-like construct, it likely serves as a precursor making people prone to developing FOP.

High levels of anxiety sensitivity may put patients at risk for high levels of interoceptive fear conditioning that likely leads to the development of fear of pain (De Peuter *et al.*, 2011). Modern day interoception refers to the cognizance of physiological sensation, both viscerally and within (Craig, 2002). This encompasses heightened awareness toward general (e.g., increased heart rate) or threat-related (e.g., muscle tension) physiological cues. While the unconscious prioritization of these sensations is essential, a heightened level of interoceptive awareness may be problematic, leaving children and adolescents at risk for chronic pain conditions (De Peuter *et al.*, 2011). In interoceptive conditioning, internal sensations are repeatedly associated with an unconditioned stimulus (De Peuter *et al.*, 2011), in this potential example, pain episodes. In patients who are particularly interoceptively aware, many cues, even sensations that are entirely unrelated to chronic pain episodes (ie. normal muscle tension after exercise) could be seen as a threat. Patients then may begin to dread normal bodily sensations, for fear that they signify the initiation of intense pain. This fear has the potential to lead to avoidance behaviors and eventually disability, in accordance with the results of this study and the fear-avoidance model (Simons and Kaczynski, 2012). Despite the feasibility of this mechanism, future research would need to explicitly examine this explanation as a method of acquisition. Notably, biological vulnerabilities may also help explain the results of this study beyond interoceptive fear conditioning. Most evidence suggests that emotional disorders such as anxiety and depression share a common genetic basis, and that environmental factors may account for observed differences (Barlow 2000). Due to the link between anxiety sensitivity and trait anxiety (Muris *et al.*, 2001), it is not unreasonable that pain related fear could in part be due to a genetic disposition for emotional disturbance, and not the proposed mechanism of interoceptive fear conditioning.

The critical nature of AS is further supported by the results suggesting that anxiety sensitivity predicts somatization both directly and indirectly. A relationship between somatization and disability has been documented in pediatric chronic pain (Harris *et al.*, 2009); because anxiety sensitivity uniquely predicts some degree of somatization outside of fear of pain, AS may be more significant than previously suggested. Fear of anxiety symptoms elucidate the heightened somatization in chronic pain patients. According to this early idea by Asmundon and Norton (1995), patients with high anxiety sensitivity are more likely to be negatively affected by otherwise equal levels of pain intensity. It would follow then, that patients with higher levels of anxiety sensitivity would experience greater negative effects due to somatization, thus accounting for the direct effect of AS on somatization found in these results. Further investigations need to explore the role of interoceptive awareness as it relates to somatization in this context (Schaefer *et al.*, 2012). While AS may propose a path for early intervention, prior to the development of significant amounts of fear, it may be easier to approach treatment from an extinction perspective due to the

proximal nature of FOP and disability. If patients are acquiring this fear through learning and conditioning processes, interoceptive exposure (Keough and Schmidt, 2012; Boswell *et al.*, 2013) may provide a valid route of treatment that significantly decreases the fear of pain and subsequently pain related disability. This could be potentially implemented among pediatric headache patients as it involves exercise to induce physiological disequilibrium (e.g., breathing through a straw).

Several limitations must be considered in the context of this study. Notably, this is a cross-sectional examination, thus we cannot prove causality or directionality between the variables. Future investigation to solidify these relationships between AS, fear of pain, and somatization utilizing methodology that permits experimental manipulation or collection of these data across multiple time points would be useful. A direct investigation into the mechanistic relationship between AS, FOP, and somatization would be useful to determine whether or not interoceptive fear conditioning can explain the relationships discovered in these results. Additionally, an experimental procedure centered on interoceptive exposure may be beneficial in determining how effective these constructs are as treatment targets. This patient population also consists solely of headache patients; while we believe this model would be upheld in an etiologically diverse pediatric pain population, it may be useful to test the applicability of this model with a diverse patient sample in future studies. Additionally, though somatization was approached as a psychological variable in this study, it's true essence is somewhat controversial (Postilnik *et al.*, 2006). While all patients were assessed during their initial visit at PTS, we did not consider the duration of their chronic pain prior to this time, thus it is possible AS, somatization, FOP, and disability could be functions of time. Specifically, as initially proposed by Weems *et al.* (Weems *et al.*, 2002) AS may have both trait and learned characteristics. Anxiety sensitivity may, then, be at least partially dependent on the anxiety-evoking life experiences and may not be stable over time. AS is known to have complex relationships with many other pertinent psychosocial variables; specifically anxiety sensitivity is shown to be a predictor of both catastrophizing (Tsao *et al.*, 2009) and generalized anxiety disorder (Naragon-Gainey 2010). While these variables may influence the mechanism investigated here, their immediate impact on the mechanism is beyond the scope of this study. Finally, it is important to acknowledge that the majority of data was gathered through self-report questionnaires.

The results of this present study identify anxiety sensitivity as a potential risk factor affecting pain-related disability as mediated by fear of pain in a pediatric chronic headache sample. AS may be a potential target for future treatments. Treatments designed to target AS in the context of pain may also prove to be beneficial due to the link between fear of pain and anxiety sensitivity.

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What's already known about the topic?

- A link exists between anxiety sensitivity and fear in adults and healthy adolescents
- Interoceptive fear conditioning is a potential cause for the acquisition of fear of pain

What does this study add?

- Anxiety sensitivity, fear of pain, and somatization are linked in pediatric chronic headache
- Fear of pain appears to explain the link between anxiety sensitivity and disability, whereas there is a direct pathway from anxiety sensitivity to somatization
- Describes anxiety sensitivity in the context of interoceptive fear conditioning

Figure 1.
Hypothesized model of the relationship between anxiety sensitivity and pain-related disability in pediatric chronic headache patients

Table 1

Descriptive statistics and two-tailed Pearson correlations for all variables

Variable	1	2	3	4	5	6	Mean	SD
1 CFOPQ_fear	1.0	.68**	.49**	.49**	.20**	.07	19.18	10.46
2 CASI	–	1.0	.46**	.40**	.07	.14	29.32	7.15
3 CSI	–	–	1.0	.59**	.21**	.13	21.95	15.31
4 FDI	–	–	–	1.0	.22**	.01	17.55	10.72
5 Typical Pain Intensity	–	–	–	–	1.0	.05	5.98	1.62
6 Age	–	–	–	–	–	1.0	13.64	2.58

Note:

**
p < .01

Table 2

Path analysis and bootstrap results for the fear of pain mediated relationship between anxiety sensitivity and disability

Path	β	SE	p	BCACI
(a) Effect of AS on FOP	1.00	.08	< .001	-
(b) Effect of FOP on disability	.39	.05	< .001	-
(c) Total effect of AS on disability	.55	.10	< .001	-
(c') Direct effect of AS on disability	.16	.12	.59	-
(ab) Indirect effect via FOP	.39	.10	-	.15--.66

Note: 99% confidence, with n=5,000 bootstrap resamples; BCACI: Bias Corrected and Assessed confidence intervals.

Table 3

Path analysis and bootstrap results for the fear of pain-mediated relationship between anxiety sensitivity and somatization

Path	β	SE	p	BCACI
(a) Effect of AS on FOP	1.00	.08	< .001	-
(b) Effect of FOP on somatization	.44	.13	.001	-
(c) Total effect of AS on somatization	.93	.14	< .001	-
(c') Direct effect of AS on somatization	.49	.19	< .05	-
(ab) Indirect effect via FOP	.44	.13	-	.13-.80

Note: 99% confidence, with n=5,000 bootstrap resamples; BCACI: Bias Corrected and Assessed confidence intervals.