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Are child anxiety and somatization associated with pain in pain-related functional gastrointestinal disorders?

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Abstract

This study investigated individual and incremental contributions of somatization and trait anxiety to pain report in children with pain-related functional gastrointestinal disorders. Eighty children (7–10 years) with pain-related functional gastrointestinal disorders completed the State-Trait Anxiety Inventory for Children, the Children's Somatization Inventory, and 2-week pain diaries (assessing pain frequency and maximum pain). Hierarchical regressions indicated that both trait anxiety and somatization were significantly related to maximum pain and pain frequency, with somatization explaining more variance. Trait anxiety did not significantly add to prediction above somatization. Assessment of somatization may assist with treatment planning for children with functional abdominal pain.

Keywords

anxiety; functional abdominal pain; pediatric pain; somatization

Introduction

Abdominal pain is one of the most common pain complaints in childhood (Saps et al., 2009). For a large proportion of these complaints, no known medical condition will account for these pains, and no medical treatment is indicated (Subcommittee on Chronic Abdominal Pain, 2005; Thakkar et al., 2007). The Rome diagnostic criteria categorize these pain

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Declaration of conflicting interests

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conditions broadly as pain-related functional gastrointestinal (GI) disorders (painF-GID) (Drossman et al., 2006). Children with painFGID have functional impairments similar in extent to children with organic GI disorders (Varni et al., 2006; Youssef et al., 2006) with many developing chronic symptoms (Walker et al., 2004). Cognitive-behavioral interventions for painFGIDs are increasingly recognized as the front line of treatment (Palermo et al., 2010; Sprenger et al., 2011), and therefore innovations in treatment will be facilitated through better delineation of psychological characteristics that are the most salient targets of cognitive-behavioral interventions.

In adults with medically unexplained symptoms, including painFGID, anxiety and/or somatization are frequently identified as predictors of enhanced pain experience and increased medical treatment-seeking (Boyd et al., 2005; Henningsen et al., 2003; Koloski et al., 2003; Vandvik et al., 2004; Whitehead et al., 1997). Cognitive-behavioral interventions addressing anxiety and/or somatization have been effective at decreasing symptom severity, disability, and healthcare utilization (Allen et al., 2006; Martin et al., 2007). Additionally, greater improvement in functional ability following treatment was found to be correlated with larger reductions in somatization (Creed et al., 2005).

In contrast to the long history of research on anxiety, efforts to understand somatization are more recent. Somatization can be defined as increased perception, awareness, and/or pathological interpretation of unpleasant physiological sensations. Prospective and epidemiological studies and measurement advances (Walker et al., 2009) have provided growing empirical support for the construct of somatization. Somatization has been described in childhood, adolescence, and adulthood and is stable across developmental stages (Dhossche et al., 2001; Steinhausen and Winkler Metzke, 2007). Research provides evidence for cognitive (attribution, attention) as well as perceptual (sensitivity, lowered threshold) models of somatization (Rief and Broadbent, 2007). Evidence of somatization (distress intolerance and increased body vigilance) has been observed to a greater extent in adults with irritable bowel syndrome (IBS) compared to controls (Keough et al., 2011).

As with adults, higher anxiety in children with painFGID is associated with increased pain severity (Saps et al., 2009), frequency, duration (Hyams et al., 1996), chronicity (Mulvaney et al., 2006), and disability (Walker and Greene, 1991; Wendland et al., 2010). Higher somatization is related to increased pain report in healthy children (Rocha et al., 2003), and children with painFGID have higher somatization scores compared to healthy peers and peers with mood and anxiety disorders (Czyzewski et al., 2007; Tsao et al., 2009; Walker et al., 1993). However, less is known about the relation of somatization to pain in *within* the group of children with painFGIDs.

Psychological treatment of children's pain-FGIDs typically targets manifestations of anxiety (body tension, worry, behavioral avoidance) with both cognitive and behavioral strategies (Levy and Walker, 2005; Palermo et al., 2010), while manifestations of somatization (catastrophic thinking about pain, dysfunctional beliefs about pain, misattribution of sensations) are much less frequently targeted. In comparison to the child literature, adult interventions have more examples of direct targeting of somatization ("Sometimes my

stomach just feels this way and it doesn't mean anything is wrong") (Allen et al., 2006; Boyce, 2001; Martin et al., 2007).

Associations between psychological variables and painFGID can be understood in the context of motivational priming theory (MPT) which states that affective experience emanates from two opponent motive systems, appetitive (reward or positive affect) and defensive (threat or negative affect) (Bradley et al., 2001; Lang, 1995). Appetitive activation inhibits and defensive activation facilitates pain or nociception (Rhudy et al., 2005). Thus, persons who tend to experience greater defensive activation, such as during anxiety or somatization, are likely to also experience more pain. Preparedness theory states that a negative association is more easily developed with a fear-relevant stimulus (Seligman, 1970, 1971). A fear-relevant stimulus associated with pain may over time form a stronger association and cause greater defensive activation. Thus, MPT predicts that anxiety and somatization likely cause pain facilitation, and preparedness theory predicts that somatization (which is by definition more fear relevant to pain than is anxiety) may lead to greater defensive activation and pain facilitation; however, research has not assessed this prediction.

Research clarifying relations of anxiety and somatization to pain in children with painFGID will begin to address the question of whether somatization is undertreated compared to anxiety, help identify children at risk for poor outcomes, and further tailor treatments to specifically target psychological variables most related to greater morbidity. This study sought to determine independent contributions of child somatization and trait anxiety to pain in children with painFGID, and to determine if trait anxiety has a significant contribution to pain experience beyond that explained by somatization. We hypothesized that somatization would have greater contributions to pain.

Methods

Participants

Participants were part of a larger study of physiological and psychological characteristics of children with painFGID (Shulman et al., 2007). Children aged 7–10 years with painFGID were recruited from primary and tertiary care clinics in a large academically affiliated pediatric healthcare network. This age range was selected to avoid confounds with the effects of puberty on pain and because children at this age are cognitively able to complete study questionnaires. Chart reviews were conducted by a trained research coordinator to identify potential participants with diagnoses of abdominal pain or irritable bowel syndrome (International Classification of Diseases (ICD)-9 codes 789.0 and 564.00). Parents were contacted by mail and advised to call the research coordinator if interested in participating. Interested participants were screened by telephone to assess inclusion or exclusion criteria. Children were included if, according to parent report, for at least the previous 2 months they had abdominal pain one or more times per week of moderate to severe (3/10) intensity or causing interference with activities (Apley and Naish, 1958; Von Baeyer and Walker, 1999). A chart review was completed to ensure no medical cause for abdominal pain had been identified. Children were excluded due to presence of organic GI illness (or if organic GI illness remained in the differential), other significant chronic health condition (requiring

daily medication or specialty follow-up care), decreased growth velocity, GI blood loss, unexplained fever, vomiting, chronic severe diarrhea, weight loss 5 percent of their body weight within a 3-month period, current use of anti-inflammatory medications, or previous use of GI medication that provided complete relief of symptoms. Children were also excluded if English language fluency or developmental disabilities would interfere with questionnaire completion.

Participants included 114 children and their parents. Twenty patients were excluded for missing questionnaire data and 14 because their diaries (described below) were missing 30 percent or more of the pain ratings. The final sample included 80 participants. An a priori power analysis with G-Power (Faul et al., 2009) indicated that a sample size of 74 participants would provide adequate power. All procedures were approved by the Baylor College of Medicine Institutional Review Board. A trained research coordinator provided an explanation of study procedures and reviewed consent forms prior to obtaining written consent from parents and informed assent from the child.

Procedure

Study procedures were completed during a home visit. Parents provided demographic information and children completed questionnaires in a room independent from their parent. A research assistant explained questionnaires and answered any questions. Questionnaires were read to 7-year-old children to ensure reading difficulties did not affect responses. Parents and children were then instructed (verbally and in writing) regarding completion of the pain diary (kept for 2 weeks following the home visit) and collection of a stool sample (done within 1–2 days of the home visit). A research coordinator contacted each participant weekly to ensure diary completion.

Measures

State-Trait Anxiety Inventory for Children—The State-Trait Anxiety Inventory for Children (STAIC) (Spielberger, 1973) is a measure of general anxiety symptoms designed to be relevant to most children including those with and without physical symptoms. The child-response questionnaire consists of two 20-item sub-scales: state anxiety (context specific) and trait anxiety (stable individual difference in anxiety proneness). This study used trait anxiety, which inquires about how the child “usually” feels. Items consist of emotional statements (e.g. “I worry too much”) and children indicate how often the statement is true for them on a 3-point rating (“hardly ever” = 1 point, “sometimes” = 2 points, and “often” = 3 points). Responses are summed with total scores ranging between 20 and 60 and higher scores indicating greater trait anxiety. T-scores were used for data analyses. The trait anxiety subscale has demonstrated good test–retest (6-week interval; $r_s > .65$) and internal consistency reliability ($\alpha_s > .78$) (Spielberger, 1973). Criterion-related validity has been demonstrated via high correlations with other measures of child anxiety ($r_s > .63$) (Spielberger, 1973).

Children’s Somatization Inventory—The Children’s Somatization Inventory (CSI) (Walker and Garber, 1993) assesses somatization symptoms in pediatric patients and was originally designed for use with children with recurrent abdominal pain. The original child-

report version (35 items) was used. Each item lists a symptom (e.g. “difficulty swallowing,” “headache”) and children indicate on a 5-point Likert scale (0 = “not at all,” 1 = “a little,” 2 = “some,” 3 = “a lot,” and 4 = “a whole lot”) how much they were “bothered by each symptom” during the preceding 2 weeks. Scores range between 0 and 140 with increasing scores indicating greater levels of somatization. The questionnaire has demonstrated good test–retest ($r = .66$) and internal consistency reliability ($\alpha = .92$) (Walker and Garber, 1993). The CSI is correlated with the Child Behavior Checklist (CBCL) Somatic Complaints scale ($r = .42$) suggesting good criterion-related validity (Walker and Garber, 1993).

Pain diary—Participants completed a 2-week paper diary to track abdominal pain. Children were instructed to complete the diary without parental input; however, parents were asked to prompt children to complete it. Three diary ratings were made daily (upon awakening, after lunch, and at bedtime). Children made pain ratings by placing a mark on a 100-mm horizontal visual analog scale (VAS) with anchors of “no pain at all” and “worst pain you can imagine.” Pain intensity was established by measuring the distance in millimeters from the left end of the line to the mark made by the child. Maximum pain rating was the highest pain rating (1–100 mm) given during the 2-week period. Pain frequency was defined as the number of times a child rated pain as ≥ 10 mm on a 100-mm VAS. This cutoff was selected because pain ≥ 10 mm has been accepted in the literature as representing clinically significant mild pain (Jensen et al., 2003; Kelly, 2001).

GI inflammation—Subclinical levels of inflammation have been identified in children with pain/FGID (Shulman et al., 2008), and positive correlations between GI inflammation and somatization in children with functional GI symptoms have been reported (Schurman et al., 2010). Fecal calprotectin concentration has been found to reliably reflect GI inflammation identified during more invasive and extensive testing (Fagerberg et al., 2005). In the absence of a medical diagnosis, pain symptoms caused by GI inflammation may be wrongly attributed solely to psychological factors. Because GI inflammation is a potential confound in our assessment of the association between psychological variables and pain, this study included GI inflammation in the initial step of regression models. A stool was obtained from children by placing a plastic “hat” over the toilet. Fecal calprotectin levels are expressed as micrograms per gram of stool and were analyzed using an enzyme-linked immunosorbent assay (ELISA) technique (Genova Diagnostics, Asheville, NC, USA).

Data analysis

Preliminary analyses—Independent samples *t*-tests were used to compare included and excluded participants on trait anxiety, somatization, GI inflammation, maximum pain rating, and pain frequency. Pearson correlations assessed relations between predictor and outcome variables. Descriptive analyses were conducted for somatization, trait anxiety, and fecal calprotectin.

Primary analyses—Pain outcome data were not normally distributed, and thus, these analyses were completed with Log(e) transformed data. To assess the independent contributions of the psychological variables, two parallel hierarchical regressions were conducted for each pain outcome (maximum pain rating, pain frequency), one with trait

anxiety and another with somatization as a predictor. We included sex as a covariate in analyses since research indicates that girls experience more chronic pain, greater pain intensity, and more pain-related disability compared to boys (Hakala et al., 2002; Merlijn et al., 2003; Roth-Isigkeit et al., 2005). Sex was entered first and fecal calprotectin was entered second in regressions. Trait anxiety or somatization was entered last. A final hierarchical regression was conducted for each pain outcome to assess the additive effect of trait anxiety. Sex and fecal calprotectin were entered in Step 1; somatization was entered in Step 2, followed by trait anxiety in Step 3.

Results

Demographics

Seventy-one percent ($n = 57$) of the participants were girls. The mean age of the participating children was 8.50 years (standard deviation (SD) = 1.11 years). Participants identified themselves as Caucasian (68%), Hispanic American (15%), Black or African American (10%), and Asian American (1%).

Preliminary analyses

There were no significant differences between included and excluded participants on trait anxiety, somatization, GI inflammation, maximum pain rating, or pain frequency ($ts < .68$, $ps > .49$). Table 1 presents correlations between predictor and outcome variables. The mean trait anxiety t -score was 46.93 (SD = 13.42). Fifteen participants (19%) had a trait anxiety score at or above 1 SD above the mean ($t = 60$). Thus, the trait anxiety scores were generally consistent with the normative sample (Spielberger, 1973). The mean somatization (CSI) score was 28.09 (SD = 21.42). Median calprotectin concentration was 38.00 $\mu\text{g/g}$ of stool (range = 15–403 g) with 25 participants (31.25%) having concentrations at or above 50 $\mu\text{g/g}$ of stool, which has been suggested as the upper limit of normal for both adults (Ton et al., 2000) and children (Fagerberg et al., 2003). A post hoc power analysis with G-Power (Faul et al., 2009) indicated an achieved power of .93 or greater for regression analyses.

To ensure pain-related items or symptoms (i.e. headache, lower back pain, stomach pain) on the CSI were not solely responsible for relations between the CSI and pain outcomes, the analyses were also conducted with the eight pain-related CSI items removed from CSI scores. The same pattern of results emerged; results using the full CSI questionnaire are reported to maintain the integrity of the questionnaire.

Prediction of maximum pain rating

Table 2 presents outcomes for regression analyses, including the unique variance accounted for by trait anxiety and somatization in the respective final regression models.

Trait anxiety—The final model was significant and explained 13 percent of the variance in maximum pain ratings. Significant effects were yielded for trait anxiety ($\beta = .30$, $p < .01$).

Somatization—The final model was significant and explained 17 percent of the variance. Significant effects were yielded for somatization ($\beta = .37$, $p < .01$).

Prediction of pain frequency

Trait anxiety—The final model was significant and explained 11 percent of the variance in pain frequency. Significant effects were yielded for trait anxiety ($\beta = .27, p = .02$).

Somatization—The final model was significant and explained 20 percent of the variance in pain frequency. Significant effects were yielded for somatization ($\beta = .41, p < .01$).

Incremental effect of anxiety

Individual models indicated that somatization accounted for more unique variance than anxiety for both pain outcomes. A combined model assessed whether anxiety provided incremental value to the variance accounted for by somatization. Results for the final models are presented in Table 2 and summarized below.

Maximum pain rating—Sex and GI inflammation included in Step 1 ($p = .17$) accounted for 5 percent of variance and yielded no significant correlates of maximum pain ratings. Step 2 was significant ($p < .01$) and yielded a significant effect for the added variable of somatization ($\beta = .37, p < .01$). The model accounted for 17 percent of variance in maximum pain ratings. Step 3 was significant ($p < .01$) and accounted for 20 percent of the variance in maximum pain ratings, but did not yield a significant change in variance accounted for ($p = .14$). A significant effect was yielded for somatization ($\beta = .29, p = .02$).

Pain frequency—Sex and GI inflammation included in Step 1 ($p = .25$) accounted for 4 percent of variance and yielded no significant correlates of pain frequency. Step 2 was significant ($p < .01$) and yielded a significant effect for the added variable of somatization ($\beta = .41, p < .01$). The model accounted for 20 percent of variance in pain frequency. Step 3 was significant ($p < .01$) and accounted for 21 percent of the variance in pain frequency, but did not yield a significant change in variance accounted for ($p = .33$). A significant effect was yielded for somatization ($\beta = .36, p < .01$).

Discussion

With awareness of the need to continually improve targets of cognitive-behavioral pain treatment for children, this study compared the contributions of trait anxiety and somatization to pain complaints in a group of young children with painFGID. After controlling for sex and GI inflammation, somatization, compared to trait anxiety, was more strongly associated with pain in children with painFGID, suggesting that children who are prone to report multiple somatic complaints have more intense and frequent abdominal pain. Not only did somatization explain a greater amount of variance in pain than did trait anxiety, when both constructs were entered into a combined model, trait anxiety did not significantly add to prediction above and beyond somatization. These data are consistent with findings from a study in adults which found somatization but not trait anxiety was related to outcomes in irritable bowel syndrome (Boyd et al., 2005). While research has demonstrated elevated levels of somatization in children with painFGID compared to peers without painFGID (Czyzewski et al., 2007; Walker et al., 1993), this study extends those findings by demonstrating that *within* a group of children with painFGID greater somatization is related

to increased pain. The present findings are consistent with MPT and preparedness theory which together predict that trait anxiety and somatization should lead to defensive activation and resulting pain facilitation with a stronger relation between somatization, compared to anxiety, and pain.

Psychological variables are believed to influence pain via supraspinal pathways (periaqueductal gray (PAG), rostroventromedial medulla (RVM), and dorsal horn of spinal cord) which can cause both facilitation and inhibition of pain. Imaging studies demonstrate that distraction from pain leads to pain inhibition via activation of PAG–RVM pain inhibitory pathways (Valet et al., 2004). Furthermore, pain itself is a physiological stressor which may itself increase pain facilitation (Rhudy et al., 2005). Thus, to the extent a child with high somatization is overattending to physiological sensations, they are likely to have decreases in supraspinal inhibition of pain and increases in supraspinal facilitation of pain, and therefore augmented pain experience overall.

This study has implications for treatment of children with painFGID, especially within a medical setting. Trait anxiety in this population typically does not reach clinically significant thresholds, as was the case in this study. Although anxiety may sometimes be more readily apparent, and is a concept more familiar to medical and lay-persons, somatization may provide a more specific representation of how psychological factors exacerbate pain. Failure to recognize a child's tendency to overattend to and make negative attributions about bodily sensations could result in inadvertent reinforcement of this characteristic by attention (even through repeated reassurance) from healthcare professionals—counter to what may be most helpful for pain management. Cognitive strategies targeting somatic focus and dysfunctional beliefs about physical sensations may improve therapeutic outcomes above those achieved by treatment of anxiety and pain management strategies alone. Furthermore, measures of somatization may help identify children with abdominal pain who are at risk for more impairing or chronic symptoms.

An increased tendency toward somatization has been recognized in mothers of children with painFGID (Czyzewski et al., 2007; Van der Veek et al., 2011; Walker and Greene, 1989), suggesting these mothers may be prone to attending to their children's symptoms. A healthcare provider's counsel to the parent to pay less attention to their child's symptoms is likely to be an important component of intervention and requires that the physician be aware of the relation between pain complaints and somatization, not just the relation between pain complaints and anxiety.

While some evidence supports that persons with GI inflammation report increased pain compared to those without GI inflammation (Schurman et al., 2010), this was not supported by the present data. Thirty-one percent of participants had GI inflammation, but this was not a significant contributor to pain report. Similarly, while gender differences in pain are often found, gender did not contribute unique variance in predicting these outcomes. The relatively young age of our sample may account for this lack of difference since most pediatric functional abdominal pain samples in the literature include both children and adolescents.

Use of daily diaries to gather data regarding pain is a strength of this study. The diary helps overcome potential inaccuracies inherent in retrospective self-report data, particularly with children who may be poor reporters of past symptoms. However, any child self-report data, including diaries, should be interpreted with caution as they rely on a young child's ability to accurately and reliably report symptoms.

Limitations of this study include a relatively small sample size with more female than male participants. Although inclusion of only 7- to 10-year-old children limits the generalizability to children outside this age range, it does provide a more focused understanding of prepubescent children with painFGID, a less studied population. Finally, future research should seek to replicate these results with multiple methods of assessment since reliance only on children's report of symptoms is a potential limitation.

The results of this study suggest several avenues for future research to better understand and treat painFGID in children. Future studies should compare the efficacy of psychological treatments targeting somatization versus anxiety in treatment of children with painFGID to determine if specifically targeting somatization leads to better outcomes. Future research should seek to understand whether somatization and/or anxiety are predictive of long-term pain outcomes, predispose children to painFGID, or are the result of frequent pain experiences. Regardless, these data illustrate the importance of including the assessment of somatization symptoms in research and treatment of patients with abdominal pain.

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Table 1

Correlations between predictor and outcome variables.

	GI inflammation	Trait anxiety	CSI	Maximum pain
Trait anxiety	-.01			
CSI	.23 *	.43 *		
Maximum pain	.11	.31 *	.38 *	
No. of pain episodes	.02	.28 *	.40 *	.74 *

GI: gastrointestinal; CSI: Children's Somatization Inventory.

*
 $p < .05$.

Table 2
Hierarchical regressions of individual and incremental contributions of somatization and trait anxiety.

Variable	R ²	R ² change	F	β	t	% Unique variance
Regressions assessing individual contributions of variables						
Maximum pain report (0–100 mm VAS)						
Trait anxiety regression	0.13		3.82*			
Step 1 Sex				0.16	1.44	
Step 2 GI inflammation				0.13	1.19	
Step 3 Trait anxiety		0.09		0.30	2.75*	8.64
Somatization regression	0.17		5.25*			
Step 1 Sex				0.17	1.64	
Step 2 GI inflammation				0.04	0.38	
Step 3 Somatization		0.13		0.37	3.41*	12.67
Number of pain episodes						
Trait anxiety regression	0.11		3.09*			
Step 1 Sex				0.17	1.53	
Step 2 GI inflammation				0.03	0.31	
Step 3 Trait anxiety		0.07		0.27	2.50*	7.29
Somatization regression	0.20		6.16*			
Step 1 Sex				0.18	1.75	
Step 2 GI inflammation				−0.06	−0.59	
Step 3 Somatization		0.16		0.41	3.89*	16.00
Regressions assessing incremental value of trait anxiety						
Maximum pain report (0–100 mm VAS)	0.20		4.56*			
Step 1 Sex				0.17	1.66	
GI inflammation				0.06	0.56	
Step 2 Somatization		0.21		0.29	2.43*	6.30
Step 3 Trait anxiety		0.01		0.17	1.50	2.40
Number of pain episodes	0.21		4.86*			

	Variable	R^2	R^2 change	F	β	t	% Unique variance
Step 1	Sex				0.18	1.76	
	GI inflammation				−0.05	−0.47	
Step 2	Somatization		0.21		0.36	3.05*	9.86
Step 3	Trait anxiety		0.00		0.11	0.99	1.04

VAS: visual analog scale; GI: gastrointestinal.

R^2 change is provided for trait anxiety and somatization.

* $p < .05$.