Models of Anxiety, Depression, Somatization, and Coping as Predictors of Abdominal Pain in a Community Sample of School-Age Children

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Objective To examine whether somatization mediates the relationship of coping styles and internalizing problems with abdominal pain. Methods 230 school children (M age = 11.80 years; 43.8% male; 21.3% White) completed measures of coping style, anxiety, and depression early in the school year, and subsequently reported abdominal pain symptoms weekly. Results The results showed (a) the association of anxiety and depression with abdominal pain may be mediated by somatization; (b) there are similarities and differences in the association of coping styles with pain for models including anxiety versus depression. Significant indirect effects showed higher levels of passive coping were associated with more pain via somatization and either anxiety or depression. For active coping, results differed for models including anxiety versus depression. Accommodative coping showed no independent relationship with abdominal pain. Conclusions Somatization may mediate the relationship of internalizing symptoms and coping styles with pain. Treatment implications are discussed.

Key words abdominal pain; anxiety; coping styles; depression; somatization.

Recurrent abdominal pain, that is, pain occurring at least three times over at least 3 months that interferes with the child’s activities (Apley & Naish, 1958), is common (Saps et al., 2009) and reported to occur in 7%–25% of school children and adolescents (Campo et al., 2004). In an “overwhelming majority” of cases (Campo et al., 2004), there is no medical explanation, and the medically unexplained symptoms (MUS) are termed “functional abdominal pain (FAP).” The Rome III criteria for the abdominal pain–functional gastrointestinal disorders include categories for functional dyspepsia, irritable bowel syndrome, abdominal migraine, FAP, and FAP syndrome (Rasquin et al., 2006).

Numerous studies have found an association between anxiety and/or depression with FAP in both clinic (Campo et al., 2004; Dufton, Dunn, & Compas, 2009; Garber, Van Slyke, & Walker, 1998; Garber, Zeman, & Walker, 1990; Liakopoulou-Kairis et al., 2002; Saps et al., 2009; van der Veek, Derkx, de Haan, Benninga, & Boer, 2010; Varni et al., 1996; Zuckerman, Stevenson, & Bailey, 1987) and community studies (Saps et al., 2009; van der Veek et al., 2010; Walker, Smith, Garber, & Van Slyke, 1997). The psychological processes underlying that association, however, are not well-delineated. Factors such as the child’s tendency to somatize and coping styles may affect the associations of anxiety and depression with pediatric...
abdominal pain. The present study sought to develop and test the goodness of fit of models that integrate existing findings about the relationships of anxiety and depression, somatization, and coping styles with abdominal pain in a community sample of school children. Because this model has not been proposed before, it is important to show that there is empirical support for the various components of that proposed models (Joreskog, 1993; Maruyama, 1998) presented in Figures 1 (Model 2) and 2 (Model 3).

**Somatization**

One potential mediator of the relationships of anxiety and depression with abdominal pain is the child’s tendency to experience somatic sensations in general, and not just sensations specific to abdominal pain. While somatoform disorders (American Psychiatric Association, 2000) are characterized by the presence of physical symptoms not fully explained by a general medical condition, there are individual differences in the tendency to experience MUS, and community studies show wide individual differences in the number of somatic symptoms children and adolescents report (Garber, Walker, & Zeman, 1991; Meesters, Muris, Ghys, Reuermers, & Rooijmans, 2003). Rief and Barsky (2005) proposed a biopsychosocial, perceptual-filtering model of MUS in which a neural filtering process regulates the individual’s awareness of physical sensations, with anxiety and depression among the factors hypothesized to affect the filtering processes and modulate awareness of physical sensations. Consistent with that model, several studies have reported statistically significant, low to moderate (Garber et al., 1991; Kaczynski, Simons, & Claar, 2011; Litcher et al., 2001; Meesters et al., 2003; Muris, Vlaeyen, & Meesters, 2001; van der Veek et al., 2010; Vla et al., 2009; Walker, Garvey, & Greene, 1991) correlations between anxiety and somatic complaints. Other studies have shown an association between a tendency to somatize and experience abdominal pain (Dufton et al., 2009; El-Metwally, Halder, Thompson, Macfarlane, & Jones, 2007; Garber et al., 1991; van der Veek et al., 2010). Thus, there is empirical support for the components of a mediational model, not yet tested directly, in which emotional factors are associated with abdominal pain via somatization (e.g., anxiety/depression → somatization, somatization → pain).

**Coping With Pain**

Coping, defined as “voluntary efforts to regulate emotion, thought, behavior, physiology, and the environment in response to stressful events or circumstances” (Thomsen et al., 2002), is thought to be associated with increased or decreased levels of pain (Compas & Thomsen, 1999). Presently, two different, but related, models of coping processes have been examined most commonly in studies of pediatric abdominal pain. Walker et al. (1997) identified three styles of coping: active, passive and accommodative. Active coping involves the child’s efforts to reduce or eliminate pain and includes strategies to solve the problems causing distress, seek emotional or instrumental support, or use distraction to manage pain. Accommodative coping involves the child’s efforts to adjust to pain that cannot be eliminated by using emotion-focused strategies, and involves cognitive strategies to accept pain, minimize pain, or use self-encouragement to deal with pain. Passive coping involves the child feeling or responding passively, not engaging in coping strategies, having negative cognitions and “catastrophizing” the effects of pain, and feeling no control over the pain. Compas and colleagues (2006) identified a model including three factors similar to Walker et al.’s, with primary control engagement coping similar to active coping, secondary control engagement coping to accommodative coping, and disengagement to passive coping.

The number of studies examining the relationship between coping and abdominal pain is limited (Kaczyński et al., 2011). In addition, the results are sometimes contradictory, and possible suppression effects may obscure findings. In two samples, active coping was associated with more abdominal pain (Walker et al., 1997), while in two others, active or primary engagement coping was not associated with abdominal pain (Thomsen et al., 2002; Walker et al., 1997). Results for accommodative or secondary engagement coping were also inconsistent, with positive correlations in some samples (Walker et al., 1997), negative correlations in others (Thomsen et al., 2002; Walker et al., 1997), and nonsignificant relationships in yet others (Walker et al., 1997). Results are more consistent for passive coping. Generally thought to be an ineffective strategy, higher levels of passive coping were associated with increased pain in the three samples studied by Walker et al. (1997) as well as in the sample in the report by Thomsen et al. (2002).

In the studies on coping described above, coping styles were studied separately, an approach that does not allow for an estimate of the independent relationship of each style of coping to pain. Frequently, results differ when the independent associations of each coping style with an outcome are assessed (Gaylord-Harden, Cunningham, Grant, & Holmbeck, 2010). For example, Thomsen et al. (2002) found that the correlations of primary and secondary engagement coping with pain were nonsignificant when those two coping styles were examined separately, but the
relationship to pain for both primary and secondary engagement coping was statistically significant when examined simultaneously in regression analyses. Differences in results when predictors are studied separately rather than simultaneously may be owing to statistical suppression, and careful examination of suppression effects may improve understanding of the relationship between variables (Gaylord-Harden et al., 2010).

Coping and Somatization
In other reports, the relationship between coping styles and somatization, but not pain, has been examined. Similar to the results for coping with pain, the results (a) are inconsistent for active and accommodative coping, while passive coping is consistently associated with more somatization when the relationship of each coping style and somatization is examined separately (Shirkey, Smith, & Walker, 2011; Walker, Smith, Garber, & Claar, 2005; Walker et al., 1997); (b) differ substantially when the coping styles are studied separately rather than simultaneously (Compas et al., 2006).

Coping With Anxiety and Depression Among Abdominal Pain Patients
Studies of the association of coping with anxiety and depression yield more consistent results than for coping with pain. Thomsen et al. (2002) found that primary and secondary control coping were associated with reduced anxiety and depression, while disengagement coping was not. Compas et al. (2006) found that secondary, but not primary, coping was associated with lower levels of anxiety and depression. The relationship between disengagement coping with anxiety and depression again seemed to involve suppression effects, with the correlations between disengagement coping and anxiety/depression being negative, while the path coefficients were positive. Walker et al. (1991) found that passive coping was associated with an increase in depressive symptoms, but active coping was not. Accommodative coping was not examined.

The Present Study
To summarize, existing studies have demonstrated that there is a consistent association between anxiety and
depression with somatization and with abdominal pain, as well as for somatization with abdominal pain. Prior studies, however, have not examined whether somatization might mediate the relationships of anxiety and depression with abdominal pain. Prior studies have also examined the association between coping with pain, coping with somatization, and coping with internalizing symptoms. Generally, significant associations are found, but the directions of effects for coping with pain and somatization are somewhat inconsistent, possibly due to suppression effects. When suppression occurs, a careful examination of the relationships between variables may have important theoretical implications.

In the present study, we took a two-step approach. First, we examined whether somatization mediated the emotion–pain relationship and, if so, whether it did so “partially” (i.e., the paths from anxiety or depression to somatization and from somatization to pain were significant, while the path from emotion to pain remained significant, as well) or “fully” (the direct path from emotion to pain was both small and nonsignificant when the indirect path was included). This step was needed to determine if a direct path from the emotional factor to pain should be included in the subsequent model. Prior research has shown that anxiety and depression are correlated and associated with a higher-order internalizing disorders construct. Nonetheless, because there are advantages to treating them as separate constructs (Cole, Peeke, Martin, Truglio, & Serczynski, 1998), we tested separate models for anxiety and depression. We hypothesized that there would be good fit for models in which somatization mediated the emotional factor/pain relationship but did not have a specific hypothesis about “full” versus “partial” mediation. Subsequently, we examined models of the relationships between emotions, somatization, coping styles, and abdominal pain. Prior studies suggested that each of the coping styles may be associated (a) directly with abdominal pain and (b) indirectly with abdominal pain via their effects on the regulation of anxiety, depression, and somatization.

The preponderance of existing studies suggested that the naturally occurring coping efforts of school children are associated with more subsequent abdominal pain. We hypothesized that a model examining the independent contributions of coping styles via direct effects on abdominal pain and indirect effects via emotion regulation and abdominal pain would show good model fit, with coping effects associated with higher levels of abdominal pain. Mediation is a process in which the relationship between variables X and Y is affected by variable M. That is, if variable X affects variable M, and variable M, in turn, affects variable Y, mediation is present. If the path X → M → Y is significant, then a significant indirect effect is said to be present. If the path X → Y is also significant, then there is also a significant direct effect (Holmbeck, 1997). In this report, we were interested in identifying whether there were significant direct or indirect effects on pain via coping, anxiety/depression, and somatization. Finally, if models showed good fit, we planned to determine if (a) more parsimonious models improved model fit and (b) the basic model was superior to an available alternative model.

The present study was conducted with a diverse community sample of children. A major advantage of studying a community sample is that a full range of the frequency of abdominal pain can be examined. In contrast, participants selected from a clinic sample are likely to have high levels of abdominal pain sufficiently impairing to lead to medical consultation. In clinical samples, comorbidity levels may be inflated (Caron & Rutter, 1991), and the number of children with anxiety, depression, and tendencies to somatize at high levels will increase compared with community samples. This may also attenuate the range of symptoms of anxiety, depression, and somatization and make relationships among those symptoms more difficult to identify. Findings from community studies are also applicable to clinical samples for the obvious reason that the “clinic” children were “community” children before seeking consultation.

Method
Participants
This report is part of a series examining the prevalence of abdominal pain (Saps et al., 2009), headache (Nyame et al., 2010), and the structure of somatization (Lavigne, Saps, & Bryant, 2012) in school-age children. At two public schools, 495 families were mailed study information, with 233 (47.1%) returning signed consents and completing study measures. Three children reporting no pain episodes were eliminated. For the 230 children reporting at least one episode of pain, the mean age was 11.74 years (range 8–15 years), and 99 (43.0%) were male. The sample was diverse: 47 (20.4%) White, 75 (32.6%) Black, 51 (22.2%) Latino, 19 (8.3%) Asian, 5 (2.2%) mixed race, and 25 (10.9%) Other, with 8 (3.5%) providing no information about ethnicity.

Measures
Children’s Somatization Inventory
The Children’s Somatization Inventory (CSI; Garber et al., 1991; Walker, Beck, Garber, & Lambert, 2009) is a
child-completed questionnaire assessing the perceived severity of 35 somatic symptoms derived from the list of symptoms for somatization disorder in DSM-III-R, with additional items from the Hopkins Symptom Checklist and one item, constipation, added because of its frequent occurrence in patients seen in pediatric gastrointestinal clinics. Children are asked to describe “how much you were bothered by” each symptom, with responses ranging from “not at all” to “a whole lot.” The 24 items of the revised CSI-24 were used in analyses. The CSI-24 includes an item on abdominal pain. In the present analyses, that item was removed to avoid inflating the association between somatization and abdominal pain due to item contamination of the scales. In the present study, internal consistency (i.e., Cronbach’s alpha) of the CSI-24 was 0.92. Validity has been demonstrated by showing the CSI is correlated as expected with measures of anxiety, depression, functional impairment, and quality of life (Lavigne et al., 2012).

State-Trait Anxiety Inventory
The State-Trait Anxiety Inventory (Spielberger, Edwards, Lushene, Montuori, & Platzeck, 1973) is a self-report measure of children’s anxiety. Internal consistency in prior studies ranges from 0.82 to 0.87, concurrent validity between 0.63 and 0.75, and construct validity, 0.29–0.54 (Myers & Winters, 2002). Internal consistency in the present report was 0.82. Anxiety scales contain somatic symptoms that may overlap with somatization. These items, however, are intrinsic to both anxiety and somatization, so their removal from the anxiety scale may not be theoretically justified. Anxiety with and without somatization items are highly correlated (Table I). We examined with and without somatic items on the anxiety scale and did not find important differences. Models reported herein included the somatic items on the anxiety scale.

Children’s Depression Inventory
This widely used, 27-item self-report scale of depression for children and young adolescents (Kovacs, 1985) has alpha coefficients from 0.59 to 0.88 in prior studies ranges. Concurrent validity is described as moderately high (Myers & Winters, 2002), and construct validity has been demonstrated in studies examining its relationship to self-esteem, locus of control, cognitive distortions, and underachievement (Myers & Winters, 2002). Internal consistency in the present report was 0.88.

Pain Response Inventory
The Pain Response Inventory (PRI; Walker et al., 1997) assesses children’s coping responses to pain and yields a measure of the degree to which the child engages in active, accommodative, or passive coping strategies. The scales show good internal consistency (active, 0.68–0.76; accommodative, 0.77–0.82; passive, 0.64–0.78). Validity has been demonstrated by showing the PRI is correlated as expected with measures of pain, functional impairment, and somatization (Walker et al., 1997). This measure has been used by the test developers in a sample of community school children (Walker et al., 1997).

Pain Rating
Pain ratings were obtained weekly for 24–36 weeks. Similar to the wording for the CSI, children reported at the end of each week how much they were “bothered by” stomach aches or pain during that week on a 5-point Likert-scale (0 = not at all; 5 = a whole lot). Mean pain score was calculated across rating periods for each child.

Procedure
Parents of children in grades 3 through 8 in two public schools were invited to participate by mail. Children for whom written consent was obtained then completed questionnaires at their school. Research assistants then

Table I. Correlations Between Measured Indicators

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pain</th>
<th>Anxiety</th>
<th>Anxiety-no somatic</th>
<th>Depression</th>
<th>Depression-no somatic</th>
<th>Somatization</th>
<th>Active coping</th>
<th>Accommodative coping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td>0.31**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety-no somatic symptoms</td>
<td></td>
<td>0.30**</td>
<td>0.98**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td>0.36**</td>
<td>0.68**</td>
<td>0.66**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression-no somatic symptoms</td>
<td></td>
<td>0.32**</td>
<td>0.63**</td>
<td>0.62**</td>
<td>0.98**</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatization (CSI-24)</td>
<td></td>
<td>0.58**</td>
<td>0.51**</td>
<td>0.49**</td>
<td>0.53**</td>
<td>0.47**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Active coping</td>
<td></td>
<td>0.14*</td>
<td>0.18*</td>
<td>0.19**</td>
<td>−0.04</td>
<td>−0.03</td>
<td>0.26**</td>
<td>1</td>
</tr>
<tr>
<td>Accommodative coping</td>
<td></td>
<td>0.21**</td>
<td>0.30**</td>
<td>0.30**</td>
<td>0.24**</td>
<td>0.23**</td>
<td>0.29**</td>
<td>0.56**</td>
</tr>
<tr>
<td>Passive coping</td>
<td></td>
<td>0.25**</td>
<td>0.57**</td>
<td>0.56**</td>
<td>0.53**</td>
<td>0.50**</td>
<td>0.48**</td>
<td>0.32**</td>
</tr>
</tbody>
</table>

*p < .01; **p < .01.
returned weekly for the next 24–36 weeks to obtain reports of weekly pain. The study was approved by the medical center’s institutional review board.

**Data Analysis**

Structural equation modeling (SEM) using LISREL 8.8 (Joreskog & Sorbom, 2006) was used to examine the models. We followed Brown’s method (Brown, 2006) in reporting the $\chi^2$ but not interpreting its statistical significance because its value is inflated by large sample sizes. The Satorra–Bentler (SB) $\chi^2$ is reported because it adjusts the $\chi^2$ and standard errors for multivariate kurtosis that may be present when all variables are not normally distributed. Multiple fit indices were used, including an index of absolute fit [standardized Root Mean Square Residual (SRMR)], an index adjusting for model parsimony [root mean square error of approximation (RMSEA)], and comparative fit indices [non-normed fit index (NNFI) and comparative fit index (CFI)]. Criteria for good-fitting models were SRMR < 0.08, RMSEA approximately 0.06 or lower, NNFI > 0.9, CFI > 0.9. Models were judged as showing “acceptable” fit if they met the following criteria: RMSEA < 0.08, NNFI ≥ 0.95, and CFI ≥ 0.95 (Browne & Cudeck, 1993; Hu & Bentler, 1999), SRMR < 0.08 (Brown, 2006). Completely standardized beta weights are reported as path coefficients.

While modeling causal chains including a single mediator is common, modeling “long chains” including two or more mediators is much less common. For multiple mediators, Taylor, MacKinnon, and Tein (2008) recommend the joint significance test, in which evidence for mediation is present if each component path in the mediated pathway is statistically significant. All constituent paths must be significant for the indirect effect to be significant, and the rejection rate of the null hypothesis is the product of the probabilities of the $p$-values of the individual path coefficients. The joint significant test controls well for Type I error (Taylor et al., 2008).

**Results**

**Preliminary Analyses**

As expected, correlations (Table I) between both anxiety and depression scales with and without somatic items were high ($r = .98$ for both), coping styles showed low to moderate (.32–.56) correlations with one another, and anxiety and depression were significantly correlated with pain (>.30) and somatization (>.50). Coping styles showed significant, low correlations with pain (.14–.25) and somatization (.26–.48), with more coping efforts associated with higher levels somatization and being bothered by pain. Coping styles showed a significant positive association with anxiety (.18–.57). Accommodative (.23) and passive (.53) coping were associated with significantly higher levels of depression, and active coping was unrelated to depression.

The mean pain score was .58 ($SD = 0.50$, Range = 0.09–2.75). All children had at least one episode of pain that bothered them at least “a little,” 80.1% had at least one episode that bothered them at least “some,” 51.3% that bothered them “a lot” or a “whole lot,” and 34.8% that bothered them “a whole lot.” There were no sex, $t(228) = -.46$, $p = .64$, or race, $F(4) = 0.118$, $p = .98$, differences in mean pain score, and the correlation between age and mean pain score was not significant, $r = -.02$, $p = .76$.

**Anxiety**

**Somatization as a Mediator of the Relationship Between Anxiety and Subsequent Abdominal Pain**

The first model (Model 1; Figure 1) posited that anxiety had a direct effect on pain and an indirect effect on pain via somatization. Path coefficients from anxiety to somatization and somatization to pain were large, positive, and significant. The path from anxiety to pain was not significant. As a just-identified model, fit statistics are not informative.

Because the direct effect for anxiety with pain was not significant, we also tested the more parsimonious model in which anxiety had an indirect effect on abdominal pain but no direct effect. This model showed a good fit, $SB\chi^2(1) = 0.14$, $p = .70$; RMSEA = 0.00; NNFI = 1.0; CFI = 1.0; SRMR = 0.01. Because that model is a nested version of Model 1, the two models can be compared using the corrected $\chi^2$ difference test (Bryant & Satorra, 2012), which showed no significant difference between the two models, $\Delta SB\chi^2(1) = 0.15$, $p > .66$. As a result, the more parsimonious model without a direct path from anxiety to pain is preferred.

These models are consistent with the notion that anxiety increases bodily signals that can then “break through” the perceptual filter. For most structural equation models, there are alternative models that can be supported by competing theories (Kline, 2011). When this situation exists, it is important to test plausible alternative models. A plausible alternative model suggests that anxiety affects the filtering process directly and mediates the relationship between somatic signals and pain. A model (not shown) in which somatization had an indirect effect on pain via anxiety was then tested, but showed poor model fit, $SB\chi^2(1) = 17.12$; RMSEA = 0.26; NNFI = 0.69; CFI = 0.90; SRMR = 0.17. Thus, the indirect effect model...
for anxiety on pain via somatization was preferred and used in subsequent analyses of coping effects.

**Anxiety, Coping, and Somatization as Predictors of Abdominal Pain**

Model 2 (Figure 1) examined (a) the effect of anxiety via somatization on subsequent pain without including a direct effect of anxiety on pain; (b) active coping influencing anxiety (via the active coping component involving seeking emotional support), and both somatization and pain (via the distraction component of active coping); (c) accommodative coping affecting anxiety (via the components of accommodative coping involving cognitive restructuring and self-encouragement to reduce catastrophic thinking) and both somatization and pain (via the minimizing and ignoring components of accommodative coping); and (d) passive coping affecting anxiety (via increasing anxiety by disengagement, catastrophizing) and both somatization and pain (via disengagement, self-isolation, catastrophizing).

Model 2 showed a good overall fit, $SB\chi^2(1) = 0.44$; RMSEA = 0.00; NNFI = 1.0; CFI = 1.0; SRMR = 0.01. The model shows that (a) anxiety has a significant indirect effect on pain via general somatization (indirect path coefficient = .01, $p < .001$); (b) more active coping is associated with increased somatization but not with anxiety. Active coping is associated independently with higher levels of pain via somatization (indirect effect path coefficient = .08, $p < .001$); (c) accommodative coping is not associated independently with anxiety, somatization, or pain; (d) passive coping is associated directly with increased anxiety, but not with somatization or pain. However, passive coping shows a significant indirect effect on pain via a long-chain path including anxiety and somatization (indirect path coefficient = .14, $p < .001$), with more passive coping predicting more somatization.

We considered the possibility that there were suppression effects in Model 2 because (a) the correlation of active coping with anxiety was positive and significant, but the path coefficient in the path model was negative; (b) the correlation of active coping with pain was positive, but the relationship was negative in the path model; and (c) the correlation between passive coping and pain was positive and significant, but the path coefficient in the path model was negative. To determine if suppression occurred, each coping style was examined as a predictor of anxiety separately and then when all three were predictors of anxiety simultaneously. If suppression occurred, the path coefficient would be significantly greater for one of the coping variables when modeled simultaneously rather than separately. This did not occur. Each path coefficient declined when examined independently versus simultaneously: for active coping with anxiety, the coefficient declined from .18, $p < .001$, to .02, $p = .30$; for accommodative coping, .30, $p < .001$, to .03, $p = .64$; for passive coping, .57, $p < .001$, to .22, $p < .001$). Because all the paths for coping to anxiety decreased in magnitude, the pattern of change is better described as redundancy than suppression (Paulhus, Robins, Trzesniewski, & Tracy, 2012). Similarly, we examined the possible suppression or redundancy effects of coping style with pain. Somatization and all three coping styles were modeled as predictors that possibly had a direct association with pain. Analyses of each combination of coping predictors of pain separately and simultaneously showed no increase in the magnitude of the path coefficient for any predictor, a pattern not consistent with suppression.

To address these redundancies, nonsignificant paths were eliminated from Model 2. The resulting, more parsimonious model included direct paths from active coping to pain, passive coping to anxiety, anxiety to somatization and somatization to pain, as well as a long-chain indirect path from passive coping to pain via anxiety and somatization. This model showed good model fit overall, $SB\chi^2(5) = 7.58$; RMSEA = 0.05; NNFI = 0.99; CFI = 0.99; SRMR = 0.04). However, a $\chi^2$ difference test comparing the parsimonious model with Model 2 showed better fit for Model 2, $\Delta\chi^2(4) = 11.26, p < .05$. Thus, the full Model 2 is preferred over the more parsimonious model. The full Model 2 may show a better model fit because the total indirect effects of passive coping on pain in Model 2 are significant (standardized indirect effect = 0.18, $p < .001$) but the path for passive coping to somatization was removed in the more parsimonious model.

**Alternative Model**

Kaczenski et al. (2011) recently examined a model in a cross-sectional design with a clinical sample of pain patients in which (a) anxiety had a direct effect on somatic complaints, and (b) active, accommodative, and passive coping mediated the relationship between anxiety and somatic complaints rather than having an effect on anxiety. Following from that work, we examined a model in which (a) anxiety had a direct effect on somatic symptoms and an indirect effect on abdominal pain via somatization, (b) accommodative and passive coping mediated the relationship between anxiety and somatic complaints, and (c) accommodative coping had a direct effect on pain. Because of the redundancies noted early, active coping and the path from passive coping to pain were eliminated. This model, however, showed a poor overall model fit, $SB\chi^2(3) = 40.89$;
RMSEA = 0.23; NNFI = 0.69; CFI = 0.91; SRMR = 0.09. Thus, Model 2 is preferred.

**Depression**

Somatization as a Mediator of the Relationship Between Depression and Subsequent Abdominal Pain

The analyses above were repeated using depression rather than anxiety as an exogenous predictor. A just-identified model (Model 1, Figure 1) showed significant paths for depression to somatization, and somatization to pain, but the path from depression to pain was not significant. As we found for anxiety, a model (not shown) with an indirect path from depression to pain via somatization and without a direct path from depression to pain showed a good fit, $\chi^2(1) = 1.15$; RMSEA = 0.02; NNFI = 1.0; CFI = 1.0; SRMR = 0.02. Because the two models were not significantly different, $\chi^2(1) = 1.49$, $p = .70$, the more parsimonious model is preferred. That model also showed better fit than an alternative model in which depression mediated the relationship between somatization and pain, $\chi^2(1) = 15.41$, $p = .001$; RMSEA = 0.25; NNFI = 0.75; CFI = 0.92; SRMR = 0.16).

Depression, Coping, and Somatization as Predictors of Abdominal Pain

When coping styles were included as in Model 2 for anxiety, Model 3 (Figure 2) showed a good overall fit, $\chi^2(4) = 10.13$; RMSEA = 0.04; NNFI = 0.99; CFI = 0.99; SRMR = 0.03. Model 3 shows that (a) depression has a significant indirect effect on pain via general somatization (standardized indirect path coefficient = 0.24, $p < .001$), but was not significantly associated with depression when all three coping styles were included as predictors. Thus, accommodative coping appears to be redundant in relationship to active and passive coping.

In contrast, cooperative suppression (Gaylord-Harden et al., 2010) appeared to affect the relationships of active coping and passive coping with depression. In cooperative suppression, the predictive relationship of two (or more) predictors improves when both are included in the analysis. Consistent with cooperative suppression: (a) when active coping is a predictor and neither accommodative nor passive coping is included, the path coefficient for active coping to depression is not significant ($-0.04$). When passive coping is also included, however, the path coefficient for active coping to depression increases substantially, to $-0.28$ ($p < .001$), and the increase is statistically significant (Sobel test statistic $= 4.0$, $p < .001$); (b) when passive coping is a predictor variable and neither active nor accommodative coping is included, the path coefficient for passive coping to depression is $0.53$ ($p < .001$). When active coping is also included, the path coefficient for passive coping to depression increases, albeit slightly, to $0.56$ ($p < .001$). This fits the pattern of cooperative suppression (Gaylord-Harden et al., 2010), in which the shared error variance between active and passive coping is reduced, thereby increasing the strength of their true relationships with the outcome variable. In this situation, suppression is not merely a statistical artifact; rather, it helps to clarify the true relationships between predictors and outcomes by reducing error variance (Gaylord-Harden et al., 2010; Paulhus et al., 2012). Such findings are particularly important when they are replicable, and the present findings extend the replicable suppression effects for coping styles reported previously Gaylord-Harden et al. (2010).

Subsequently, a more parsimonious model for depression was examined that eliminated the nonsignificant paths from Model 3. The parsimonious model (Figure 2, Model 4) showed a good fit overall, $\chi^2(4) = 10.13$; RMSEA = 0.04; NNFI = 0.99; CFI = 0.99; SRMR = 0.03. The $\chi^2$ difference test showed no significant difference between Model 3 and the more parsimonious Model 4, $\Delta\chi^2(6) = 11.63$, $p = .07$, so the more parsimonious model is preferred.

**Supplemental Analysis: Gender Differences**

We also examined whether the values of the path coefficients in the models differed for boys and girls. For anxiety, the full model (Model 2) for boys showed good fit:
SBχ^2(1) = 0.33, p > .56, RMSEA = 0.00, NNFI = 1.0, CFI = 1.00, SRMR = 0.01. For girls, the path model also shows good fit: SBχ^2(1) = 0.21, p > .64, RMSEA = 0.00, NNFI = 1.0, CFI = 1.00, SRMR = 0.01. Multi-sample SEM indicated that the values of the path coefficients in the models including anxiety do not differ for boys and girls, corrected Bryant–Satorra SBχ^2(11) = 4.72, p = .94, and the paths are thus equivalent across genders.

For depression, the parsimonious model for boys showed good fit: SBχ^2(4) = 4.29, p = .37, RMSEA = 0.03 NNFI = 0.99, CFI = 1.00, SRMR = 0.04. The model also showed good fit for girls, SBχ^2(4) = 3.59, p = .46, RMSEA = 0.00, NNFI = 1.0, CFI = 1.00, SRMR = 0.01. Multi-sample SEM indicated that the values of the path coefficients in the models including depression do not differ for boys and girls, corrected Bryant–Satorra SBχ^2(5) = 6.66, p = .25, and the paths are thus equivalent across genders.

**Discussion**

There are several key findings of this study. Models of MUS and results of prior studies have suggested that the child’s tendency to experience physical sensations, that is, to somatize, may mediate the relationship between anxiety and depression with abdominal pain, but this process has not been tested previously. Results of the present study show that, in a diverse community sample of school-age children, the effect of anxiety and depression on subsequent reports of abdominal pain may be mediated by the child’s tendency to somatize.

The second key finding involves the relationships between coping styles and abdominal pain. Findings of prior studies of the coping-pain relationship have yielded inconsistent results, and the directions of the effects for coping styles as predictors of pain have sometimes differed when coping styles were studied simultaneously rather than separately. When compared with those of prior studies, the results of the present study found both similarities and
differences in the association of coping styles with abdominal pain. The bivariate relationships reported previously (Compas et al., 2006; Thomsen et al., 2002; Walker et al., 1997) have shown that the association of accommodative or secondary engagement coping with abdominal pain is inconsistent. The present study found no direct independent relationship of accommodative coping with abdominal pain when active and passive coping styles were also predictor variables.

The literature shows more consistent results for passive or disengagement coping with pain, with passive or disengagement coping associated with increased abdominal pain at low to moderate levels (Compas et al., 2006; Thomsen et al., 2002; Walker et al., 1997, 2005). In addition, passive/disengagement coping is significantly associated with increased anxiety and/or depression in prior reports (Compas et al., 2006; Thomsen et al., 2002; Walker et al., 1997). In models including either anxiety or depression, the present study found no direct relationship between passive coping and pain. For both anxiety and depression, however, an indirect, long chain path was significant, in which increased use of passive coping was associated with more pain via somatization and either anxiety or depression. While prior studies indicate that there is a relationship between passive coping and pain, the present study suggests this relationship may be indirect, via the effects of passive coping on emotions and somatization.

For active coping, results differed for models including anxiety versus depression. While prior work has found a direct relationship between active coping and pain, the present study found that active coping did not show an independent direct relationship with pain in models including somatization, other coping styles, and either anxiety or depression. There were indirect effects, however, with increased use of active coping associated with increased pain via its association with increased somatization. In models including depression, higher levels of active coping were also associated with less pain via the association of more active coping with lower levels of depression and somatization. This long-chain indirect effect was not significant in models including anxiety.

Theoretically, the present models highlight the possible role that individual differences in physical symptom perception may play in developing clinically significant symptoms of abdominal pain. There are individual differences in people’s awareness of physical symptoms just as there are for a variety of other sensory events, and some children may be more sensitive than others to subclinical levels of physical symptoms. Emotional factors may play an important role in increasing the level of somatic symptoms the child experiences to a state in which a particular symptom, such as abdominal pain, emerges as clinically significant, with the association of anxiety or depression with pain mediated by somatization. Not addressed in the present study are the processes leading one symptom such as abdominal pain to emerge from among the other physical symptoms the person may be experiencing and become clinically impairing. Of course, physiological factors may increase the intensity of certain symptoms so that they become more prominent and concerning. In addition, Fordyce (1976) long ago described how individual pain behaviors may each be affected by differences in contingent reinforcement (e.g., a parent responding to crying but, say, not to a child’s statement of being in pain) and differences in learning history might also lead one symptom to become more prominent than others.

The results of this study may have treatment implications. There is empirical support for psychological interventions for FAP, but the effect sizes identified in meta-analysis suggest there is room for improvement. One meta-analyses indicated the effect size for CBT for FAP was moderate (Sprenger, Gerhards, & Goldbeck, 2011), a second study reported a similar effect size (Eccleston et al., 2012), and third systematic review concluded there were too few studies to conduct a meta-analysis (Huertas-Ceballos, Logan, Bennett, & Macarthur, 2009). One limitation to existing studies may concern their treatment of anxiety and depression among children with FAP. As noted above, prior studies indicate that many children with FAP have problems with anxiety and/or depression, but existing treatment programs may not provide enough attention to the treatment of these internalizing problems. Presently, the mean number of sessions in cognitive-behavior therapy (CBT) for pediatric pain for conditions other than headache is 3.5 sessions (Eccleston et al., 2012). Because manualized treatments for anxiety and depression typically involve 12 or more sessions, CBT programs for FAP may need to be lengthened to include interventions for internalizing disorders as well as pain. In addition, the present study suggests that treatments for FAP may also need to attend to other symptoms of somatization rather than focusing on symptoms of pain alone.

It is not uncommon for children with FAP to be referred for treatment to help them “cope” with their pain, but how best to do this has not been clearly determined. Presently, no existing studies have examined the components of the CBT treatment packages. Interventions routinely include training in coping techniques in various ways, but also may include parent training in managing child symptoms. Studies are needed to determine how effective the coping components of the CBT packages are, and how best to maximize their effectiveness. For example,
the results of this study and others suggest that passive/disengagement coping is associated with increased anxiety and depression and with subsequent abdominal pain. Along with attempting to reduce passive coping with pain, attention may also need to be directed to reducing the child’s passive coping approaches to high levels of internalizing disorders. If the child is discouraged from using passive coping techniques, however, then finding more beneficial alternatives is important. Results of the present study suggest that encouraging more active coping techniques to reduce depression may be useful because of the significant indirect path from more active to less pain via depression, but active coping techniques may not be useful to help the anxious child with abdominal pain because the path from active coping to abdominal pain via anxiety was not significant. Furthermore, prior studies have not consistently found a relationship of more use of accommodative coping with less pain, and the results of the present study find no significant independent association of accommodative coping with pain, somatization, or internalizing problems. Because accommodative coping involves the child’s use of emotion-focused strategies to deal with pain, these results are concerning to those of us using cognitive-behavioral interventions for pain management. These results suggest that a great deal of work remains to be done to do identify the best coping approaches to help children deal with FAP.

It is important to note that the present study examined children’s naturally occurring coping styles rather than the use of coping techniques that were taught systematically in psychotherapy. These “spontaneous” or “natural” coping procedures may or may not be less effective than those taught in psychotherapy. Research is needed to determine if coping techniques taught systematically in psychotherapy lead to more effective use of coping techniques than are used by children spontaneously.

The present study has several limitations. First, no information was available on nonparticipating families, so it is not clear how participating and nonparticipating families differ. Second, all psychological measures, including the PRI were gathered at the beginning of the study, while the pain ratings were collected prospectively. Thus, it could not be guaranteed that all the participants had much experience with coping with abdominal pain in months immediately before the study. By limiting the sample to children who reported at least some experience with abdominal pain over the subsequent 24–36 weeks, however, it is likely that most children also had some experience with abdominal pain before completely the baseline measures. Third, discordant results for raters are common in studies of children (Achenbach, McConaughy, & Howell, 1987). The present study relied on child reports only, and additional information from parents would have been useful, but were difficult to collect in the school setting where this study was conducted. Fourth, strength of this study is that pain reports were obtained prospectively; however, reports of internalizing symptoms, coping, and somatization were obtained at the same time. The study would have benefited from measuring those symptoms longitudinally, as well, so that bidirectional influences of pain, somatization, and internalizing symptoms could have been examined. The possibility exists for bias in both cross-sectional and “partially longitudinal designs” such as this one (Maxwell, Cole, & Mitchell, 2011). While fully longitudinal studies of mediation may be preferable, they are also not without bias, as unbiased longitudinal studies of mediation are dependent on including all relevant variables and clearly establishing the causal order of the variables (e.g., do the coping skills precede somatization, or vice versa?) (Shrout, 2011). Unless these assumptions can be met, partially longitudinal designs may provide the best estimate of the concurrent relationships between variables as well as an estimate of possible mediational processes that could be studied in a fully longitudinal design where critical assumptions are met. Fifth, the sample size was not large enough to examine developmental differences in the model, a potentially important issue to be addressed in future research.

Further research will also be needed to be determined if models of coping, somatization, anxiety, depression, and pain differ for community versus clinical samples. It is certainly possible that moderated mediation effects will be identified in which the direct and indirect effects for anxiety/depression, coping, and somatization with pain will be moderated by the child’s pain intensity, frequency, or chronicity. There are, however, some indications that the models examined in this community sample may be similar to those identified in clinical samples because studies conducted with clinical samples often find a similar pattern of correlations between coping and pain reports (Kaczynski et al., 2011; Walker et al., 1997). Studies with clinical samples will also need to address the possible effects of restricted ranges for reports of pain and somatization and their effects on models tested in clinical samples.

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References


