Posttraumatic Stress Symptoms and the Diathesis-Stress Model of Chronic Pain and Disability in Patients Undergoing Major Surgery

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Objectives: To (1) use structural equation modeling (SEM) to examine relationships proposed in Turk's diathesis-stress model of chronic pain and disability as well as (2) investigate what role, if any, posttraumatic stress symptoms (PTSS) play in predicting pain disability, relative to some of the other factors in the model.

Methods: The study sample consisted of 208 patients scheduled for general surgery, 21 to 60 years of age (mean age = 47.18 y, SD = 9.72 y), who reported experiencing persistent pain for an average of 5.56 years (SD = 7.90 y). At their preadmission hospital visit, patients completed the Anxiety Sensitivity Index, Pain Catastrophizing Scale, Pain Anxiety Symptoms Scale-20, Pain Disability Index, posttraumatic stress disorder Checklist, and rated the average intensity of their pain (0 to 10 numeric rating scale). SEM was used to test a model of chronic pain disability and to explore potential relationships between PTSS and factors in the diathesis-stress model.

Results: SEM results provided support for a model in which anxiety sensitivity predicted fear of pain and catastrophizing, fear of pain predicted escape/avoidance, and escape/avoidance predicted pain disability. Results also provided support for a feedback loop between disability and fear of pain. SEM analyses provided preliminary support for the inclusion of PTSS in the diathesis-stress model, with PTSS accounting for a significant proportion of the variance in pain disability.

Discussion: Results provide empirical support for aspects of Turk's diathesis-stress model in a sample of patients with persistent pain. Findings also offer preliminary support for the role of PTSS in fear-avoidance models of chronic pain.

Key Words: diathesis-stress, fear-avoidance, PTSS, chronic pain

The role of psychologic and behavioral factors in the development and maintenance of chronic pain and disability has received increased attention in recent years. In particular, anxiety sensitivity, fear of pain, catastrophizing, and escape/avoidance behaviors are theorized to play a

critical role in the maintenance of many chronic pain conditions.[1,2] In an effort to integrate these various factors and offer an explanation for individual differences in recovery after a trauma, Turk[3] proposed a diathesis-stress model of chronic pain and disability (Fig. 1). The model posits that individuals are more likely to develop avoidance responses and subsequent pain disability after a trauma if they are highly anxiety-sensitive, and thus experience increased body awareness and a fear of physiologic reactions; if they engage in catastrophic thinking involving interpreting pain sensations, and associated symptoms, as indicative of serious harm; and if they are predisposed to respond fearfully to pain.

Converging lines of evidence provide empirical support for various components of the diathesis-stress model.[3] Clinical data indicate a link between anxiety sensitivity and chronic pain. Anxiety sensitivity is the fear of anxiety-related sensations, such as a rapidly beating heart, based on the belief that these sensations will have harmful consequences.[4] Highly anxiety-sensitive individuals experience increased body awareness and a fear of physiologic reactions, which in turn may make them more vigilant to pain-related sensations and more fearful of the noxious sensations associated with pain.[5] High anxiety sensitivity is associated with more fearful appraisals of pain and greater pain-related escape/avoidance behaviors in individuals with chronic back pain,[6] musculoskeletal pain,[7] and recurrent headaches.[8] Anxiety sensitivity has also been shown to predict fear of pain better than depression and pain severity in a heterogenous chronic pain population.[9] Furthermore, in a sample of children and adolescents with chronic pain, anxiety sensitivity accounted for 38.6% of the variance in fear of pain and fear of pain accounted for 39.9% of the variance in pain-related disability.[10]

In addition to anxiety sensitivity, a number of studies have linked fear of pain to increased functional impairment and decreased levels of physical performance.[11–14] Fear and avoidance behaviors have been linked to distress and disability in patients with headache, sickle cell disease, and musculoskeletal pain.[1] Furthermore, fear of pain has been shown to predict disability and interference with activities of daily living better than anxiety, depression, and sensory pain.[15]
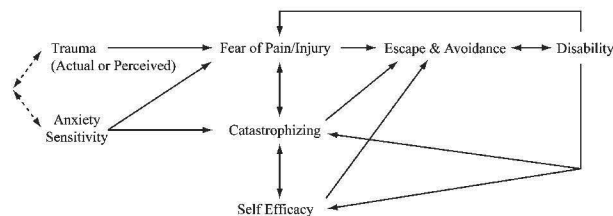
Research and clinical data also provide support for the role of catastrophizing in chronic pain.[16] Similar to anxiety sensitivity, catastrophizing is thought to play a role in fearful and avoidant responses to pain through hypervigilance to threatening somatic cues and excessive focus on pain sensations.[17] Pain catastrophizing is characterized by "...an exaggerated negative 'mental set' brought to bear during actual or anticipated pain experience" (pp. 53 in Ref. 18), unrealistic beliefs that the current situation will

FIGURE 1. Turk's diathesis-stress model of chronic pain and disability.[3] Reproduced with permission.

lead to the worst possible pain outcome,[19] and negative thoughts about the future and self.[3]

Research demonstrates an association between catastrophizing and low back pain,[20] mixed chronic pain,[21] pain related to dental procedures,[22] and acute postsurgical pain.[23–25] Furthermore, Sullivan and colleagues[17] found catastrophizing assessed 10 weeks before a painful procedure was predictive of subsequent pain ratings. Pain catastrophizing also predicts preoperative and postoperative pain ratings in patients undergoing total knee arthroplasty.[26–28]

Several fear-avoidance models have been proposed to integrate these various findings. On the basis of earlier studies,[29–31] Vlaeyen and colleagues[13] proposed a fear-avoidance model of chronic pain in which catastrophic misinterpretations of pain set in motion a cycle of pain-related fear, escape/avoidance, and hypervigilance, leading to increased disability and impairment.[2] Cook et al[32] recently validated this model through the use of structural equation modeling (SEM). Asmundson and Taylor[7] proposed a model in which anxiety sensitivity directly exacerbates fear of pain and indirectly increases pain-related escape/avoidance behaviors through its influence on fear of pain. They found that anxiety sensitivity accounted for 30% of the variance in fear of pain and fear of pain accounted for 68% of the variance in escape/avoidance behavior in adults with musculoskeletal injury. Norton and Asmundson[33] subsequently replicated this model in a sample of patients with recurrent headache pain.

Turk's[3] diathesis-stress model expands on Asmundson and Taylor's[7] model by including the influence of pain catastrophizing and self-efficacy. To date, the relationships among various components of the diathesis-stress model[3] have been independently supported. However, additional research is required to simultaneously test the various links proposed in the diathesis-stress model to validate the model as a whole. Research is also needed to investigate the role of other factors that may broaden the diathesis-stress model. Specifically, the relationship between posttraumatic stress disorder (PTSD) and chronic pain is of growing interest. Research and clinical practice indicate these 2 disorders are highly comorbid.[34–36]

Two hypotheses, the shared vulnerability and the mutual maintenance hypotheses, have been proposed to account for the link between PTSD and chronic pain. The shared vulnerability hypothesis suggests that certain factors, namely anxiety sensitivity, may predispose individuals to the development of one or both of these conditions.[34] Conversely, the mutual maintenance hypothesis suggests that certain components of each disorder may interact in such a way as to maintain or exacerbate the other.[35] For example, for individuals with PTSD, chronic pain may serve as an ongoing reminder of the traumatic event, which in turn may lead to increased avoidance of pain sensations and ultimately increased levels of distress and disability.[35] Given these observed links between PTSD and chronic pain, and the inclusion of trauma (perceived or actual) as a variable in the diathesis-stress model,[3] the potential role of posttraumatic stress symptoms (PTSS) is of great interest.

This study had 2 aims: (1) to use SEM to test the relationships proposed in the diathesis-stress model of chronic pain and disability,[3] and (2) to investigate the role, if any, played by PTSS in predicting pain disability, relative to other factors in the model. To address these aims, 2 statistical models were created and evaluated in 208 patients with persistent pain who were scheduled for major surgery. The first tested a Turk-based model of pain disability[3]; and the second explored potential relationships between PTSS and the variables in the first model.

MATERIALS AND METHODS

Participants

The study sample consisted of 208 patients scheduled to undergo major surgery [abdominal (68.72%), thoracic (17.95%), and other (13.33%); 124 females and 83 males; 21 to 60 years of age (mean age 47.18 y, SD 9.72 y)], who were experiencing persistent pain. Pain locations are presented in Table 1. Fifty-one percent of patients reported pain at more than one site. Of the 208 patients, 21 were unable to identify when their pain began. Four patients reported their pain began within the last year but were unable to identify the month of onset. The remaining 183 patients reported having experienced a persistent pain problem for an average of 5.56 years (SD = 7.90 y) and reported an average pain intensity of 5.70 (SD = 2.45) on a 0 to 10 numeric rating scale (NRS).

The present sample was selected from a study database examining predictors of postoperative pain and the transition from acute to persistent pain after surgery. An earlier publication from this database examined the factor structure of PTSS.[37] Patients were screened for preexisting pain conditions before their surgery and the present sample represents those individuals who reported a persistent pain problem. The nature of the pain was based on patient self-report. Information regarding formal diagnoses and whether their surgery was related to their persistent pain condition is unknown.

Measures and Procedure

The research study was reviewed and approved by the Research Ethics Board at the Toronto General Hospital,

TABLE 1. Pain Locations Reported by Patients at Their Preadmission Hospital Visit

Location of Pain	N	%
Abdomen	60	28.85
Back	37	17.79
Lower limb	31	14.90
Thorax	26	12.50
Pelvis	20	9.62
Head/neck	17	8.17
Upper limb	9	4.33
Whole body	5	2.40
Missing	3	1.44
Total	208	100

University Health Network, and by the Human Partici-
pants Review Committee at York University. Prospective
patients were recruited at the preadmission hospital visit
approximately 7 to 10 days before surgery. After informed
written consent, patients were asked to rate the intensity of
their current pain on a 0 to 10 NRS and to complete the
following questionnaires: the Anxiety Sensitivity Index
(ASI),[38] the Pain Catastrophizing Scale (PCS),[17] the Pain
Anxiety Symptoms Scale (PASS),[39] the Pain Disability
Index (PDI),[40] and the PTSD Checklist—Civilian Version
(PCL-C).[41]

ASI

The ASI[38] is a widely used, 16-item scale that measures
concerns that anxiety and anxiety-related symptoms will
lead to harmful negative consequences. Each item is rated
on a 5-point scale ranging from very little (0) to very much
(4). The ASI yields a total score and 3 factor analytic
derived subscale scores, including (1) fear of somatic
symptoms/physical concerns, (2) fear of cognitive symp-
toms/mental incapacitation concerns, and (3) fear of
publicly observable symptoms/social concerns.[42] The ASI
demonstrates good test-retest reliability ($r = 0.72$) and
research demonstrates some evidence for the discriminant
validity of the 3 subscales among anxiety disorder out-
patients.[43]

PCS

The PCS[17] consists of 13 items describing thoughts
and feelings that individuals may experience when they are
in pain. Each item is rated on a 5-point scale ranging from
not at all (0) to all the time (4). The PCS yields a total score
and 3 subscale scores assessing (1) rumination, (2)
magnification, and (3) helplessness. The PCS demonstrates
adequate-to-excellent internal consistency in community
($\alpha = 0.88$ to 0.95) and pain outpatient samples ($\alpha = 0.75$ to
0.92).[44] Concurrent validity is also evidenced by a moderate
correlation between the PCS and self-report measures of
anxiety ($r = 0.32$) and negative affectivity ($r = 0.70$).[45] The
PCS shows good test-retest reliability at 6 ($r = 0.75$) and 10
weeks ($r = 0.70$).[45]

PASS-20

The PASS-20[39] is a shortened 20-item version of the
original PASS[15] designed to measure fear and anxiety
responses specific to pain. The PASS-20 has four 5-item
subscales, including (1) cognitive anxiety, (2) escape and
avoidance, (3) fearful thinking, and (4) physiologic anxiety.
Each item is rated on a 6-point scale ranging from never (0)
to always (5), with total scores ranging from 0 to 100. The
PASS-20 has been shown to have good internal consistency
($\alpha = 0.81$), and good convergent validity with the original
40-item PASS ($r = 0.95$).[39] Concurrent validity of the
PASS-20 is demonstrated through its moderate-to-high
correlations with related measures such as ASI ($r = 0.56$),
fear of pain (fear of pain questionnaire-III; $r = 0.53$), and
PCS ($r = 0.38$).[46]

PDI

The PDI[40] assesses the extent to which persistent pain
interferes with an individual's ability to engage in 7
different areas of everyday activity including: (1) family/
home responsibilities, (2) recreation, (3) social activity, (4)

occupation, (5) sexual behavior, (6) self-care, and (7) life-
support activity. Each item is rated on an 11-point scale
ranging from no disability (0) to total disability (10). The
PDI demonstrates good construct validity as evidenced by
its significant relationship with other measures of pain-
related disability and distress.[47,48] Test-retest reliability of
the PDI is good ($r = 0.91$)[49] and the internal consistency is
high ($\alpha = 0.86$).[50]

PCL-C

The PCL-C[41] is a 17-item self-report measure. Each
item is a statement based on the current Diagnostic and
Statistical Manual of Mental Disorder-IV symptoms for
PTSD. Respondents are asked to indicate how much they
have been bothered by each symptom over the past month
on a 5-point scale ranging from not at all (1) to extremely
(5). The questionnaire produces a total score and 4 subscale
scores,[51] including (1) re-experiencing, (2) avoidance, (3)
numbing, and (4) hyperarousal. Test-retest reliability for
the PCL-C over a 2 to 3 day retest interval is 0.96.[52] Cut-off
scores of 44[52] and 50[53] have been shown to reliably predict
PTSD diagnosis.

NRS for Pain Intensity

Pain intensity was measured using a self-report NRS[54]
ranging from 0 to 10, with endpoints representing no pain
(0) and the most intense pain imaginable (10). Patients were
asked to choose the number that best corresponds to the
average intensity of their pain. The NRS provides a simple,
efficient, and minimally intrusive measure of pain intensity.
This scale is commonly used in clinical settings[55] and is the
preferred pain rating scale among patients.[56] The NRS is
highly correlated ($r = 0.94$) with the Visual Analog Scale[55]
and is sensitive to change following pharmacologic inter-
ventions.[54]

Data Analysis

The first objective of this study was to use SEM to
allow a comprehensive investigation of the relationships
proposed in the diathesis-stress model of chronic pain and
disability.[3] The hypothesized model (Fig. 2) predicts a
direct effect of anxiety sensitivity on fear of pain (path 1),
anxiety sensitivity on catastrophizing (path 2), fear of pain
on escape/avoidance behaviors (path 3), catastrophizing on
escape/avoidance behaviors (path 4), and escape/avoidance
behaviors on pain disability (path 5). Furthermore, this
model predicts that pain intensity will have a direct effect
on fear of pain (path 6), catastrophizing (path 7), escape/
avoidance (path 8), and disability (path 9). Pain intensity
was included in the model to make explicit the contribution
of pain to disability and because it is the main factor
prompting patients to seek help. Two of the feedback loops
proposed in the model[3] were also tested; these include a
direct effect of disability on fear of pain (path 10) and
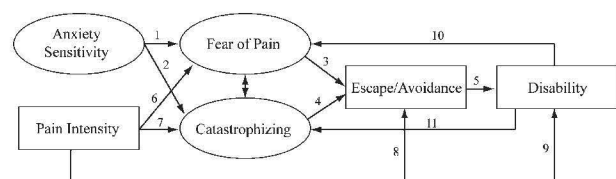
FIGURE 2. Path diagram of hypothesized relationships in Turk
diathesis-stress model of chronic pain and disability.[3]

disability on catastrophizing (path 11). Self-efficacy was not evaluated in this adaptation of the diathesis-stress model.[3]

In the model in Figure 2 and in subsequent figures, anxiety sensitivity, fear of pain, and catastrophizing are depicted as latent variables (using ovals) rather than observed variables (using rectangles) because multiple indicators, namely the subscale scores from each measure, were available for these constructs. Using latent variables for constructs leads to path estimates that are unbiased by measurement error. This strategy was based on results of Coffman and MacCallum,[57] who found that fitting models using item parcels (ie, the subscale scores) as latent variable indicators is superior to using path analyses with observed total score indicators adjusted by estimated reliability.

The second objective of this study was to investigate what role, if any, PTSS play in predicting pain disability, relative to other factors in the diathesis-stress model.[3] Although this study did not use a psychiatric population, community-based studies indicate that the lifetime prevalence for experiencing a traumatic event is 89.6%[58] and the lifetime prevalence for PTSD is approximately 8%.[59] Furthermore, many more individuals not meeting diagnostic criteria for a full diagnosis of PTSD may experience partial or sub-threshold PTSS.[60] We, therefore, anticipated a range of severity in PTSS within the present sample and found 22.7% of PCL-C scores fell within the clinical range (Fig. 3).

To address the second objective of this study, a second model (Fig. 4) was developed based on the shared vulnerability and mutual maintenance hypotheses linking PTSD and chronic pain. As described earlier, the shared vulnerability hypothesis proposes that anxiety sensitivity may predispose individuals to the development of both PTSD and chronic pain.[34] As such, a direct path from anxiety sensitivity to PTSS was hypothesized. Furthermore, the mutual maintenance hypothesis suggests that certain components of chronic pain may maintain symptoms of PTSD and vice versa.[35] Accordingly, pain intensity was hypothesized to have a direct relationship with PTSS and PTSS were hypothesized to have a direct effect on escape/avoidance behavior. Bidirectional relationships among fear of pain, catastrophizing, and PTSS were also proposed.
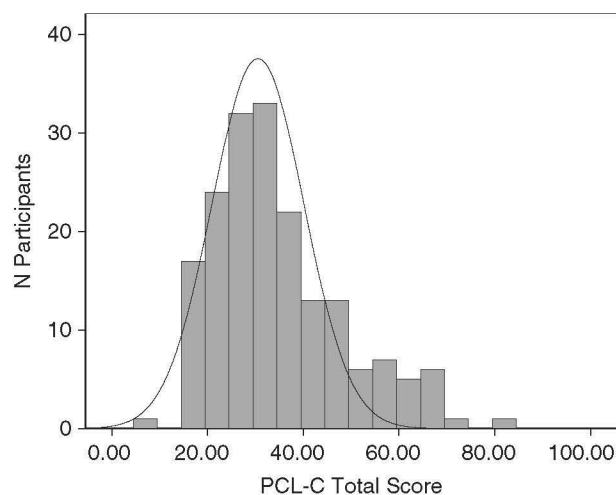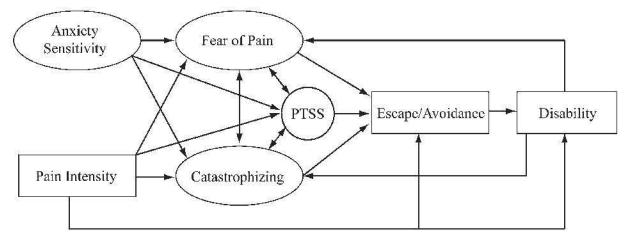
FIGURE 4. Path diagram of hypothesized relationships in a modified diathesis-stress model[3] that includes posttraumatic stress symptoms.

Before estimating the models, the manifest variables were examined for univariate and multivariate outliers and distribution. Table 2 presents descriptive statistics for each of the manifest variables. SEM was performed according to a 2-stage process using AMOS 7.0 (SPSS Inc, Chicago, IL). First, separate confirmatory factor analysis models were used to verify that the subscale scores used to measure each of the latent variables formed a single factor. To include cases with missing values, a one-factor model was fitted to the observed covariance matrix using full information maximum likelihood estimation[61] and fit statistics were examined to confirm that a one-factor model was a good fit to the data for a given set of subscales.

After verifying the factor structure of each of the latent variables, the hypothesized models in Figures 2 and 3 were estimated, again using full information maximum likelihood. In addition to χ^2 goodness-of-fit tests, 5 fit statistics were computed for each model: root-mean-square error of approximation (RMSEA), comparative fit index (CFI), Tucker-Lewis index (TLI), Akaike information criterion (AIC), and Browne-Cudeck criterion (BCC). The RMSEA reflects the magnitude of difference between the fitted and actual covariance matrices with a parsimony correction for number of parameters. Values of RMSEA ≤ 0.05 indicate close fit and RMSEA $0.05 \leq 0.08$ suggests a reasonable model fit.[62] The CFI assesses the relative improvement in fit of the proposed model compared with a null model with no specified relations.[62] Values of CFI ≥ 0.90 indicate reasonable model fit. The TLI assesses the relative improvement per degree of freedom of the target model over the null model.[63] Similar to the CFI, values of TLI ≥ 0.90 indicate reasonable model fit. Finally, the AIC assesses model fit in hypothetical replication samples of the same size and randomly drawn from the same population as the research sample.[62] The AIC is generally used to select among non-nested competing models estimated with the same data. The BCC[64] operates in the same manner as the AIC but imposes greater penalties.[65] The model with the smallest AIC and BCC is considered to have the best fit.

RESULTS

Evaluation of a Diathesis-Stress Model of Chronic Pain Disability

Means and SDs for each of the measures used in the analyses are presented in Table 2 and correlation coefficients among model variables are presented in Table 3. On the basis of the proposed diathesis-stress model,[3] a model with 3 manifest and 3 latent variables was developed (Model 1, Fig. 5). As illustrated in Figure 5, the 3 subscales of the ASI: ASI—fear of somatic symptoms/physical

FIGURE 3. Distribution of posttraumatic stress disorder Checklist—Civilian Version scores across participants.

TABLE 2. Descriptive Statistics of Measures Used in Structural Equation Modeling (N=208)

Measure	Mean	SD	α	% Missing
Pain intensity	5.70	2.45	—	6.70
ASI				
Fear of somatic symptoms	11.61	7.32	0.89	12.00
Fear of cognitive symptoms/mental incapacitation	2.62	3.18	0.86	12.50
Fear of publicly observable symptoms/social concerns	5.52	2.37	0.50	12.00
PCS				
Rumination	7.68	4.55	0.91	11.50
Magnification	3.81	2.74	0.73	11.50
Helplessness	7.84	5.78	0.91	11.10
PASS-20				
Physiologic anxiety	7.71	6.35	0.84	12.50
Cognitive anxiety	12.57	6.25	0.83	12.50
Fearful thinking	8.77	6.73	0.82	12.50
Escape and avoidance	11.85	6.12	0.77	12.50
PCL-C				
Re-experiencing	9.45	4.60	0.89	13.00
Avoidance	3.89	2.02	0.73	13.50
Emotional numbing	10.17	4.48	0.83	13.50
Hyperarousal	11.38	4.49	0.81	13.50
PDI	27.19	18.87	—	11.50

ASI indicates Anxiety Sensitivity Index; PASS-20, Pain Anxiety Symptoms Scale-20; PCL-C, Posttraumatic Stress Disorder Checklist—Civilian Version; PCS, Pain Catastrophizing Scale; PDI, Pain Disability Index.

concern, ASI—cognitive symptoms/mental incapacitation concerns, and ASI—publicly observable symptoms/social concerns, formed an anxiety sensitivity latent variable; the 3 subscales of the PASS-20: PASS—cognitive anxiety, PASS—fearful thinking, and PASS—physiologic anxiety, formed a fear of pain latent variable; and finally the 3 subscales of the PCS: PCS—rumination, PCS—magnification, and PCS—helplessness, formed a pain catastrophizing latent variable. Pain intensity, escape/avoidance, and pain disability were estimated by the NRS, PASS-Escape/Avoidance subscale, and PDI total score, respectively.

Model fit statistics indicated adequate fit of the hypothesized model to the data (RMSEA = 0.07; CFI = 0.97; and TLI = 0.94). The χ^2 statistic was significant [χ^2 (45, N = 208) = 87.15, $P < 0.05$]; however, this statistic is generally not regarded as an ideal fit statistic as it is highly sensitive to sample size and the size of correlations[62] and tests the unrealistic null hypothesis of perfect fit.[66] Therefore, results provide support for the hypothesized model. As expected, each manifest variable had a salient loading on its specified latent variable. All of the standardized path coefficients shown in Figure 5 were significant with the

TABLE 3. Zero-order Correlations Among Variables in Structural Model

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Pain	1	0.08	0.10	0.06	0.28**	0.18*	0.18*	0.20*	0.20**	0.15*	0.24**	0.25**	0.25**	0.18*	0.21**	0.29**
2. ASI-SOM	—	1	0.68**	0.43**	0.49**	0.59**	0.54**	0.54**	0.61**	0.68**	0.44**	0.32**	0.32**	0.38**	0.44**	0.22**
3. ASI-COG	—	—	1	0.47**	0.38**	0.56**	0.55**	0.48**	0.49**	0.60**	0.29**	0.34**	0.31**	0.33*	0.40**	0.25**
4. ASI-PUB	—	—	—	1	0.34**	0.43**	0.39**	0.32**	0.34**	0.36**	0.22**	0.18*	0.21**	0.25**	0.25**	0.17*
5. PCS-RUM	—	—	—	—	1	0.70**	0.71**	0.54**	0.62**	0.60**	0.54**	0.39**	0.40**	0.40**	0.37**	0.32**
6. PCS-MAG	—	—	—	—	—	1	0.73**	0.64**	0.56**	0.71**	0.49**	0.43**	0.40**	0.43**	0.36**	0.31**
7. PCS-HELP	—	—	—	—	—	—	1	0.59**	0.58**	0.63**	0.48**	0.33**	0.34**	0.35**	0.41**	0.33**
8. PASS-PA	—	—	—	—	—	—	—	1	0.63**	0.76**	0.61**	0.43**	0.46**	0.35**	0.36**	0.41**
9. PASS-CA	—	—	—	—	—	—	—	—	1	0.70**	0.62**	0.41**	0.43**	0.41**	0.44**	0.43**
10. PASS-FT	—	—	—	—	—	—	—	—	—	1	0.59**	0.40**	0.46**	0.41**	0.39**	0.40**
11. PASS-E/A	—	—	—	—	—	—	—	—	—	—	1	0.28**	0.34**	0.27**	0.25**	0.47**
12. PCL-RE-EXP	—	—	—	—	—	—	—	—	—	—	—	1	0.70**	0.62**	0.58**	0.29**
13. PCL-AVOID	—	—	—	—	—	—	—	—	—	—	—	—	1	0.69**	0.54**	0.35**
14. PCL-NUMB	—	—	—	—	—	—	—	—	—	—	—	—	—	1	0.70**	0.46**
15. PCL-AROUS	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	0.39**
16. PDI	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1

*$P < 0.05$.
**$P < 0.01$.

ASI indicates Anxiety Sensitivity Index; ASI-COG, ASI—fear of cognitive symptoms/mental incapacitation concerns; ASI-PUB, ASI—fear of publicly observable symptoms/social concerns; ASI-SOM, ASI—somatic symptoms/physical concerns; PASS, Pain Anxiety Symptoms Scale; PASS-CA, PASS—cognitive anxiety; PASS-E/A, PASS—escape/avoidance; PASS-FT, PASS—fearful thinking; PASS-PA, PASS—physiological anxiety; PCL, PTSD Checklist; PCL-AROUS, PCL—hyperarousal; PCL-AVOID, PCL—avoidance; PCL-NUMB, PCL—numbing; PCL-RE-EXP, PCL—re-experiencing; PCS, Pain Catastrophizing Scale; PCS-HELP, PCS—helplessness; PCS-MAG, PCS—magnification; PCS-RUM, PCS—rumination; PDI, Pain Disability Index total score.
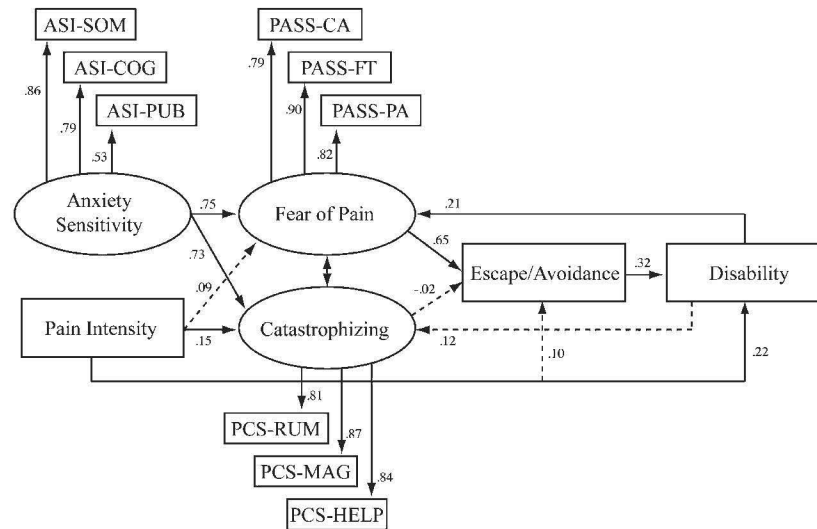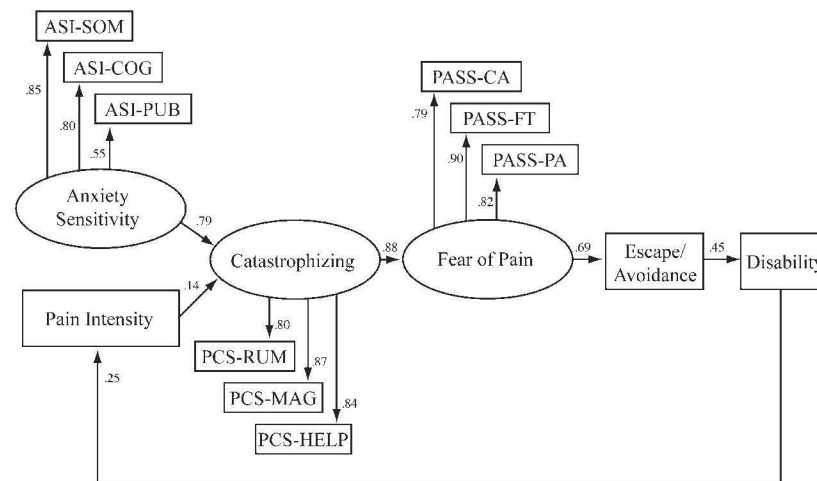
FIGURE 5. Structural equation Model 1, based on the diathesis-stress model of chronic pain and disability.[3] Standardized path coefficients are presented. ASI indicates Anxiety Sensitivity Index; ASI-COG, ASI—fear of cognitive symptoms/mental incapacitation concerns; ASI-PUB, ASI—fear of publicly observable symptoms/social concerns; ASI-SOM, ASI—somatic symptoms/physical concerns; Disability, Pain Disability Index total score; Escape/Avoidance, PASS-20—escape/avoidance; PASS, Pain Anxiety Symptoms Scale; PASS-CA, PASS-20—cognitive anxiety; PASS-FT, PASS-20—fearful thinking; PASS-PA, PASS-20—physiologic anxiety; PCS, Pain Catastrophizing Scale; PCS-HELP, PCS—helplessness; PCS-MAG, PCS—magnification; PCS-RUM, PCS—rumination. Dotted lines represent nonsignificant paths ($P > 0.05$).

exception of the paths represented by dotted lines. Specifically, the paths from pain intensity to fear of pain ($P = 0.15$), pain intensity to escape/avoidance ($P = 0.09$), catastrophizing to escape/avoidance ($P = 0.89$), and pain disability to catastrophizing ($P = 0.08$) were not significant.

Interpretation of SEM analyses is strengthened by comparison with alternative models.[67] Thus, a modified model was tested in which pain intensity and anxiety sensitivity were hypothesized to influence pain catastrophizing directly, which in turn was hypothesized to have a direct influence on fear of pain (Model 2, Fig. 6). Furthermore, a feedback loop from disability to pain

intensity was also proposed. These hypothesized alternate relationships were based on the Vlaeyen-Linton fear-avoidance model[2] and subsequent modifications to this model by Norton and Asmundson,[68] who proposed a direct effect of anxiety sensitivity on pain catastrophizing. Although the χ^2 test was significant [χ^2 (51, $N = 208$) = 103.64, $P < 0.05$], other model fit indices indicate an adequate fit of Model 2 to the data (RMSEA = 0.07; CFI = 0.96; and TLI = 0.93). Furthermore, as illustrated in Figure 6, all standardized path coefficients were significant. The AIC and BCC values for Model 1 (AIC = 177.15 and BCC = 183.18) were smaller than those for Model 2

FIGURE 6. Structural equation Model 2. Standardized path coefficients are presented. ASI indicates Anxiety Sensitivity Index; ASI-COG, ASI—fear of cognitive symptoms/mental incapacitation concerns; ASI-PUB, ASI—fear of publicly observable symptoms/social concerns; ASI-SOM, ASI—somatic symptoms/physical concerns; Disability, Pain Disability Index total score; Escape/Avoidance, PASS-20—escape/avoidance; PASS, Pain Anxiety Symptoms Scale; PASS-CA, PASS-20—cognitive anxiety; PASS-FT, PASS-20—fearful thinking; PASS-PA, PASS-20—physiological anxiety; PCS, Pain Catastrophizing Scale; PCS-HELP, PCS—helplessness; PCS-MAG, PCS—magnification; PCS-RUM, PCS—rumination. Dotted lines represent nonsignificant paths ($P > 0.05$).

(AIC = 181.64 and BCC = 186.86), suggesting the Turk-based Model 1 offers a closer fit to the data.

Role of PTSS in Predicting Pain Disability

A second set of SEM analyses were computed to investigate what role, if any, PTSS play in predicting pain disability in conjunction with the other factors in the diathesis-stress model.[3] Specifically, the hypothesized structural model predicts a direct effect of anxiety sensitivity and pain intensity on PTSS and a direct effect of PTSS on escape/avoidance behaviors (Model 3, Fig. 7). Bidirectional relationships between PTSS, fear of pain, and catastrophizing were also proposed. Although the χ^2 test was significant [χ^2 (90, $N = 208$) = 185.00, $P < 0.05$], other model fit indices indicate close fit of the model to the data (RMSEA = 0.07; CFI = 0.94; and TLI = 0.91). The standardized path coefficients shown in Figure 8 were significant with the exception of the paths represented by dotted lines. Specifically, the paths from pain intensity to fear of pain ($P = 0.07$), pain intensity to escape/avoidance ($P = 0.06$), catastrophizing to escape/avoidance ($P = 0.98$), PTSD symptoms to escape/avoidance ($P = 0.23$), disability to fear of pain ($P = 0.12$), and disability to catastrophizing ($P = 0.65$) were not significant.

An alternate competing model was also tested based on recent findings that PTSS and pain intensity independently predict pain disability[69] and the possibility that high levels of fear, avoidance, and catastrophizing may be indicative of a traumatic stress reaction to pain (involving symptoms of intrusive thoughts, emotional numbing, behavioral avoidance, and hyperarousal), which in turn may relate to increased pain disability. As such, an alternate model was proposed in which fear of pain, catastrophizing, and avoidance behaviors were hypothesized to directly influence PTSS, which in turn were hypothesized to directly influence pain disability (Model 4, Fig. 8). Goodness-of-fit indices indicate close fit of Model 4 to the data (RMSEA = 0.07; CFI = 0.95; and TLI = 0.92), again with the exception of the χ^2 statistic, [χ^2 (92, $N = 208$) = 181.68, $P < 0.05$]. Standardized path coefficients for this model are shown in Figure 8. All paths were significant with the exception of the paths from escape/avoidance to PTSS ($P = 0.18$) and catastrophizing to PTSS ($P = 0.41$). The AIC and BCC values for Model 4 (AIC = 301.68 and BCC = 312.42) were lower than those for Model 3 (AIC = 309 and BCC = 320.10), suggesting Model 4 offers a better fit to these data.

DISCUSSION

The results of this study provide preliminary support for the diathesis-stress model of chronic pain proposed by Turk.[3] The first model we examined (Model 1, Fig. 5), identified anxiety sensitivity as having significant direct effects on fear of pain and catastrophizing, accounting for 75% and 73% of the variance, respectively. Furthermore, fear of pain had a direct effect on escape/avoidance, accounting for 65% of the variance, and escape/avoidance had a direct effect on pain disability, accounting for 32% of the variance. Preliminary evidence for a potential feedback loop proposed in the diathesis-stress model of chronic pain[3] was also evident as pain disability had a direct effect on fear of pain, accounting for 21% of the variance.

However, pain catastrophizing did not have a significant effect on escape/avoidance and disability did not
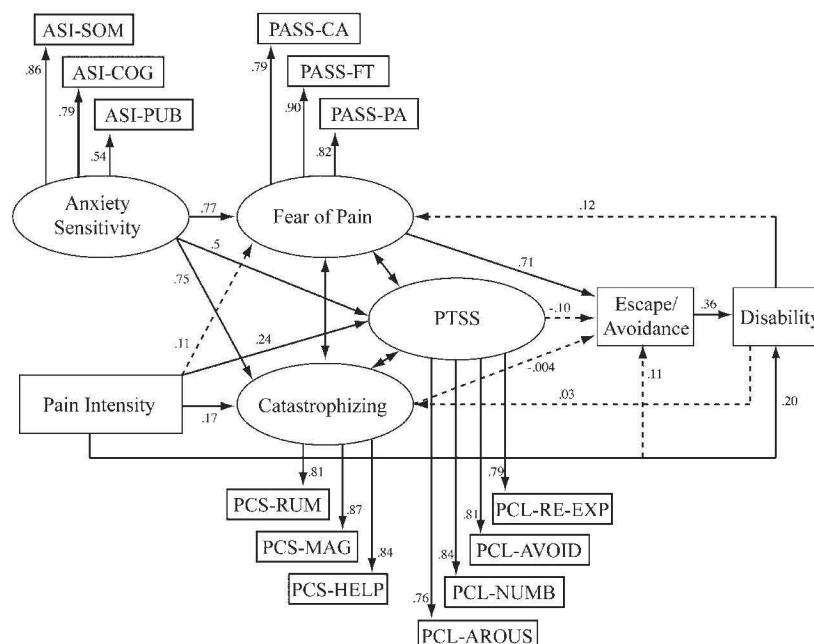
FIGURE 7. Structural equation Model 3, based on a modified diathesis-stress model[3] incorporating posttraumatic stress symptoms. Standardized path coefficients are presented. ASI indicates Anxiety Sensitivity Index; ASI-COG, ASI—fear of cognitive symptoms/mental incapacitation concerns; ASI-PUB, ASI—fear of publicly observable symptoms/social concerns; ASI-SOM, ASI—somatic symptoms/physical concerns; Disability, Pain Disability Index total score; Escape/Avoidance, PASS-20—escape/avoidance; PASS, Pain Anxiety Symptoms Scale; PASS-CA, PASS-20—cognitive anxiety; PASS-FT, PASS-20—fearful thinking; PASS-PA, PASS-20—physiological anxiety; PCL, PTSD Checklist; PCL-AROUS, PCL—hyperarousal; PCL-AVOID, PCL—avoidance; PCL-NUMB, PCL—numbing; PCL-RE-EXP, PCL—re-experiencing; PCS, Pain Catastrophizing Scale; PCS-HELP, PCS—helplessness; PCS-MAG, PCS—magnification; PCS-RUM, PCS—rumination; PTSS, posttraumatic stress symptoms. Dotted lines represent nonsignificant paths ($P > 0.05$).
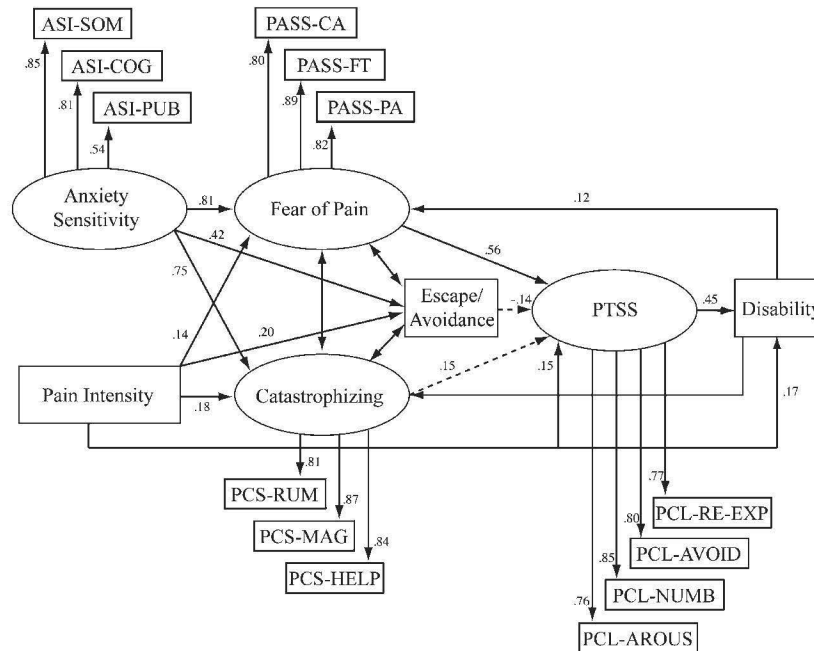
FIGURE 8. Structural equation Model 4. Standardized path coefficients are presented. ASI indicates Anxiety Sensitivity Index; ASI-COG, ASI—fear of cognitive symptoms/mental incapacitation concerns; ASI-PUB, ASI—fear of publicly observable symptoms/social concerns; ASI-SOM, ASI—somatic symptoms/physical concerns; Disability, Pain Disability Index total score; Escape/Avoidance, PASS-20—escape/avoidance; PASS, Pain Anxiety Symptoms Scale; PASS-CA, PASS-20—cognitive anxiety; PASS-FT, PASS-20—fearful thinking; PASS-PA, PASS-20—physiological anxiety; PCL, PTSD Checklist; PCL-AROUS, PCL—hyperarousal; PCL-AVOID, PCL—avoidance; PCL-NUMB, PCL—numbing; PCL-RE-EXP, PCL—re-experiencing; PCS, Pain Catastrophizing Scale; PCS-HELP, PCS—helplessness; PCS-MAG, PCS—magnification; PCS-RUM, PCS—rumination; PTSD, posttraumatic stress disorder. Dotted lines represent nonsignificant paths ($P > 0.05$).

have a significant direct effect on pain catastrophizing. Although the hypothesized direct effect of catastrophizing on escape/avoidance was not significant, it is still possible that catastrophizing had an indirect effect on escape/avoidance through its relationship with fear of pain. Further research is needed to determine the extent to which pain catastrophizing plays a causal role in pain disability as predicted by the diathesis-stress model.[13]

The results also indicate that pain intensity had a significant direct effect on pain catastrophizing and pain disability. However, contrary to expectation, pain intensity did not have a significant direct effect on fear of pain or escape/avoidance behaviors. Earlier research also indicated that the direct effect of pain severity on escape/avoidance is small among patients with recurrent headache pain.[33]

This study also sought to examine the potential role of PTSS in predicting pain-disability in relation to some of the other factors in the diathesis-stress model.[3] Results provided preliminary support for a model in which anxiety sensitivity and pain intensity had a direct effect on fear of pain, fear of pain and pain intensity had direct effects on PTSS, and PTSS and pain intensity had direct effects on pain disability (Model 4, Fig. 8). Pain intensity accounted for 15% of the variance in PTSS, providing further support for a relationship between PTSS and persistent pain. Pain intensity also had a direct effect on pain disability, accounting for 17% of the variance; however, PTSS had an even larger effect on pain disability, accounting for 45% of the variance.

Model 4 allowed for correlations among fear of pain, catastrophizing, and escape/avoidance and predicted that each in turn would have a direct effect on PTSS. Interestingly,

only fear of pain had a significant direct effect on PTSS, accounting for 56% of the variance. However, although catastrophizing and escape/avoidance behaviors did not have direct effects on PTSS, this does not rule out possible indirect effects of these variables on PTSS through their relationship with fear of pain. That is, high levels of catastrophizing and escape/avoidance behaviors may increase fear of pain, which in turn exacerbates PTSS.

Although analyses suggest that Models 1 and 4 have an adequate fit to the data, it is important to note that these findings do not rule out the possibility of equivalent, or superior, alternate models not tested in this study. Furthermore, the differences in AIC and BCC values between each of the competing models were small, indicating a minimal improvement in fit for Model 1 relative to Model 2 and for Model 4 relative to Model 3. Replication in a different sample is advised before conclusive statements can be made regarding the superiority of one model over the others.

It is also important to note these findings do not imply causality; the data were cross-sectional and as such direction of influence cannot be confirmed. It is therefore difficult to determine how and why PTSS may be related to the variables in the models we examined (Figs. 6 and 7). One potential hypothesis, consistent with Model 4 (Fig. 8), is that individuals with persistent pain who experience a dysfunctional fear-avoidance cycle may be more vulnerable or less able to cope with stressful life events, thus making them more susceptible to the development of PTSS.[70] This explanation leaves open the possibility that PTSS may develop in relation to an initiating painful event.

There are several limitations to this study. First, although numerous patients endorsed the presence of

PTSS, the exact nature of the traumatic event to which patients were referring is unknown. Therefore, although a significant positive relationship between PTSS and pain disability was observed, the extent to which PTSS were related to a painful traumatic event is indeterminate. In addition, information pertaining to the lifetime or current psychologic histories of these patients is unknown and may provide valuable insight into the relationships among the variables in the proposed models. It is possible that some individuals were experiencing PTSS in response to the event that caused their chronic pain, whereas others were not. This is an important distinction that has yet to be addressed in the literature.[34,71] In addition, the PTSS reported by some patients may have been exacerbated in response to a major medical diagnosis and their upcoming surgery. The present data offer a preliminary investigation into the potential relationships among PTSS and the variables in the diathesis-stress model of chronic pain and disability[3]; future study in surgical patients and in other populations is needed to replicate the present findings.

Furthermore, these data were collected at one point in time and experimental manipulations were not applied; therefore, as mentioned earlier, the direction of causality is unknown. Finding a model that fits the data closely does not rule out the possibility of other potential causal models. The small ratio of participants to observed variables is also important to note, as covariances become less stable when estimated from small sample sizes.[72] Future research is needed to examine these models prospectively in larger samples of patients to better understand the nature of vulnerability and the direction of causality. Finally, an important omission from our model was the role of self-efficacy, which is included in the original model.[3] Future research should continue to investigate the relationships proposed in the diathesis-stress model, particularly with respect to the role of self-efficacy and the addition of PTSS.

In conclusion, these results provide empirical support for aspects of Turk's diathesis stress model[3] in a heterogeneous sample of patients with persistent pain. These findings also offer preliminary support for the potential role of PTSS in fear-avoidance models of chronic pain, thus elaborating the complex inter-relationships proposed in the diathesis stress model[3] and adding to the growing evidence-base for a relationship between PTSD and chronic pain. Clinicians treating individuals with pain or PTSS should be aware of the frequent concurrence of these 2 conditions as modifying treatment protocols to address and manage symptoms of both conditions will likely improve the treatment outcomes.

ACKNOWLEDGMENT

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