A cross-sectional evaluation of the fear-avoidance model of chronic pain: Assessing the relationship between neuroticism, negative affect, and pain catastrophizing using structural equation modeling

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Abstract

Purpose: Previous research on the Fear-Avoidance Model (FAM) of chronic pain suggest the personality traits neuroticism and negative affect (NA) influenced pain catastrophizing. However, their influence on pain catastrophizing remains unclear. This study examined four possible models of relationships between neuroticism, NA, and pain catastrophizing within the FAM framework using structural equation modeling.

Methods: A total of 401 patients with chronic musculoskeletal pain completed measures of neuroticism, NA, three core FAM components (pain catastrophizing, pain-related fear, and pain anxiety), and adjustment outcomes (pain-related disability and depression).

Results: Regression analyses refuted the possibility that neuroticism and NA moderated each other's effect on pain catastrophic thoughts (p>0.05). Results of SEM evidenced superior data-model fit for the collapsed models in which neuroticism and NA were two secondary traits underlying a latent construct, negative emotion (Disability: CFI=0.93; Depression: CFI=0.91).

Conclusions: The results offer preliminary evidence that patients presented with more neurotic symptom and heightened NA probably elicit more catastrophic thoughts about pain.

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Keywords: Fear Avoidance Model; neuroticism; negative affect; structural equation modeling.

Introduction

The fear-avoidance model (FAM) of chronic pain [1, 2] (Figure 1), which emphasizes the role of fear-avoidance in the development of pain problems, is a prevailing cognitive-behavioral model of chronic pain in the field. Building on previous research [3-5], the FAM posits that negative appraisal of acute pain evokes pain-related fear leading to avoidance and/or escape behaviors. While avoidance behaviors such as limping and resting can be adaptive for reducing pain during the acute phase, prolonged engagement in avoidance behaviors is detrimental physically, with disuse syndrome producing loss of muscle strength, range of motion, mobility and fitness. Psychologically, avoidance behaviors may facilitate depression and loss of self-esteem may amplify disability [6]. The FAM has attracted considerable research interest in the past 20 years, with studies producing substantial empirical data. The role of pain catastrophic thinking as a precursor of pain-related fear was suggested in both correlational [7, 8] and prospective studies [9-11]. Pain anxiety, on the other hand, has been shown to predict decreased physical performance [12, 13] and disability [14-16], even after controlling for pain severity. Pain-related fear predicated perceived disability at 6-month [17] and 12-month follow-up [18].

Despite this evidence supporting the FAM, contradictory findings were also reported. Several prospective studies reported that fear did not predict pain [19-21], that changes in pain catastrophic cognition failed to predict changes in pain related fear [22, 23], and that the casual path between pain-related fear, pain catastrophizing, and pain disability as specified in the FAM was not significant [24]. One prospective study found no significant sequential relationship between changes in pain catastrophizing and pain-related fear [23]. Recent reviews of the FAM concluded that factors including measurement issues, and the underlying assumptions of the model may have contributed to the mixed findings and have advanced proposals to enhance and expand the model [25-28].

The key mechanism underpinning the FAM is that catastrophic thinking influences avoidance behavior and subsequently amplifies disability. Previous research has presented inconsistent evidence that differences in the primary appraisal of pain experience (catastrophizing vs. non-catastrophizing) affects pain avoidance behaviors and disability [1, 2, 22-24]. In light of these findings, research has attempted to examine why such individual differences exist. It has been hypothesized that negative affect (NA), defined as a mood-dispositional dimension featuring negative emotionality and self-concept [29], and neuroticism, a personality trait characterized by anxiety, moodiness, worry and jealousy [30], possibly underlie individual differences in

pain-catastrophic cognitions [6, 31]. Theoretically, individuals with higher NA should consistently scan their environment for threat indicators, selectively interpreting ambiguous stimuli in a negative and threatening manner [32]. Previous research has documented pain catastrophizing mediated NA and somatic complaints in a sample of children with chronic pain [33], whereas elsewhere the relationship between NA and disability was mediated by fear of (re)injury [34]. Regarding neuroticism, Esteve and Camacho [35] reported a significant moderate relationship between neuroticism and catastrophizing. Goubert et al [36] found that among individuals reporting more neurotic symptoms, even low pain intensity would provoke catastrophic thoughts about pain. These findings were interpreted as indicating a moderating role of neuroticism in the relationship between pain severity and pain catastrophic thoughts. However, in a different 9-month follow-up study, neuroticism did not predict change in catastrophizing [37]. Thus far, little research has empirically corroborated the dispositional role of these two personality traits in pain catastrophizing.

In the wider personality literature, researchers generally support the postulation that neuroticism and NA should be conceptualized at different levels in the hierarchy of personality. Specifically, neuroticism is postulated to represent a higher-order, core personality trait, while NA is postulated to represent a lower-order, and more peripheral secondary trait, . In a large longitudinal study on temporal stability of personality [38], the Big Five traits measured using the Big Five Inventory [39] yielded stability correlations ranging from 0.59 to 0.72 (mean *r*=.64). Trait affectivity scales measured by the Positive and Negative Affect Schedule [40] yielded significantly lower stability correlations of 0.51 on the Positive Affect subscale and 0.49 on the Negative Affect subscale. Similar findings on the differential stability of personality traits have been reported [41, 42]. In a study that examined the relationship between NA, neuroticism, and self-reported physical symptoms, Hull et al. [43] found that only NA predicted symptom reporting whilst neuroticism related to symptoms via NA. The relationships between NA and other personality traits were mediated by the higher-order factor of neuroticism. Grounded on these data, we speculate that a hierarchical model of NA, neuroticism and their relations to pain catastrophizing would be illuminating. Specifically, it is hypothesized that neuroticism is a primary trait related to pain catastrophizing through mediation by NA, which is conceived as a secondary trait. Considering this hierarchical model within the FAM framework, high neuroticism may therefore predispose individuals to interpret sensations as threatening or pain-related, thereby triggering both negative affect and more negative cognition.

However other interpretations suggest that NA and neuroticism can also be treated interchangeably, both in theory and measurement. For instance, Wasan et al. [44] employed a neuroticism scale (the NEO Personality Inventory) [45] to assess NA. When addressing the issue of construct redundancy, Quartana et al [27] pointed out that in previous studies pain catastrophizing has consistently shown to share a significant amount of variance with other general negative affectivity constructs such as depression, anxiety, neuroticism and negative affect [46-49]. These data thus raise the concern of construct distinctiveness, leading to the hypothesis that these variables may belong to a broader negative affect construct.

, Consequently, the aim of the present study was to examine the hypothesized hierarchical model in which NA mediated the relationship between neuroticism and pain catastrophizing within the FAM framework for two pain-related adjustment outcomes, depression and pain-related disability (Figure 2: Model 1 and 2), in a sample of Chinese patients with chronic musculoskeletal pain. Besides the "standard" hypothesized hierarchical model, three alternative models were also tested. First, the "reversed" hierarchical model hypothesized neuroticism mediated NA and pain catastrophizing (Figure 3, Model 3 and 4). Second, the "collapsed" model hypothesized neuroticism and NA both as secondary traits underlying a higher-order, latent construct, "negative emotion", which predicted pain catastrophizing (Figure 4: Model 5 and 6). Finally, we also explored a reciprocal moderating model in which neuroticism and NA moderated each other's effects on catastrophic pain cognition.

Methods

Participants

Following Institution Review Board approval, consecutive patients with chronic musculoskeletal pain in two multidisciplinary pain clinics were invited to participate in this project. Eligible patients met the following criteria: (1) over 17 years of age; (2) native Chinese speakers; (3) no communication or physical problems preventing completion of the interview; (4) no cognitive impairment documented in medical record; and (5) having chronic musculoskeletal pain for at least 3 months

A total of 401 patients with informed consent completed the interview. The mean age of the sample was 43.66 (SD=9.68) years and 58.4% were female. About 52.3% reported monthly household incomes of <HK$15,000 (US$1,923) and 56.4% were married or cohabited; 68.6% had completed secondary and 10.3% tertiary education. While 62.5% reported no particular religious belief, 19.8% endorsed Buddhism, Daoism or

ancestor worship. Less than half (41.4%) of the patients reported being in full-time employment, whereas 34.9% and 12.7% of the sample respectively described themselves as unemployed or homemakers.

Measures

Face-to-face interviews were conducted at the clinics using a structured questionnaire, comprising standardized measures of the FAM components and questions on socio-demographic and clinical characteristics.

Chronic Pain Severity and Disability: Chronic pain severity and disability were assessed using the Chronic Pain Grade (CPG) questionnaire [50], a seven-item instrument that measures three domains of pain severity in the 3 months preceding the day of the interview: persistence, intensity and disability/interference. Three intensity items ask respondents to rate their current, average and worst pain intensity on 0–10 Numerical Rating Scales (NRS) (0="No pain at all"; 10="Pain as bad as could be"). A Characteristic Pain Intensity Score is derived by averaging the responses to the intensity items and multiplying this by 10. Three CPG items assess pain interference with (1) daily activities, (2) social activities, and (3) working ability using 0–10 NRSs (0="No interference/change"; 10="Extreme change/Unable to carry on activities"). The CPG Disability Score (Pain-Dis) is derived by multiplying the average of the three interference items by 10. Persistence is assessed in the CPG by asking the respondent to indicate the number of days out of the past three months that he/she was disabled by pain. The Disability Score and the number of disability days are recoded into 5-point scales and summed, yielding "Disability Points". Based on the Pain Intensity Score and Disability Points, the CPG classifies subjects into five hierarchical grades (see Table 1). The English version of the CPG possesses good psychometric properties [51] and is responsive to change in pain severity over time [52]. The underlying structure of the Chinese version of the CPG demonstrated good psychometric properties, with Cronbach's αs for the CPG Disability and Characteristic Intensity scales of 0.87 and 0.68 [53].

Depression: Depression was evaluated using the 7-item depression subscale of the Hospital Anxiety and Depression Scale (HADS-Dep) [54]. The HADS-Dep subscale is scored between 0 and 21, with higher scores indicating more depressive symptoms. Test-retest reliability for the HADS-Dep is good (r=0.92) and internal consistency high (Cronbach's α=0.90) [55]. The Chinese version has been validated, yielding good

internal consistency (Cronbach's αs ranging from 0.77 to 0.86) and test-retest reliability [56, 57]. The HADS-Dep was chosen as a measure of depression in the current study as the scale places more emphasis on affective and behavioral symptoms of depression than on cognitive and physical symptoms, thereby minimizing the sequelae of chronic pain rather than emotional dysfunction.

Neuroticism: The 12-item neuroticism subscale from the Big Five Personality Questionnaire (NEO-N) was employed for measuring neuroticism [45]. Respondents were asked to rate on a 5-point scale (ranging from "strongly disagree" to "strongly agree") and the scores can range between 12 and 60, with higher scores indicating more neurotic symptoms. The factor structure of the Chinese version closely resembles the structure of the original instrument with acceptable internal consistency (Cronbach's α=0.84) [58].

Negative Affect: The 10-item Negative Affect subscale from the Positive and Negative Affect Schedule (PANAS-NA) was used to assess NA [40]. Respondents were asked to indicate the extent to which they experienced NA during the week preceding the interview on a 5-point scale (1="very slightly or not at all"; 5="extremely"), with higher scores indicating more negative affect. The PANAS is a reliable, valid, and widely used measure [40]. The Chinese version of PANAS possesses good reliability (Cronbach's α=0.87) [59].

Pain Catastrophizing: Pain-related catastrophizing was assessed using the 13-item Pain Catastrophizing Scale (PCS), which consists of three subscales: Rumination (PCS-RUM), Magnification (PCS-MAGN), and Helplessness (PCS-HELP) [60]. Respondents were asked to reflect on past painful experiences and to indicate the frequency with which they experienced each of 13 thoughts or feelings when experiencing pain on a 5-point scale (0="not at all"; 4="very often"), with higher scores indicating higher pain catastrophizing. The PSC has demonstrated good internal consistency (Cronbach's α for the total scale=0.87), test-retest reliability at 6 weeks (r=0.75), and construct validity [60]. The Chinese PCS also showed good psychometric properties (Cronbach's α for the total score=0.93, item-total correlation coefficients ranging from 0.58 to 0.78) [61].

Pain-Related Fear: Pain-related fear was assessed by the Tampa Scale for Kinesiophobia (TSK) [62]. The 11-item TSK, with two subscales including Somatic Focus (TSK-SF) and Activity Avoidance (TSK-AA), was designed to measure fear of (re)injury and movement. Respondents were asked to rate on a 4-point scale (1="strongly disagree"; 4="strongly agree") and higher scores suggest higher pain-related fear. Previous studies showed that the scale possessed good internal consistency and test-retest reliability [63, 64]. The Chinese version of TSK has been validated and demonstrated acceptable psychometric properties (Cronbach's α for the total score=0.67) [65].

Pain Anxiety: The short version of the Pain Anxiety Symptoms Scale (PASS-20) was employed to measure pain anxiety [66]. Consisting of 20 items, the PASS-20 was developed to assess anxiety that is associated with clinical pain symptoms. Rating on a 6-point scale (1="never"; 5="always"), the scale is composed of 4 subscales: Cognitive Anxiety (PASS-CA), Avoidance (PASS-AV), Fear (PASS-FE), and Physiological Anxiety (PASS-PA). Higher scores indicate higher pain anxiety. Internal consistency, reliability, and construct validity were evidenced in a sample of chronic pain patients [66]. The four subscales and the entire scale of the Chinese version of the PASS-20 also demonstrated good internal consistency (Cronbach's αs: 0.72–0.92) [67].

Statistical Analyses

Descriptive statistics were used to summarize the pain and psychosocial characteristics of the sample. A Pearson correlation matrix displayed bivariate relationships between outcome and psychosocial variables. Multiple regression models were used to test the hypothesized relationship between neuroticism and NA in predicting pain catastrophizing. For NA to be a mediator of neuroticism and pain catastrophizing, four criteria [68] need to be met: (1) neuroticism (predictor) should significantly predict NA (mediator); (2) NA (mediator) should significantly predict pain catastrophizing (outcome); (3) neuroticism (predictor) should significantly predict pain catastrophizing (outcome); and (4) controlling for NA (mediator), the relationship between neuroticism (predictor) and pain catastrophizing (outcome) should be reduced or no longer significant. Perfect mediation is established if the association between neuroticism and pain catastrophizing is reduced to zero. Sobel test [69] was used to determine whether the mediating effect of NA in the relationship between neuroticism and pain catastrophizing was statistically significant (p<0.05). A series of four regression models

were used to test each of these three-variable mediation chains. To test the relationship between neuroticisim and NA in the "reversed" hierarchical models, two more regression models were fitted with (1) NA (predictor) predicting neuroticism (mediator), and (2) NA (predictor) predicting pain catastrophizing (outcome) controlling for neuroticism (mediator). To test the possible moderation pathway of the relationship between neuroticism, NA, and pain catastrophic cognition, pain catastrophizing was regressed on neuroticism and NA to study the main effects, and on a two-way interaction term Neuroticism × NA, to examine interaction effect. Variables were centered in the regression analyses of moderation pathway to control for the effects of multicollinearity [70]. All regression models were adjusted for age, gender, average pain intensity, pain duration and number of pain sites. Low multicollinearity (observed tolerance values 0.48-0.99) between variables was observed. Descriptive statistics and regression analyses were conducted using SPSS [71].

Cross-sectional structural equation modeling (SEM) was conducted to assess the goodness of fit of three competing models in the FAM framework for two pain adjustment outcomes, pain-associated disability and depression. The hypothesized hierarchical FAM (Models 1-2) incorporated the hierarchical relation between neuroticism and NA into the original FAM model, in which NA was hypothesized to mediate the relationship between neuroticism and pain catastrophizing. The reversed hierarchical FAM (Models 3-4) incorporated a reversed hierarchical relation between neuroticism and NA into original FAM model, in which neuroticism was hypothesized to mediate the relationship between NA and pain catastrophizing. In the collapsed FAM (Models 5-6), pain catastrophizing (the predictor variable of the FAM) was predicted by a latent construct of negative emotion, which was represented by neuroticism and NA. All SEM models were adjusted for average pain intensity. Model fit was assessed using Satora-Bentler χ^2 (S-Bχ^2) statistics, comparative fit index (CFI), non-normed-fit index (NNFI), root mean square error of approximation (RMSEA), 90% confidence interval (CI) of RMSEA, and standardized root mean square residual (SRMR). CFI and NNFI value of ≥0.90, RMSEA value of ≤0.05, and SRMR value of ≤0.08 are indicative of adequate fit [72, 73]. All SEM was performed using MPlus [74].

Results

Pain characteristics

The present sample averaged 3.20 (SD=2.41; range: 1-16) pain sites with 20.4% reporting a single pain site and 79.4% multiple pain sites (Table 1). The three most common pain sites were low back (38.6%), followed by foot (44.1%), and neck (37.4%). Patients reportedly experienced an average of 5.24 years (SD=5.15, median=3.49, range, 3 months to 30 years) of pain problems. About 34.1% had experienced pain for up to 2 year's duration and 32.6% had suffered from chronic pain for more than 5 years. The mean scores of present, average, and worst pain were 5.32 (SD=2.36), 6.42 (SD=1.66), and 8.41 (SD=1.62), respectively. On pain interference measures, the sample reported a mean score of 6.37 (SD=2.25), 6.39 (SD=2.82), and 6.65 (SD=2.91) for daily activity, social activity, and working ability interference, respectively. The sample reported an average of 35.16 days (SD=41.27; range: 0-90 days) of pain-associated disability. The CPG classification placed 60.9% of the sample as Grade III or above (high disability and moderately-to-severely limiting).

Means, standard deviation and intercorrelations of the measurement scales

All measurement scales were significantly correlated with each other ($p<0.01$) (Table 2). The strength of relationship between the two variables that were hypothesized to explain pain catastrophizing, neuroticism and NA, was high at $r=0.80$ ($p<0.01$). Regarding their relationships with the outcome variables, neuroticism and NA were more strongly correlated with HADS-Dep (*r*s: 0.60-0.66) than Pain-Dis (*r*s: 0.37-0.38). The correlations of neuroticism and NA with the subscales of PCS were moderate, ranging from 0.46-0.68 (all $p<0.01$).

Multiple regression analyses for the mediation pathway of the link between neuroticism, NA, and pain catastrophizing

In testing the mediating role of NA in the link between neuroticism and pain catastrophizing, results of multiple regression analyses showed that, after controlling for potential confounding variables, higher neuroticism was significantly associated with higher NA ($\beta=0.67$, $p<0.001$) and higher pain catastrophizing ($\beta=0.69$, $p<0.001$) (Table 3). NA was significantly associated with pain catastrophizing in a positive direction ($\beta=0.74$, $p<0.001$). When NA was controlled, neuroticism was significantly associated with pain catastrophizing ($\beta=0.49$, $p<0.001$) (Sobel $z=3.63$, $p<0.001$).

As for the mediating role of neuroticism in the relationship between NA and pain catastrophizing, results of multiple regression models indicated that, after controlling for potential confounding variables, higher NA was significantly associated with higher neuroticism (β=0.92, p<0.001). When neuroticism was controlled, NA was significantly associated with pain catastrophizing (β=0.29, p<0.001) (Sobel z=7.04, p<0.001).

Multiple regression analyses for the moderation pathway of the link between neuroticism, NA, and pain catastrophizing

In examining the possible moderation pathway, the result of multiple regression analyses revealed that the main effect of neuroticism on pain catastrophizing was significant (β=0.48, p<0.001) (Table 4). NA also significantly predicted pain catastrophic thought (β=0.32, p<0.001). However, the two-way interaction term, Neuroticism × NA, was not significant (β=-0.01, p>0.05). These findings excluded the possible moderation pathway of NA and neuroticism on pain catastrophizing.

Multivariate model testing

Prior to testing our hypothesized model with latent variables, the measurement model was evaluated. The latent construct of neuroticism and NA were specified by NEO-N and PANAS-NA respectively. Pain catastrophizing was estimated by the three subscales of PCS: PCS-RUM, PCS-MAGN, and PCS-HELP. Pain-related fear was specified by the two TSK subscales: TSK-AA and TSK-SS. Pain anxiety was specified by the four PASS-20 subscales: PASS-AV, PASS-FE, PASS-CA, and PASS-PA. Negative emotion was specified by NEO-N and PANAS-NA. The two pain adjustment outcomes were specified by the CPG Disability score and the HADS-Dep score. Results of the SEM showed a good fit of the measurement model to the data: S-Bχ^2(60)=185.58 (p<0.001), CFI=0.96, NNFI=0.92, RMSEA=0.08 (90% CI: 0.07, 0.09). These findings suggest that the measurement scales employed in the model can be considered valid operationalizations of the latent constructs of neuroticism, NA, pain catastrophizing, pain-related fear, pain anxiety, and the two pain adjustment outcomes (HADS-Dep and Pain-Dis).

The results of SEM on the hypothesized hierarchical models (Model 1-2) for the two pain adjustment outcomes (Table 5) indicated adequate data-model fit (CFI=≥0.91) for the disability model (Model 1), with all pain coefficients were statistically significant (p<0.001) (Figure 2). The CFI of depression model (Model

4) was 0.89, suggesting inadequate data-model fit (Figure 1). The two "reversed" hierarchical models, Model 3 and Model 4, demonstrated adequate data-model fit (CFI≥0.90), with all path coefficients statistically significant (p<0.05) (Figure 3). Of the 6 models tested, the two "collapsed" models (Model 5-6) (Figure 4) reported the best data-model fit (CFI≥0.91), with Model 7 yielding the highest CFI (=0.93). The standardized path coefficients of the collapsed models were all statistically significant (p<0.05). However the model chi-square test is grounded on the assumption of multivariate normality {Bentler, 1980 #2562;McIntosh, 2006 #3962}, and since our data were non-normally distributed, so the chi-square statistics of all models are significant (p<0.001) suggesting poor fit and, misleadingly, model rejection. For this reason, chi-square statistics were disregarded in the interpretation of the findings.

Discussion

This cross-sectional study examined the four competing models of relationship between neuroticism, NA, and pain catastrophic cognition within the FAM framework. Univariate analyses indicated both neuroticism and NA were associated with pain catastrophizing, pain-related fear, pain anxiety and the two pain adjustment outcomes assessed. Although the results of both multiple regression analyses and SEM suggested significant effects of neuroticism and NA on pain catastrophic thought, the hypothesized hierarchical model, where NA played a mediating role in the link between neuroticism and pain catastrophizing, was not the best fitting model amongst the three competing SEM models tested. Instead, the collapsed model in which neuroticism and NA were hypothesized as parameters explaining a latent construct, Negative Emotion, was shown to be the best fitting model tested. These findings suggest an intricate entanglement of the two (possibly homologous) personality constructs, neuroticism and NA, and their impact on pain catastrophic cognition.

Results of multivariate regression analyses offered no evidence for NA and neuroticism moderating each other's effect on pain catastrophic thoughts. After controlling for sociodemographic and pain variables in the multivariate regression model, the interaction variable (neuroticism × NA) failed to demonstrate a significant effect on pain catastrophic thought, suggesting that lower NA does not buffer the detrimental effects of neuroticism, and vice versa, on pain catastrophizing among patients with chronic pain. Yet, results of regression analyses demonstrated that both neuroticism and NA partially mediated the effects of the other on

pain catastrophizing. The indirect effect of NA on the link between neuroticism and pain catastrophizing was 0.20 (without mediation minus with mediation), suggesting up to 29% of the impact of neuroticism on pain catastrophic cognition reflects variation in NA, but about 68% of the effect was directly due to the stable trait of neuroticism. However, when neuroticism was a mediator, its indirect effect on the link between NA and pain catastrophizing was 0.45 (without mediation minus with mediation), suggesting nearly 61% of the impact NA on pain catastrophizing reflects variation in neuroticism. The higher z scores in the model with neuroticism being the mediator further suggested this model has a higher precision than the model with NA being the mediator in explaining the variance of the NA-neuroticism-pain catastrophizing link. The stronger mediating effects of NA found in this study are also in line with the SEM when the relationships were assessed in the FAM framework. The two reversed hierarchical models (Model 3 and 4) obtained a better data-model fit than that of the hypothesized hierarchical models (Model 1 and 2). These findings depart from previous research that proposed neuroticism 1) influences patients' NA thereby affecting pain catastrophic thoughts indirectly, and 2) is influenced by more widely recognized direct effects [75-77]. Our data also contradict the empirical evidence for the theoretical postulation that neuroticism and NA can be conceptually related using a hierarchical model, wherein the former is the primary and the latter a secondary trait [78, 79].

Notably, our SEM data evidenced the collapsed models (Model 5 and 6) that reflect a latant negative-affective trait, generated superior fit indices compared to other hierarchical models tested. These findings are not surprising given the high correlation between neuroticism and NA (r=0.80) in univariate analysis, commensurate with previous studies [41, 42]. These data suggest neuroticism and NA are better taken as two dispositions at the same conceptual level, instead of as hierarchical: Neuroticism as a stable personality trait increases the tendency to perceive threat in neutral or ambiguous stimuli; NA as a mood dispositional that drives attention towards unpleasant aspects of the world, such as pain symptoms. Jointly, they seem to amplify or increase the likelihood of catastrophic cognitions regarding pain. These cognitions in turn may feed forward amplifying both NA and neuroticism tendencies.

Unfortunately, our data cannot determine whether neuroticism and NA can be treated interchangeably [44]. While the correlations of neuroticism and NA with depression were moderately high (rs ranging from 0.60 to 0.66), the correlations of the two dispositions with other negative pain variables, such as pain catastrophizing (rs ranging from 0.46 to 0.68) and pain anxiety (rs ranging from 0.59 to 0.60) were not

consistently high in this study. Furthermore, except for a few correlations that were higher than <0.60 (PCS-HELP-PASS-FE, PCS-HELP-PASS-CA, PCS-MAGN-PASS-FE, PCS-MAGN-PASS-CA), the relationships amongst the negative pain variables (pain catastrophizing, pain-related fear, and pain anxiety) were not high (*r*s ranging between 0.29 and 0.59). None of the correlations between pain variables and depression was higher than 0.60. Our data hence fail to confirm Quartana et al's [27] speculation about construct redundancy of negative pain schema and negative affect constructs. This issue of construct redundancy or uniqueness awaits resolution through more research by taking into account issues related to the limitations of measurements, underlying assumptions, dimensionality, and other methodological elements [26-28]. In particular, since neuroticism and NA are both negative affect constructs, the present examination of three competing models is largely grounded in a psychopathology approach {Pincus, 2010 #3950}. Other possible approaches with which pain experiences and responses are seen in a more normative and culturally endorsed perspective would help delineate a more accurate account of the relationship between pain variables and adjustment outcomes [25].

The results of SEM in this study add to the existing pain literature on FAM. Thus far, the studies that examined the bidirectional relationships of the key components of the FAM were all conducted in Western populations [19-23, 36, 80, 81]. Findings of this study are consistent with the view that the basic premises of the FAM appear to generalize across cultures. These findings provide an important theoretical and empirical base for future research to clarify cultural similarities and differences among patients with chronic pain.

The relationships between neuroticism and NA in all models tested remained consistent with the FAM framework. Except for Model 2, all models tested in SEM achieved adequate data-model fit (CFI≥0.90). The FAM of chronic pain was postulated to account for the development and maintenance of chronic pain, and the constitutive components of the model mainly involve pain-related antecedents (pain catastrophizing, pain-related fear, and pain anxiety) of negative pain adjustment outcomes. Research has attempted to examine dispositional factors that explain underlying individual differences in the pain-related components [6, 31, 32, 34-37]. Our findings extend previous data that neuroticism and NA can be incorporated in the FAM as dispositional parameters associated with pain catastrophic thinking. These dispositions appear largely interchangeable, within the context of the FAM, which perpetuates questions about their conceptual independence. This elaborated model may help explain the complex underlying individual differences in

pain-related components in the FAM, as previous studies have inconsistently demonstrated the associations of neuroticism and NA with catastrophic cognition in pain [32, 33, 35]. Clinically, this elaborated model offers practical value by suggesting how personal determinants associate with pain catastrophizing. With more data on the unique cluster of risk factors (which could consist of sociodemographics, general and pain-related psychological factors, and clinical characteristics) associated with poor pain adjustment outcomes, screening programs could be designed to identify patients at risk for developing pain avoidance behaviours and subsequent disability. Matching education and intervention programs may also be designed to accommodate patients with at-risk characteristics. For instance, cognitive-behavioural education/intervention programs could be designed to teach newly diagnosed pain patients with high neuroticism and NA about reappraising and managing general negative affective states and dispositions using positive and adaptive methods before the maladaptive avoidance pain cycle develops. Further research is needed that tests the elaborated FAM under different clinical and cultural conditions to further clarify causal pathways in predicting pain adjustment outcomes.

Study limitations include, first, the cross-sectional nature of the data, limiting etiological inference and causality. Despite illuminating the complexity of the studied interrelationships the causal directions remain unknown. Future research utilizing longitudinal prospective designs and experimental studies could help delineate these causal chains. Second, pain diagnosis, previous/concurrent medical services sought, and other medical comorbidity, were not assessed in this study. These may influence cognitive-affective dynamics. Third, all patients had a mix of pain problems, and it is possible that the different meanings attributable to these generate different response characteristics. Fourth, as the hierarchical and collapsed models of neuroticism and NA in this study were evaluated in the context of chronic pain, replication and extension of our findings in other populations and context are encouraged. Fifth, we employed SEM to examine the hypothesized models as this modelling method has the advantage of ruling out errors of variance. However, because three latent constructs examined in this study (neuroticism, negative affect, and pain adjustment) were assessed using only single measures, path analyses could be a more appropriate approach in this regard. Finally, the SEM model fit was not excellent, suggesting the imprecision of the original and elaborated FAMs in Chinese populations, or measurement error remains to be accounted for. More research is needed to clarify how much such error is attributable to cultural and/or other methodological differences.

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Declaration:

This study conformed to the Helsinki Declaration concerning human rights and informed consent, and it also followed correct procedures concerning treatment of humans and animals in research.

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Table 1: Pain characteristics of the sample (n=401)

Pain Characteristics	%
Number of pain sites; M (SD)	3.20 (2.41)
1	20.7
2	28.2
3-5	38.7
≥6	12.5
Pain site	
Head	11.7
Neck	37.4
Shoulder	30.4
Hand/arm	30.2
Chest	8.0
Spine	10.2
Upper back	17.2
Low back	59.8
Pelvis	18.2
Knee	24.7
Foot	44.1
Joint	3.7
Muscle	3.5
Others	11.2
Pain duration (days); M (SD)	1914 (1879)
Pain duration (days)	1275
≥ 3 months - 2 years	34.1
> 2 years - 5 years	33.3
> 5 years - 10 years	21.6
> 10 years	11.0
Pain intensity[a]; M (SD)	
Present pain	5.32 (2.36)
Average pain	6.42 (1.66)
Worst pain	8.41 (1.62)
Pain interference[b]; M (SD)	
Daily activities	6.37 (2.25)
Social activities	6.39 (2.82)
Working ability	6.65 (2.91)
Pain associated disability (days); M (SD)	35.16 (41.27)
Chronic Pain Grade classification[c]	
Grade Zero	---
Grade I	8.1
Grade II	31.0
Grade III	21.4
Grade IV	39.5

Note: The pain intensity and pain interference scores were drawn from individual items of the Chronic Pain Grade questionnaire.

[a] Scores range from 0-10; higher scores indicate higher intensity of pain.

[b] Scores range from 0-10; higher scores indicate higher level of interference.

[c] Grade Zero: pain free; Grade I: low disability-low intensity; Grade II: low disability-high intensity; Grade III: high disability-moderately limiting; Grade IV: high disability-severely limiting.

Table 2: Mean, standard deviations (SD), and correlations of measurement scales

Scale	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12
1. Pain Disability	64.78 (23.13)	-											
2. HADS-Depression	9.32 (5.90)	0.48**	-										
3. PCS-Helplessness	14.97 (6.64)	0.39**	0.52**	-									
4. PCS-Magnification	6.54 (3.84)	0.34**	0.55**	0.76**	-								
5. PCS-Rumination	9.57 (4.62)	0.29**	0.34**	0.64**	0.65**	-							
6. TSK-Somatic Focus	13.61 (3.00)	0.30**	0.39**	0.43**	0.50**	0.32**	-						
7. TSK-Activity Avoidance	16.97 (3.29)	0.28**	0.32**	0.35**	0.43**	0.32**	0.58**	-					
8. PASS-Avoidance	18.13 (5.89)	0.47**	0.47**	0.42**	0.41**	0.36**	0.33**	0.40**	-				
9. PASS-Fear	13.12 (6.85)	0.39**	0.52**	0.67**	0.71**	0.53**	0.50**	0.44**	0.49**	-			
10. PASS-Cognitive Anxiety	16.28 (6.97)	0.51**	0.58**	0.64**	0.63**	0.54**	0.43**	0.40**	0.62**	0.71**	-		
11. PASS-Physiological Anxiety	10.81 (6.53)	0.38**	0.42**	0.40**	0.42**	0.33**	0.35**	0.29**	0.44**	0.52**	0.59**	-	
12. NEO-Neuroticism	34.24 (12.34)	0.37**	0.66**	0.60**	0.68**	0.51**	0.43**	0.38**	0.37**	0.60**	0.57**	0.48**	-
13. PANAS-Negative Affect	25.09 (10.69)	0.38**	0.60**	0.58**	0.67**	0.46**	0.39**	0.40**	0.33**	0.59**	0.57**	0.53**	0.80**

**Correlation is significant at the 0.01 level

Table 3: Multivariate regression analyses of mediation pathway of the link between neuroticism, negative affect, and pain catastrophizing

	β	SE	95% CI	P value
Neuroticism → Negative affect	0.67	0.03	0.62, 0.72	<0.001
Age (Controlled variable)	-0.02	0.03	-0.09, 0.04	ns
Sex (Controlled variable)	-0.49	0.66	-1.79, 0.80	ns
Number of pain site (Controlled variable)	-0.12	0.14	-0.39, 0.16	ns
Pain intensity (Controlled variable)	0.08	0.02	0.04, 0.12	<0.001
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Negative affect → Neuroticism	0.92	0.04	0.85, 0.99	<0.001
Age (Controlled variable)	-0.08	0.04	-0.16, 0.00	ns
Sex (Controlled variable)	0.69	0.77	-0.83, 2.21	ns
Number of pain site (Controlled variable)	0.14	0.17	-0.18, 0.47	ns
Pain intensity (Controlled variable)	-0.01	0.03	-0.06, 0.04	ns
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Negative affect → Pain catastrophizing	0.74	0.05	0.64, 0.85	<0.001
Age (Controlled variable)	-0.02	0.06	-0.13, 0.10	ns
Sex (Controlled variable)	-0.27	1.09	-2.42, 1.88	ns
Number of pain site (Controlled variable)	0.07	0.23	-0.39, 0.53	ns
Pain intensity (Controlled variable)	0.10	0.04	0.03, 0.18	<0.01
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Neuroticism → Pain catastrophizing	0.69	0.04	0.60, 0.77	<0.001
Age (Controlled variable)	0.01	0.06	-0.10, 0.12	ns
Sex (Controlled variable)	-0.76	1.04	-2.81, 1.29	ns
Number of pain site (Controlled variable)	-0.03	0.22	-0.47, 0.41	ns
Pain intensity (Controlled variable)	0.13	0.03	0.06, 0.19	<0.001
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Neuroticism (Predictor) → Pain catastrophizing (Outcome) | Negative affect (Mediator)[a]	0.49	0.07	0.36, 0.63	<0.001
Age (Controlled variable)	0.02	0.05	-0.09, 0.13	ns
Sex (Controlled variable)	-0.61	1.03	-2.63, 1.40	ns
Number of pain site (Controlled variable)	0.00	0.22	-0.43, 0.44	ns
Pain intensity (Controlled variable)	0.11	0.03	0.04, 0.18	<0.01
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Sobel test	$Z = 3.63$		$P < 0.001$	
Negative affect (Predictor) → Pain catastrophizing (Outcome) | Neuroticism (Mediator)[b]	0.29	0.08	0.13, 0.45	<0.001
Age (Controlled variable)	0.02	0.05	-0.09, 0.13	ns
Sex (Controlled variable)	-0.61	1.03	-2.63, 1.40	ns
Number of pain site (Controlled variable)	0.00	0.22	-0.43, 0.44	ns
Pain intensity (Controlled variable)	0.11	0.03	0.04, 0.18	<0.01
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns
Sobel test	$Z = 7.04$		$P < 0.001$	

Note: β: Beta coefficient; SE: standard error; CI: confidence interval; NS: non-significant P value at 0.05 level. Four separate regression models were generated to test the mediation pathway of negative affect on the link between neuroticism and pain catastrophizing.

[a] Negative affect, as mediator, was controlled in the regression equation.

[b] Neuroticism, as mediator, was controlled in the regression equation.

Table 4: Multivariate regression analyses of moderation pathway of the link between neuroticism, negative affect, and pain catastrophizing

	β	SE	95% CI	P value
Neuroticism	0.48	0.07	0.35, 0.62	<0.001
Negative affect	0.32	0.08	0.16, 0.47	<0.001
Neuroticism × Negative Affect	-0.01	0.00	-0.01, 0.00	ns
Age (Controlled variable)	0.03	0.05	-0.08, 0.13	ns
Sex (Controlled variable)	-0.51	1.03	-2.53, 1.51	ns
Number of pain site (Controlled variable)	-0.01	0.22	-0.44, 0.42	ns
Pain intensity (Controlled variable)	0.11	0.03	0.04, 0.18	<0.01
Pain duration (Controlled variable)	-0.00	0.00	-0.00, 0.00	ns

Note: β: Beta coefficient; SE: standard error; CI: confidence interval; NS: non-significant P value at 0.05 level. All regression equations were controlled for age, sex, number of pain site, pain intensity, and pain duration. Using pain catastrophizing as dependent variable, one regression model was generated to test the moderation pathway of the link between neuroticism, NA, and pain catastrophizing.

Table 5: Results of SEM testing four competing FAMs for two pain adjustment outcomes

Model	S–Bχ^2	*df*	*p value*	CFI	NNFI	RMSEA	90% CI	SRMR
Hypothesized Hierarchical Models: NA mediating the link between neuroticism and pain catastrophizing								
Model 1: Disability	299.92	62	<0.001	0.91	0.89	0.10	0.09, 0.11	0.08
Model 2: Depression	365.72	62	<0.001	0.89	0.86	0.11	0.10, 0.13	0.09
"Reversed" Hierarchical Models: Neuroticism mediating the link between NA and pain catastrophizing								
Model 3: Disability	271.16	62	<0.001	0.92	0.90	0.09	0.08, 0.11	0.07
Model 4: Depression	332.69	62	<0.001	0.90	0.88	0.11	0.10, 0.12	0.08
"Collapsed" Models: NA and neuroticism represent a latent construct, negative emotion, predicting pain catastrophizing								
Model 5: Disability	238.44	61	<0.001	0.93	0.92	0.09	0.08, 0.10	0.06
Model 6: Depression	300.40	61	<0.001	0.91	0.89	0.10	0.09, 0.11	0.06

Note: All models are adjusted for pain intensity. Disability was indexed by the Chronic Pain Grade Disability score; Depression was indexed by the Depression subscale of the Hospital Anxiety and Depression Scale; S–Bχ^2 = Satorra & Bentler scaled chi-square statistics; *df* = degrees of freedom; CFI = comparative fit index; NNFI = non-normed fit index; RMSEA = root mean square error of approximation; CI = confidence interval; SRMR = standardized root mean square residual.

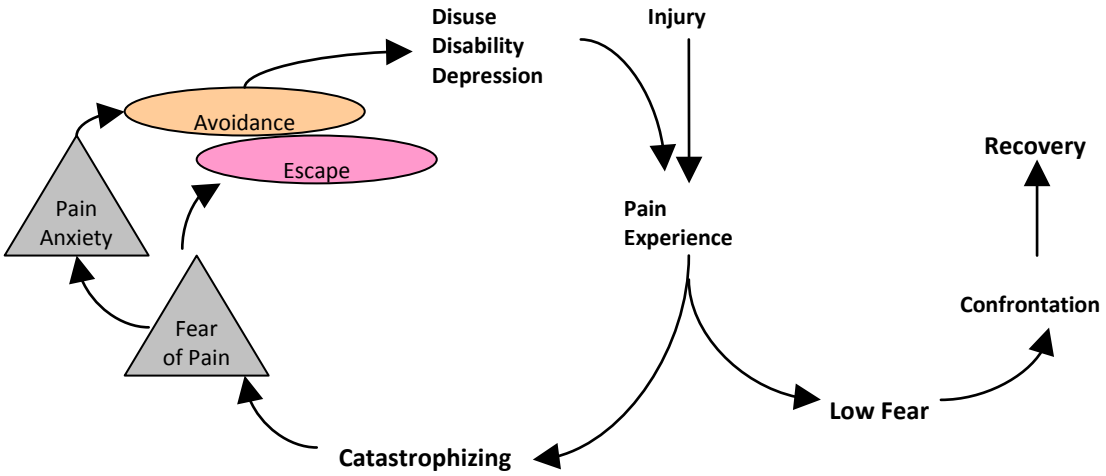
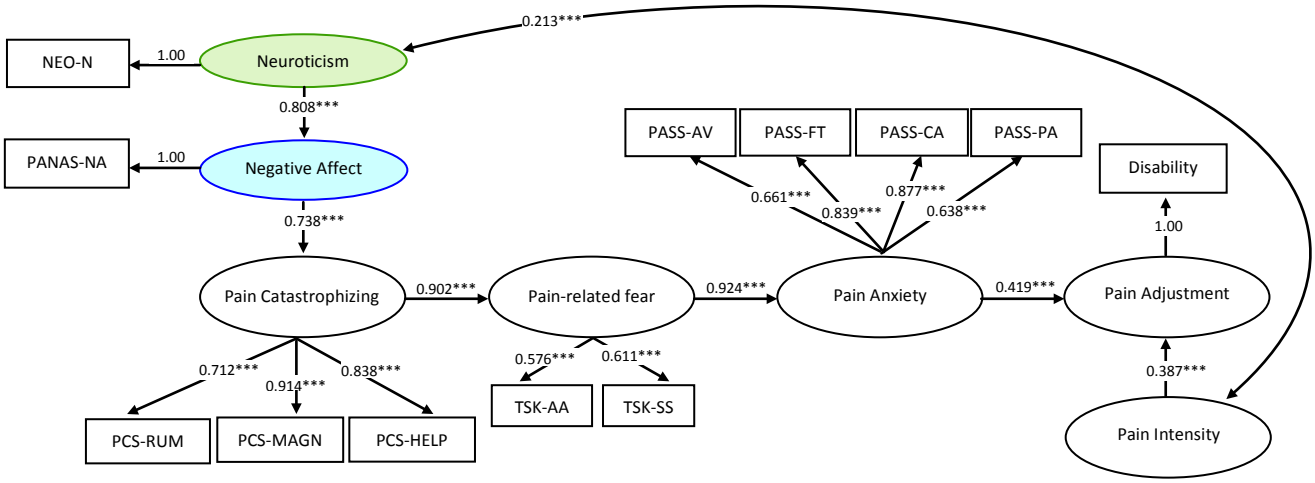
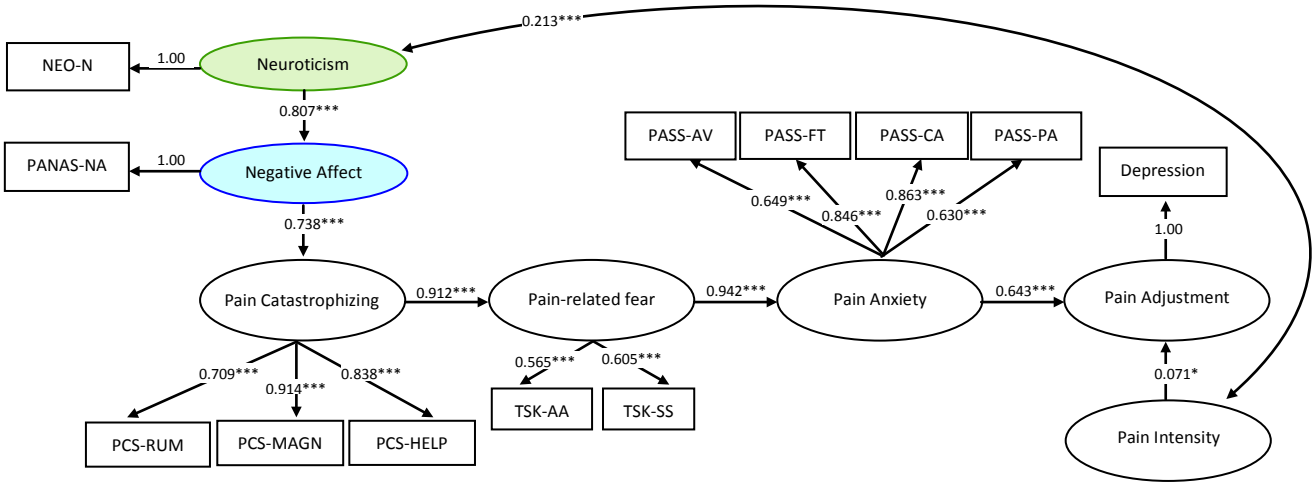


Figure 1: The fear-avoidance model of chronic pain. Adapted from Leeuw et al.[31]

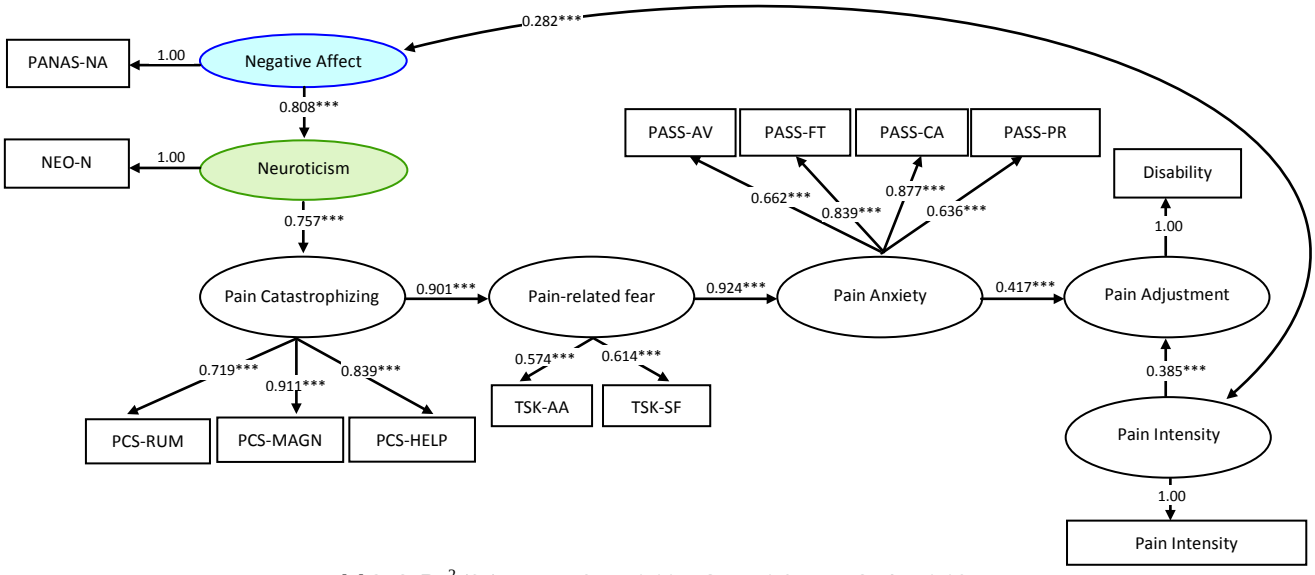


Model 1: S–Bχ^2 (62) = 299.92, p< 0.001, CFI = 0.91, RMSEA = 0.10.

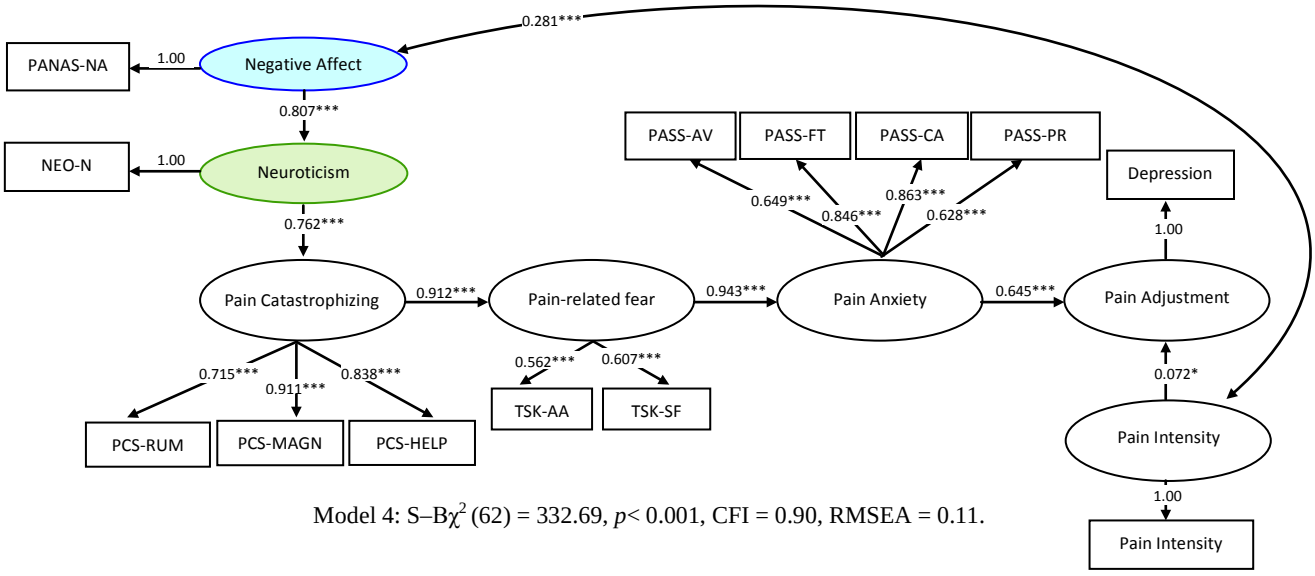


Model 2: S–Bχ^2 (62) = 365.72, p< 0.001, CFI = 0.89, RMSEA = 0.11.

Figure 2: The hypothesized hierarchical models fitted for two pain adjustment outcomes. Numbers are standardized β coefficients. PANAS-NA, the negative affect subscale of PANAS; NEO-N, neuroticism subscales of NEO; PCS, Pain Catastrophizing Scale; RUM, the PCS Rumination subscale; MAGN, the PCS Magnification subscale; HELP, the PCS Helpnessless subscale; TSK, the Tampa Scale for Kinesiophobia; AA, the TSK Activity Avoidance subscale; SF, the TSK Somatic Focus subscale; PASS, Pain Anxiety Symptoms Scale; AV, the PASS Avoidance subscale; FT, the PASS Fear subscale; CA, the PASS Cognitive Anxiety subscale; PR, the PASS Physiological Responses subscale; S – Bχ^2 = Satorra & Bentler scaled chi-square statistic; CFI, comparative fit index; RMSEA, root-mean-square error of approximation. *p<0.05, ***p<0.001.

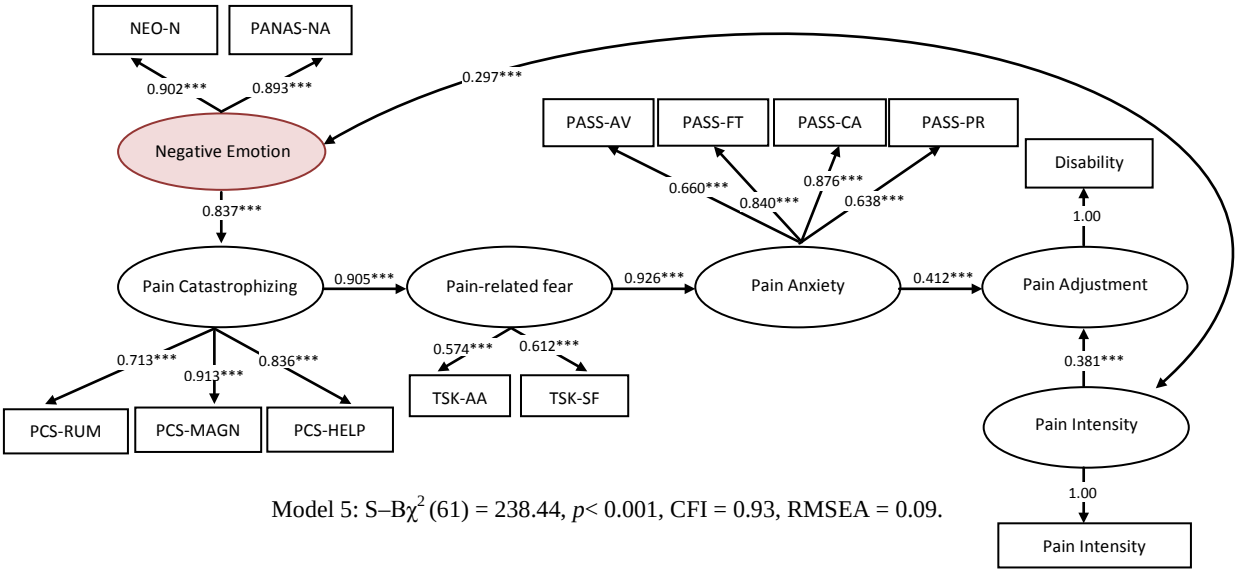


Model 3: S–Bχ^2 (62) = 271.16, p< 0.001, CFI = 0.92, RMSEA = 0.09.

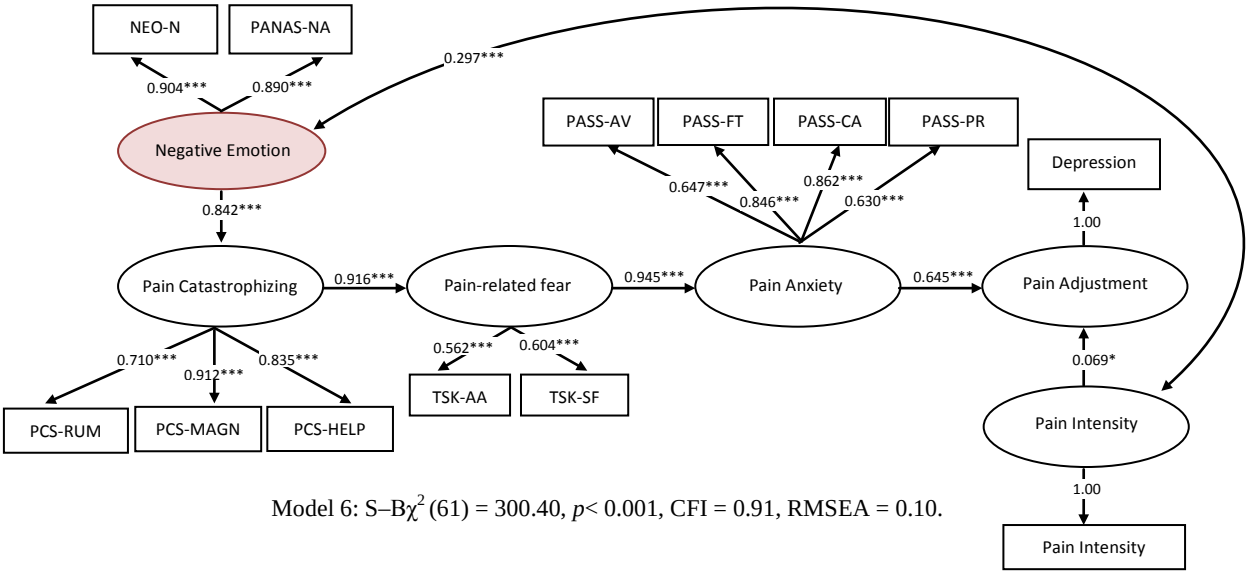


Model 4: S–Bχ^2 (62) = 332.69, p< 0.001, CFI = 0.90, RMSEA = 0.11.

Figure 3: The "reversed" hierarchical models fitted for two pain adjustment outcomes. Numbers are standardized β coefficients. PANAS-NA, the negative affect subscale of PANAS; NEO-N, neuroticism subscale of NEO; PCS, Pain Catastrophizing Scale; RUM, the PCS Rumination subscale; MAGN, the PCS Magnification subscale; HELP, the PCS Helplessness subscale; TSK, the Tampa Scale for Kinesiophobia; AA, the TSK Activity Avoidance subscale; SF, the TSK Somatic Focus subscale; PASS, Pain Anxiety Symptoms Scale; AV, the PASS Avoidance subscale; FT, the PASS Fear subscale; CA, the PASS Cognitive Anxiety subscale; PR, the PASS Physiological Responses subscale; S – Bχ^2 = Satorra-Bentler scaled chi-square statistic; CFI, comparative fit index; RMSEA, root-mean-square error of approximation. *p<0.05, ***p<0.001.



Model 5: S–Bχ^2 (61) = 238.44, p< 0.001, CFI = 0.93, RMSEA = 0.09.



Model 6: S–Bχ^2 (61) = 300.40, p< 0.001, CFI = 0.91, RMSEA = 0.10.

Figure 4: The "collapsed" models fitted for two pain adjustment outcomes. Numbers are standardized β coefficients. Negative emotion is indexed by NEO-N and PANAS-NA; NEO-N, neuroticism subscale of NEO; PANAS-NA, the negative affect subscale of PANAS; PCS, Pain Catastrophizing Scale; RUM, the PCS Rumination subscale; MAGN, the PCS Magnification subscale; HELP, the PCS Helplessness subscale; TSK, the Tampa Scale for Kinesiophobia; AA, the TSK Activity Avoidance subscale; SF, the TSK Somatic Focus subscale; PASS, Pain Anxiety Symptoms Scale; AV, the PASS Avoidance subscale; FT, the PASS Fear subscale; CA, the PASS Cognitive Anxiety subscale; PR, the PASS Physiological Responses subscale; S – Bχ^2 = Satorra-Bentler scaled chi-square statistic; CFI, comparative fit index; RMSEA, root-mean-square error of approximation. *p<0.05, ***p<0.001.