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Pain Catastrophizing in Patients with Non-Cardiac Chest Pain: Relationships with Pain, Anxiety, and Disability

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Abstract

Objective—This study examined the contributions of chest pain, anxiety, and pain catastrophizing to disability in 97 patients with non-cardiac chest pain (NCCP). We also tested whether chest pain and anxiety were indirectly related to greater disability via pain catastrophizing.

Methods—Participants completed daily diaries measuring chest pain for seven days prior to completing measures of pain catastrophizing, trait anxiety, and disability. Linear path model analyses examined the contributions of chest pain, trait anxiety, and catastrophizing to physical disability, psychosocial disability, and disability in work, home, and recreational activities.

Results—Path models accounted for a significant amount of the variability in disability scales ($R^2=.35$ to $.52$). Chest pain and anxiety accounted for 46% of the variance in pain catastrophizing. Both chest pain ($\beta=.18$, Sobel test $Z=2.58$, $p<.01$) and trait anxiety ($\beta=.14$, Sobel test $Z=2.11$, $p<.05$) demonstrated significant indirect relationships with physical disability via pain catastrophizing. Chest pain demonstrated a significant indirect relationship with psychosocial disability via pain catastrophizing ($\beta=.12$, Sobel test $Z=1.96$, $p=.05$). After controlling for the effects of chest pain and anxiety, pain catastrophizing was no longer related to disability in work, home, and recreational activities.

Conclusions—Chest pain and anxiety were directly related to greater disability and indirectly related to physical and psychosocial disability via pain catastrophizing. Efforts to improve functioning in NCCP patients should consider addressing pain catastrophizing.

Keywords

non-cardiac chest pain; catastrophizing; anxiety; disability

Chest pain is one of the most frequent complaints seen in primary medical care settings and annually, more than six million patients with chest pain are admitted to hospitals in the U.S. (1). Over 50% of patients presenting with chest pain do not have identifiable cardiac ischemia or other major physical disorders that account for their symptoms (2–5). Chest pain with no evidence of underlying cardiac disease is known as non-cardiac chest pain (NCCP; 6). Despite having a good medical prognosis (7,8), many NCCP patients experience significant limitations

in activity, reductions in quality of life, increased psychosocial disability, occupational impairment, and high levels of medical utilization related to chest pain (8–14). Following medical evaluations with negative or normal cardiac test results, many patients continue to experience chest pain and significant impairments in daily functioning (9,15). Two studies followed NCCP patients for five years and found that symptoms, impaired functioning, and over utilization of medical care persisted in the majority of patients (16,17).

Studies suggest that psychiatric disorders, particularly anxiety disorders, are prevalent in patients with NCCP (18–22), and many NCCP patients who do not meet criteria for a psychiatric diagnosis experience subclinical symptoms that contribute to impairment (3). The rates of current and lifetime anxiety disorders in NCCP patients are higher than reported rates in patients with established coronary heart disease (23). A recent study using structured diagnostic interviews reported that 75% of NCCP patients met criteria for a current anxiety disorder or a subclinical anxiety disorder, and 55% met criteria for a lifetime history of an anxiety disorder (12). In contrast, a recent study among patients with established coronary heart disease reported that 36% met criteria for a current anxiety disorder and 45% met criteria for a lifetime history of an anxiety disorder (23). Other studies in patients with established coronary heart disease have focused on specific anxiety disorders such as panic disorder (estimates 0–29%), social phobia (estimates 5–21%), and specific phobia (estimates 1–15%) (24–27).

Data show that NCCP patients with an anxiety disorder have more severe and frequent chest pain, more life interference due to pain, more affective distress related to pain, and higher rates of healthcare utilization compared to patients with no psychiatric diagnosis (12). In NCCP patients, anxiety has been related to bodily monitoring which can perpetuate worry about illness (14), disease-related fear (28), poorer coping strategies (17) and lower levels of reported emotional support (14). Moreover, patients who are anxious about their unexplained chest pain often stay active in the healthcare system resulting in more substantial economic burden (29). There is limited information about the relationship between the presence of an anxiety disorder and healthcare use in patients with known cardiac disease.

Several etiological models have been proposed to explain the processes involved in the initiation and maintenance of NCCP, and the persisting functional impairment found in NCCP patients (3,30,31). These models suggest that individuals with NCCP misinterpret minor physical symptoms as signs of serious disease causing increased pain, anxiety, and disability, which maintain the problem in a vicious cycle (3). Psychological problems, particularly severe anxiety and panic attacks, are viewed as important precipitating factors for the development and maintenance of NCCP. Cognitive factors such as catastrophic thinking, negative appraisals, and selective attention play a central role in all of the etiological models. Cognitions are thought to affect patients' emotional and behavioral responses to physical symptoms leading to avoidance of activities, social withdrawal, disruptions in work, and physical and psychosocial disability (3,30,31).

One important cognition to consider in this population is pain catastrophizing, which is the tendency to exaggerate the threat value of pain sensations, ruminate upon them, and feel helpless when experiencing pain (32). NCCP patients are more likely to report catastrophizing thoughts when faced with pain compared to other patients (33), and reducing catastrophic thoughts about the medical causes of chest pain has been associated with decreased chest pain in NCCP patients (34). It is assumed that pain, anxiety, and catastrophizing thoughts contribute to a vicious cycle that ultimately leads to disability in NCCP patients (3). While pain catastrophizing may be a common mechanism through which both pain and anxiety lead to disability, formal tests of these relationships have not been conducted in NCCP patients. Given the prominent role of anxiety in NCCP, it is unclear whether pain catastrophizing will demonstrate strong relationships with disability after accounting for the impact of anxiety.

Further, it is not known if the effects of pain catastrophizing differ across domains of functioning (e.g., physical disability vs. psychosocial disability).

Understanding pain catastrophizing's contribution to disability across different domains of functioning is important for developing and improving treatments for NCCP patients. For example, if pain catastrophizing is found to significantly contribute to psychosocial disability, we would expect that intervention strategies (e.g., cognitive restructuring) specifically designed to target catastrophic thoughts would be needed to yield benefits in psychosocial functioning. In this case, providing patients with medication alone or strategies that target other behavioral factors (e.g., avoidance of physical activity) may not result in improved psychosocial functioning. It is also possible that pain catastrophizing may no longer contribute to a particular domain of disability after accounting for anxiety. In this case, strategies to reduce anxiety would be needed to yield benefits. A better understanding of the relationships between pain catastrophizing and particular domains of functioning would help clinicians develop more targeted and efficient interventions (35).

This study examined the contributions of chest pain, trait anxiety, and pain catastrophizing to disability in a sample of patients with NCCP. Linear path model analyses were conducted to examine these relationships across multiple domains of disability including physical disability, psychosocial disability, and disability in work, home, and recreational activities. We tested whether chest pain and trait anxiety were indirectly related to disability via pain catastrophizing. Because these data are correlational, the direction of the effects we tested was assumed. Separate path model analyses were conducted for each area of disability to examine whether pain catastrophizing played a more central role in some areas of disability than others.

Methods

Participants and Procedure

The present study includes patients diagnosed with non-cardiac chest pain who completed a baseline evaluation prior to randomization in a treatment outcome study testing the efficacy of pain coping skills training and medication (Sertraline). All study procedures and materials were approved by and in compliance with the Duke University Medical Center institutional review board. Participants were recruited from Duke University Medical Center and satellite clinics from February 2002 to October 2005. Eligibility criteria for study entry were a) presented for medical care with complaints of chest pain in the previous six months, b) received a negative stress test, normal coronary angiogram, or had a survival probability $\geq 98\%$ at 2 years (calculated from a prognostogram developed by statistical modeling from the Duke Cardiovascular Database) (36), c) a low likelihood of significant coronary artery disease ($<25\%$) on the National Cholesterol Education Program's (NCEP) modification of the Framingham Risk Calculator (FRC), and d) age 18–85 years. Exclusion criteria included other cardiac problems associated with chest pain, current use of psychotropic medications or medications significantly affecting pain, a history of drug or alcohol abuse within the past six months, pregnancy, or severe psychopathology (i.e., suicidal patients, severe depression, psychosis). All patients were screened for psychopathology using the Diagnostic Interview Schedule (37).

After providing informed consent, participants completed daily diaries measuring pain intensity and pain unpleasantness for seven days prior to a baseline in-person evaluation. The in-person evaluation included self-report measures of pain catastrophizing, anxiety, disability, and a demographic questionnaire. Eighteen participants were excluded from the present analyses because they did not complete daily diaries measuring pain, resulting in a sample size of 97. There were no statistically significant differences on demographic characteristics, pain

catastrophizing, anxiety, or disability between participants ($n=18$) without diary data and those ($n=97$) with diary data (all p values $> .20$).

Participants were 97 patients with NCCP. The sample included 34 (35%) men and 63 (65%) women. Participants ranged in age from 23 to 74 years ($M=48$, $SD=12$) and average education was 14 years ($SD=3$, range 6 to 23). Forty-nine (51%) participants were white, 44 (45%) participants were African American, one (1%) participant was Pacific Islander, and three (3%) participants self-identified as multiracial. Approximately half (54%) of the sample was married and 61% reported working in the past 12 months. Participants completed the evaluation for this study an average of 2 months ($SD=1.77$, range=0 to 6) after presenting for medical care for chest pain. Forty percent of these patients had a documented history of chronic noncardiac chest pain and had completed prior medical evaluations for chest pain.

Measures

Chest pain—Chest pain scores were derived from pain diaries completed over seven days prior to the baseline in-person evaluation. Use of daily pain ratings minimizes retrospective error by assessing pain experiences close to the real-time occurrence of pain (38,39). Visual analog scales were used to assess daily chest pain intensity and unpleasantness. Visual analog scales have been shown to be reliable and valid methods of assessing pain (39) and have been found to be sensitive to changes in pain (40–43). Participants rated chest pain intensity on a scale ranging from 0 (*no pain*) to 100 (*pain as bad as it can be*) and pain unpleasantness on a scale ranging from 0 (*not at all unpleasant*) to 100 (*unpleasant as possible*). Participants were instructed to complete pain ratings before retiring each night. Participants who completed four or more days of pain diaries were included in the study. Rates of diary completion were as follows: four days $n=4$; five days $n=5$, six days $n=11$, and seven days $n=77$. Participants ($n=7$) who reported no pain episodes and rated pain intensity and unpleasantness as zero on all days were given scores of zero for average intensity and unpleasantness. Chest pain intensity and pain unpleasantness ratings were highly correlated ($r=.95$) in this sample. Thus, chest pain intensity and unpleasantness ratings for the week were averaged to create a single chest pain score for each participant.

Trait Anxiety—Anxiety was assessed during the in-person evaluation using the Trait Anxiety scale of State-Trait Anxiety Inventory (STAI; 44). While state anxiety is defined as an acute response to a threatening or challenging situation, trait anxiety is defined as a stable and enduring tendency to be anxious. The STAI Trait Anxiety scale is a 20 item self-report inventory. Each item is rated on a four-point scale (1=*almost never*, 4=*almost always*). Items are summed to create a total Trait Anxiety score with higher scores indicating greater anxiety. The STAI, developed as a tool for investigating anxiety in normal adult populations, has been used to assess anxiety in neuropsychiatric, medical, and surgical patients (45,46). The Trait Anxiety scale has high test-retest reliability ranging from .73 to .85 and alpha coefficients range from .83 to .92 indicating good internal consistency (44). Internal consistency in this sample was high ($\alpha = .91$).

Pain Catastrophizing—The 13-item Pain Catastrophizing Scale (PCS; 47) was used to assess the degree to which participants experienced thoughts or feelings of helplessness (6 items; e.g., “It’s awful and I feel that it overwhelms me.”), magnification (3 items; e.g., “I wonder whether something serious will happen.”), and rumination (4 items; e.g., “I keep thinking about how much it hurts.”) during pain. Each item is rated on a 5-point scale (0=*not at all* to 4=*always*). Items were summed to create a total PCS score. The PCS total score was used because the helplessness, magnification, and rumination domains were highly correlated ($r=.70$ to $.81$). The PCS total score has high internal consistency (48) and has been found to contribute to variability in pain and pain-related disability ratings even after controlling for

measures of anxiety, fear of pain, negative affect, and neuroticism (32). The PCS total score demonstrated high internal consistency in the current sample (Cronbach's alpha = .94).

Disability—The Sickness Impact Profile (SIP; 49) was used to measure disability. The SIP is a widely used measure of disability that indicates changes or limitations in a person's behavior due to illness (49). The SIP has been broadly tested, has demonstrated high internal consistency, and has been found to be sensitive to change in a wide range of patients including patients with persistent pain, cardiovascular disease, chronic illness, musculoskeletal injury, and sleep problems (48,50–57). The SIP includes 136 behaviorally-based, health status items that assess everyday activities. Participants are asked to endorse (yes vs. no) items that describe their health. Each item is weighted depending on the severity of dysfunction. Scores for each of the disability scales are summed and expressed as a percentage of the maximum score possible with higher scores representing greater dysfunction. For the current study, physical disability, psychosocial disability, activity scales were used. The physical disability scale includes 45 items from the body care and movement, mobility, and ambulation categories (alpha = .91). The psychosocial disability scale includes 48 items from the emotional behavior, social interaction, alertness behavior, and communication categories (alpha = .92). To measure disability in activities, we computed a work, home, and recreational activity scale using 27 items from these three SIP categories (alpha = .80).

Data Analysis

Descriptive statistics and intercorrelations are provided for chest pain, trait anxiety, pain catastrophizing, and disability scales. Linear path model analysis, which provides simultaneous path estimation, was conducted to examine the direct and indirect relationships between study variables. We were specifically interested in testing whether chest pain and trait anxiety were indirectly related to measures of disability via pain catastrophizing. It should be noted that because the data in this study were cross-sectional, the direction of effects (i.e., indirect, direct) is assumed, but cannot be tested. Separate linear path models were estimated for each of the three disability scales. Figure 1 displays the model tested. The Sobel test (58) was used to test the significance of indirect effects. Because assumptions of the Sobel test are often violated in small samples, we also examined the significance of indirect effects using a bootstrap approach (with 5000 resamples) to obtain 95% confidence intervals for the direct and indirect effects (59,60). Power was computed for each observed model parameter using the Monte Carlo study approach recommended by Muthén and Muthén (61) and was carried out using Mplus 5.1 (three seeds with 10,000 replications were used for each analysis to ensure that stability was reached). A two-tailed alpha of .05 was used for the power analysis. Each model included age, education, gender (0=male, 1=female), and race (0=White, 1=African American, Pacific Islander, or multiracial) as control variables because these variables were related ($p < .10$) to disability, anxiety, or chest pain in bivariate analysis (Pearson and point-biserial correlations). Mplus 5.1 (62) was used to calculate maximum likelihood estimates for model parameters. Because the models were saturated, indices of model fit were not examined. Instead, we provide the total R^2 for disability in each model.

Prior to conducting path model analysis, data were evaluated for normality and outliers. Variables with non normal distributions were identified including chest pain, physical disability, and psychosocial disability scales (skew > 1.5). We reran the models after transforming these variables. Both natural log and rank order transformations were examined. Model results were similar when including transformed variables. Because interpretation of outcomes is more difficult with transformed variables, results from models with original variables are presented.

Results

Descriptive statistics

Means, standard deviations, ranges, and correlations (Pearson correlations) of study variables are displayed in Table 1. Correlations between all variables were statistically significant ($p < .05$) and in the expected direction. Less formal education was associated with greater chest pain ($r = -.34, p = .01$), higher pain catastrophizing ($r = .32, p = .01$), and greater disability ($r = -.23$ to $-.36, p$ values $< .05$). Women reported higher trait anxiety ($r = .21, p = .04$), greater physical disability ($r = .24, p = .02$), and greater psychosocial disability ($r = .26, p = .01$). White participants reported less disability on physical, psychosocial, and activity scales ($r = -.21$ to $-.30, p$ values $< .05$). Younger age was associated with higher trait anxiety ($r = -.20, p = .05$). The amount of time (number of months) that elapsed between presenting for medical care due to chest pain and the study evaluation was not associated with any of the study variables (p values $> .20$). Education, gender, race, and age were included in linear path models as control variables.

Linear path model analysis

Physical disability—Table 2 displays standardized beta coefficients and indirect effects for the linear path model analyses.¹ Chest pain ($\beta = .49, p < .001$) and trait anxiety ($\beta = .38, p < .001$) were statistically significantly associated with pain catastrophizing, and chest pain and trait anxiety accounted for 46% of the variance in pain catastrophizing. Pain catastrophizing showed a positive association with physical disability ($\beta = .36, p = .01$). There was a direct relationship between chest pain and physical disability ($\beta = .27, p = .04$), which indicated that chest pain was associated with physical disability after controlling for trait anxiety and pain catastrophizing. The direct relationship between trait anxiety and physical disability was not significant. Both chest pain (indirect effect $\beta = .18$, Sobel test $Z = 2.58, p = .01$) and trait anxiety (indirect effect $\beta = .14$, Sobel test $Z = 2.11, p = .05$) demonstrated indirect relationships with physical disability via pain catastrophizing. The indirect relationship between chest pain and physical disability accounted for 40% of chest pain's total effect (direct + indirect relationship) on physical disability. The indirect relationship between trait anxiety and physical disability accounted for 52% of trait anxiety's total effect on physical disability. The path model accounted for 49% of the variability in physical disability.

Psychosocial Disability—Again, both chest pain and trait anxiety were statistically significantly associated with pain catastrophizing (p values $< .001$). As shown in Table 2, pain catastrophizing was associated with psychosocial disability ($\beta = .25, p = .04$). While the direct relationship between chest pain and psychosocial disability ($\beta = .21, p = .06$) was moderate in magnitude (.63), it was not statistically significant. Trait anxiety showed a direct relationship with psychosocial disability ($\beta = .33, p = .001$), which indicated that trait anxiety was associated with psychosocial disability after controlling for chest pain and pain catastrophizing. Chest pain demonstrated an indirect relationship with psychosocial disability via pain catastrophizing (indirect effect $\beta = .12$, Sobel test $Z = 1.96, p = .05$). The indirect relationship between chest pain and psychosocial disability accounted for 36% of chest pain's total effect (direct + indirect relationship) on psychosocial disability. The indirect relationship between trait anxiety and psychosocial disability was not significant. The path model accounted for 52% of the variability in psychosocial disability.

¹We also conducted a set of analyses that included the STAI state anxiety scale in place of the trait anxiety scale. The linear path models showed a similar pattern of results. However, the magnitude of the path coefficients between state anxiety and the disability scales were lower than the path coefficients found for trait anxiety. In this sample, the correlation between the state anxiety and trait anxiety scales was $r = .68$.

Work, home, and recreational activities—As shown in Table 2, chest pain and trait anxiety were statistically significantly associated with pain catastrophizing (p values $< .001$). Pain catastrophizing was not associated with disability in work, home, and recreational activities. Chest pain ($\beta=.28, p=.04$) and trait anxiety ($\beta=.33, p=.001$) demonstrated a direct relationships with disability in work, home, and recreational activities. There were no indirect relationships with work, home, and recreational activities via pain catastrophizing. The path model accounted for 35% of the variability in activities.

Discussion

Etiological models of NCCP suggest that chest pain, anxiety, and catastrophizing contribute to a vicious cycle that results in disability and impaired functioning (3). This study used linear path model analyses to formally test these relationships across three domains of functioning and important findings emerged. First, chest pain and anxiety were indirectly associated with physical disability via pain catastrophizing, and chest pain was indirectly related to psychosocial disability via pain catastrophizing. These findings suggest that pain catastrophizing played an important role in physical and psychosocial functioning. Second, pain catastrophizing did not contribute to disability in work, home, and recreational activities. Instead, chest pain and anxiety were directly associated with disability in these activities suggesting that other factors (e.g., avoidance coping, other anxiety-related cognitive distortions, selective attention) may contribute to disability in these domains.

Cognitive-behavioral treatments (CBT) for NCCP have shown benefits for reducing chest pain and disability (64–67). These intervention protocols include multiple strategies such as breathing training, relaxation training, behavioral experiments, stress and anxiety management, and cognitive restructuring. A central goal of these CBT interventions is to correct misattributions regarding physical symptoms and reduce catastrophic thoughts about chest pain (31,35). To date, studies have not examined which CBT intervention strategies are responsible for reductions in pain and disability in NCCP patients. Identifying the active ingredients or mechanisms of change in CBT interventions for NCCP patients represents an important next step in this area of research (68). Our data suggest that intervention studies testing for mechanisms of change should focus on catastrophic thoughts about pain such as exaggerating the threat value of pain sensations, ruminating about pain, and helplessness when experiencing pain. In recent intervention studies in patients with chronic pain (e.g., low back pain, temporomandibular disorder pain), reductions in pain catastrophizing have been linked with improvements in physical functioning and psychological adjustment (69–71). Additional intervention studies are needed in NCCP patients to determine whether reductions in pain catastrophizing lead to reductions in physical and psychosocial disability.

In this study, pain catastrophizing was not associated with disability in work, home, and recreational activities. Other behavioral and biological factors that may contribute to disability in these activities warrant further study. The cardiophobia literature suggests that individuals who are fearful of cardiac sensations use behavioral avoidance coping in response to activities believed to elicit uncomfortable physiological symptoms (72). Increased awareness of cardiac sensations and behaviors designed to avoid cardiac exertion may contribute to disability in work, home, and recreational activities (73). The fear-avoidance model proposed by Vlaeyen and colleagues (74) may be relevant for understanding the processes that lead to increased disability in NCCP patients. This model suggests that misattributions and catastrophic thoughts about pain initiate a vicious cycle that leads to pain-related fear, avoidance of activities, and ultimately greater disability. Vlaeyen and colleagues (74,75) have developed in vivo graded exposure techniques that enable patients with pain to experience and habituate to activities they might normally avoid. Graded exposure might be a useful method for reducing disability in work, home, and recreational activities in NCCP patients because it requires patients to engage

in and accomplish a feared or painful activity. In a psychological group intervention for NCCP patients, Potts and colleagues (66) included graded exposure to activities that were avoided because of pain and light physical exercise. This intervention resulted in improvements in disability and exercise tolerance.

While trait anxiety was indirectly related to physical disability via pain catastrophizing, it was directly related to psychosocial disability and disability in work, home, and recreational activities. Additional research is needed to better understand how anxiety contributes to disability in NCCP patients. Because NCCP patients have a tendency to monitor bodily sensations (76), physiologic arousal associated with anxiety may play an especially important role in functioning for these patients. Autonomic and hormonal responses associated with anxiety can influence esophageal and cardiac functioning, and increased tension in thoracic muscles elicited by anxiety can contribute to chest pain and altered breathing patterns (77–80). Most CBT treatments for NCCP patients include strategies (e.g., breathing and relaxation training) for reducing anxiety-related physiologic arousal (64–67). Additional intervention studies with NCCP patients are needed to test whether strategies for reducing anxiety-related physiologic arousal lead to reductions in physical symptoms and improved functioning.

NCCP patients in this study completed their study evaluation an average of 2 months (range=0 to 6) after presenting for medical care for chest pain. Many (40%) of these patients had a documented history of chronic noncardiac chest pain and had completed prior medical evaluations for chest pain. Because of the cross-sectional nature of this study and similar methodological limitations in prior studies, we can only say that pain, anxiety, and catastrophizing are associated with disability. How these relationships develop following an initial episode of chest pain and how these relationships unfold over time is not evident from these data. While many NCCP patients continue to experience chest pain after medical evaluation, some patients do not limit their activities or experience increased levels of disability. Longitudinal studies that capture NCCP patients at the time of their initial medical evaluation for chest pain could provide valuable information about the processes that contribute to disability and how these processes evolve over time.

Characteristics of this study offered several advantages, including a racially diverse sample and daily diary assessments of pain which are less vulnerable to retrospective reporting. We also note limiting characteristics of this study. First, this study used a correlational design, which prevents us from making causal attributions about the relationships between chest pain, anxiety, pain catastrophizing, and disability. The correlational study design makes it impossible to determine whether pain or anxiety influenced disability through catastrophizing or vice versa. Future research is needed to more closely evaluate potential cause-effect relationships. For example, studies could be conducted to examine whether interventions that alter anxiety lead to significant changes in pain catastrophizing and whether alterations in pain catastrophizing affect levels of disability. Second, our findings may be less generalizable because data were obtained from patients who agreed to participate in a treatment study. Individuals who voluntarily seek participation in clinical trials may differ from patients in the general population. The findings obtained may not generalize to the larger population of patients with NCCP seen in clinical settings. Finally, the sample size precluded the use of structural equation models or a more complicated model that simultaneously examined all three domains of disability. While this study included well-validated measures, the use of manifest variables in the analyses may have increased bias due to measurement error (81). Future studies should consider using multiple measures or parcels to create latent variables for each of the constructs. Additionally, when interpreting the results of this study, it is important to consider that our use of separate models resulted in an increased risk of type I error. Given the family-wise type I error rate in this study, we suggest that readers focus on the effect sizes of the path coefficients rather than statistical significance.

NCCP is associated with substantial financial and personal costs for patients. Patients having NCCP continue to incur high costs over the long-term due to additional medical evaluations, hospitalizations, and taking inappropriately prescribed medications (1,11). The personal costs of NCCP are also high including occupational impairment, significant limitations in activity, and reductions in quality of life (8–12). Effective treatments are needed to reduce the burden of NCCP. Cognitive-behavioral treatments show promise for reducing the frequency and intensity of chest pain and the disability associated with NCCP (30,31,35). The findings of this study suggest that structuring psychosocial treatment efforts for NCCP patients so as to more directly address pain catastrophizing may yield benefits for physical and psychosocial functioning. Additional research is needed to replicate these findings and to explore other biopsychosocial factors that may contribute to the persistent functional impairment seen in many patients with NCCP.

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Abbreviations

NCCP, non-cardiac chest pain.

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Figure 1.

Linear path model testing the direct and indirect relationships between chest pain and disability and trait anxiety and disability.

Note: Separate linear path model analyses were conducted for each disability scale. Analyses included gender, age, education, and race as control variables.

Table 1

Descriptive statistics and intercorrelations (N=97).

	Mean	SD	Range	1.	2.	3.	4.	5.	6.
1. Chest pain	16.73	9.14	0 – 100	1.00					
2. Trait anxiety	38.47	9.84	20 – 69	.20	1.00				
3. Pain catastrophizing	14.35	11.61	0 – 52	.56	.48	1.00			
4. Physical disability	5.07	9.29	0 – 42	.55	.39	.62	1.00		
5. Psychosocial disability	10.46	13.72	0 – 73	.48	.54	.60	.83	1.00	
6. Work, home, and recreational activities	15.30	16.51	0 – 73	.44	.45	.45	.70	.67	1.00

Note: All correlations significant at $p < .05$.

Table 2
Standardized path coefficients and indirect effects for linear path model analyses (N=97).

	Path Coefficient	95% Confidence Interval	Sobel test Z of the indirect effect	Proportion of total effect	Power for observed effects
Physical Disability (Total $R^2 = .49$)					
Chest pain → pain catastrophizing	.49*	.29, .70			.99
Trait anxiety → pain catastrophizing	.38*	.22, .54			.99
Pain catastrophizing → physical disability	.36*	.10, .62			.95
Chest pain → physical disability	.27*	.01, .54			.85
Trait anxiety → physical disability	.13	-.10, .36			.34
Chest pain → pain catastrophizing → physical disability	.18*	.04, .31	2.58*	40%	.94
Trait anxiety → pain catastrophizing → physical disability	.14*	.01, .27	2.11*	52%	.93
Psychosocial disability (Total $R^2 = .52$)					
Chest pain → pain catastrophizing	.49*	.29, .70			.99
Trait anxiety → pain catastrophizing	.38*	.22, .54			.99
Pain catastrophizing → psychosocial disability	.25*	.01, .49			.76
Chest pain → psychosocial disability	.21	-.01, .43			.69
Trait anxiety → psychosocial disability	.33*	.14, .53			.97
Chest pain → pain catastrophizing → psychosocial disability	.12*	.001, .25	1.96*	36%	.72
Trait anxiety → pain catastrophizing → psychosocial disability	.10	-.01, .20	1.83	23%	.69
Work, home, and recreational activities (Total $R^2 = .35$)					
Chest pain → pain catastrophizing	.49*	.29, .70			.99
Trait anxiety → pain catastrophizing	.38*	.22, .54			.99
Pain catastrophizing → activities	.10	-.11, .31			.17
Chest pain → activities	.28*	.01, .54			.77
Trait anxiety → activities	.33*	.14, .52			.91
Chest pain → pain catastrophizing → activities	.05	-.06, .16	0.91	15%	.15
Trait anxiety → pain catastrophizing → activities	.04	-.05, .13	0.87	11%	.13

Path Coefficient	95% Confidence Interval	Sobel test Z of the indirect effect	Proportion of total effect	Power for observed effects
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Note: Models controlled for age, gender, education, and race.

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 $p \leq .05$