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The Role of Disease Conviction: Exploring Its Effects on Chest Pain and Anxiety-Related Models of Non-cardiac Chest Pain

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Abstract

The aim of this study was to investigate the role of disease conviction in the chest pain and life interference of patients with non-cardiac chest pain (NCCP), after controlling for anxiety sensitivity and body vigilance. While all three psychological constructs are theoretically implicated and empirically associated with the experience of NCCP, no research has examined the influence of disease conviction in the context of other relevant constructs. The sample included 229 participants with NCCP who were recruited after a medical evaluation failed to elicit an organic explanation for their chest pain. Hierarchical regression analyses revealed that while anxiety sensitivity significantly predicted chest pain severity and interference, only body vigilance contributed significant additional variance to chest pain severity, and only disease conviction contributed significant additional variance to chest pain interference. While anxiety sensitivity, body vigilance, and disease conviction all appear to affect those with NCCP, it seems that their impact is manifest in different domains (i.e., pain perception vs. psychosocial impairment).

Keywords Non-cardiac chest pain · Health anxiety · Anxiety sensitivity · Disease conviction

Introduction

Chest pain can be one of the most frightening medical complaints due to its association with myocardial infarction and coronary artery disease, one of the leading causes of death worldwide (Finegold, Asaria, & Francis, 2012). Among the 6 million Americans who annually require urgent medical care due to chest pain suggestive of myocardial infarction (American Heart Association, 2006), as many as 75% are left without an organic explanation for their chest pain (Kroenke & Mangelsdorff, 1989; Pope, Ruthazer, Beshansky, Griffith, & Selker, 1998). Despite being informed that their symptoms are not medically significant, a substantial portion of these patients continue to report chest pain, anxiety surrounding their symptoms, as well as disability not unlike patients diagnosed with coronary artery disease (Chambers & Bass, 1990; Eifert, Hodson, Tracey, Seville, & Gunawardane, 1996). For those individuals who experience recurrent angina-like pain in the absence of identifiable organic etiology, a diagnosis of non-coronary artery disease chest pain, more commonly referred to as non-cardiac chest pain (NCCP), may be assigned (Eslick, Coulshed, & Talley, 2002; Fleet & Beitman, 1997).

NCCP is estimated to occur in 23–33% of the general population, and for some, the condition is disabling (Eslick, Jones, & Talley, 2003; Lampe et al., 1998; Locke, Talley, Fett, Zinsmeister, & Melton, 1997). Patients often report impaired quality of life (Eslick et al. 2003; Hadlandsmyth, White, & Krone, 2013; Wong et al., 2002), and show mortality rates comparable to those with cardiac chest pain (Eslick & Talley, 2008). Despite the urgency of these findings, our understanding of NCCP has been limited by its classification as a "diagnosis" of exclusion (White, 2007), which by nature results in complex and varied presentations (Fass & Eslick, 2007). This picture is further complicated by the fact that physical abnormalities are found within the NCCP population (Richter, Beitman, & Cannon, 1991) and not all patients in the NCCP literature have received a coronary angiography (Hadlandsmyth et al., 2013), the gold standard procedure in assessing for cardiac etiologies (Noto et al., 1991).

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Despite these factors, the pain experienced by the majority of NCCP patients does not always seem to have a distinct physical cause (Richter et al., 1991). Without a satisfactory medical explanation, researchers have begun to examine the influence of psychological phenomena on the experience of NCCP (Beitman et al., 1987; Eifert, 1991), with particular respect to anxiety and panic (Fleet & Beitman, 1997).

A number of theoretical models for NCCP have been developed conceptualizing the syndrome within a biopsychosocial framework (Eifert, Zvolensky, & Lejuez, 2000; Mayou, 1998; White & Raffa, 2004). These models assert that the etiology of NCCP is multi-causal and interactive, and common features include affective components (e.g., worry, rumination) that are basic to our theoretical conceptualizations of anxiety disorders. For instance, cognitive misappraisal of benign cardiac sensations is considered central to the development and maintenance of NCCP (Aikens, Zvolensky, & Eifert, 2001; Bradley, Scarinci, & Richter, 1991; Mayou, 1998; Schroeder, Gerlach, Achenbach, & Martin, 2015). Research into the underlying psychological constructs that modulate cognitive misappraisal have strengthened the connection between models of anxiety and NCCP. Three such constructs include anxiety sensitivity (AS), the trait tendency to fear physiological symptoms associated with anxiety due to the belief that those sensations are signs that something harmful is occurring, or is about to occur (McNally, 2002; Reiss, 1991; Reiss, Peterson, Gursky, & McNally, 1986), body vigilance (BV), the tendency to consciously attend to and monitor for internal sensations (Schmidt, Lerew, & Trakowski, 1997), and disease conviction (DC), the belief that one has a medical illness that persists despite appropriate medical evaluation and positive reassurance (Olatunji, Deacon, & Abramowitz, 2009). Each of these constructs has been extensively researched in the context of anxiety pathology and, due to their proposed theoretical connection to NCCP, has recently been examined in the NCCP population.

Conceptually, models of hypochondriasis (HC) have been proposed as potentially useful in elucidating our understanding of NCCP, in part because of the hypothesized importance of disease conviction. HC, now categorized as Somatic Symptoms Disorder in DSM-V, is characterized by cognitive misinterpretations of benign physical sensations as evidence of a serious medical illness (Kellner, 1986). Disease conviction is conceptualized as an underlying dimension of HC distinct from the fear of having a medical illness (Barsky, 1992), and early models of NCCP have suggested that disease conviction is relevant to the experience of NCCP (Eifert et al., 1996). In fact, Mayou's (1998) model of NCCP posits that appraising minor physical sensations as evidence of a serious medical illness is an essential feature of the disorder (Mayou, 1998). Furthermore, NCCP patients have been shown to report higher levels of disease conviction than

surgical inpatients with diagnosed cardiovascular disease (Eifert et al., 1996).

However, anxiety sensitivity and body vigilance, rather than disease conviction, have been of particular interest to NCCP researchers because of their close connection to panic disorder (PD). Panic Disorder has similar symptomatology to NCCP (Fleet & Beitman, 1997; White & Raffa, 2004), and the two disorders are highly comorbid. It has been established that NCCP patients show elevated levels of PD compared to the general population, with estimated rates anywhere from 12 to 40% (Beitman et al., 1989; Eifert et al., 1996; White et al., 2008). Hence, evidence-based models of PD have substantially contributed to our current conceptualizations of NCCP (White & Barlow, 2002; White & Raffa, 2004), and treatments derived from this model have been implemented in emergency settings for NCCP patients (Esler et al., 2003; Tyrer et al., 2017).

Substantial evidence that anxiety sensitivity (Maller & Reiss, 1992; Plehn & Peterson, 2002; Schmidt, Lerew, & Jackson, 1999) and body vigilance (Schmidt, Lerew, & Trakowski, 1997; Schmidt & Trakowski, 1999) contribute to the pathogenesis of Panic Disorder has led to these constructs being investigated in the context of NCCP. For instance, fear of cardiac-related sensations has been associated with the chest pain reported by NCCP patients (Aikens et al., 1999; Eifert et al., 1996; Lipsitz et al., 2004), and anxiety sensitivity has been associated with both chest pain severity and interference caused by chest pain (White, McDonnell, Gervino, 2011). The same investigation also found that NCCP patients are indeed hypervigilant to cardiac-related sensations (White, Craft, & Gervino, 2010), and that there is a direct relationship between body vigilance and the interference caused by chest pain, which may be partially mediated by fear of interoceptive sensations. Taken together, the importance of anxiety sensitivity and body vigilance to the experience of NCCP illustrates the relevance of PD models in describing NCCP symptomatology.

However, given that PD frames our current conceptualizations of NCCP, research has focused less on models of hypochondriasis as they pertain to NCCP. There are important similarities as well as distinctions between PD and HC in this context. While both PD and HC patients experience intense somatic sensations for which they assign an organic explanation (e.g., heart attack), PD is marked by discrete episodes of catastrophization, while HC is characterized by long-standing or persistent somatic complaints (Hiller, Leibbrand, Rief, & Fichter, 2005). Although this is a crucial difference, anxiety sensitivity and body vigilance are constructs also implicated in the development and maintenance of HC (Fergus & Valentiner, 2010). Anxiety sensitivity has been shown to be elevated in individuals with hypochondriacal concerns (Abramowitz, Deascon, & Valentiner, 2007; Deacon & Abramowitz, 2006b; Otto, Demopulos, McLean,

Pollack, & Fava, 1998; Otto, Pollack, Sachs, & Rosenbaum, 1992), and levels of anxiety sensitivity are comparable across patients with hypochondriasis and patients with panic disorder (Deacon & Abramowitz, 2008). Additionally, body vigilance has been shown to be elevated in hypochondriacal populations and at comparable levels to those found in panic disorder (Deacon & Abramowitz, 2008; Olatunji, Deacon, Abramowitz, & Valentiner, 2007). However, disease conviction, which is specific to HC, has not been examined as extensively as the vulnerabilities that are common to both hypochondriasis and panic disorder. Determining the relative contribution of disease conviction to the experience of chest pain when compared to anxiety sensitivity and body vigilance is important in distinguishing whether NCCP is best conceptualized in the context of PD or HC. Understanding which underlying mechanisms contribute more substantially to NCCP symptomatology has a notable impact on the specific interventions that might be most helpful. To date, no research has examined whether disease conviction contributes to the phenomenology of NCCP, beyond what is predicted by anxiety sensitivity and body vigilance.

The purpose of this study was to investigate whether disease conviction provides unique variance to the pain severity and interference experienced by those with NCCP, after examining anxiety sensitivity and body vigilance. We predicted that there would be a significant positive correlation between anxiety sensitivity and chest pain severity as well as chest pain interference. We also expected there to be a significant positive correlation between body vigilance and chest pain severity and interference. We expected to find a significant positive correlation between disease conviction and chest pain severity and interference. Lastly, we predicted that disease conviction will be a significant predictor of chest pain severity as well as interference, after accounting for anxiety sensitivity and body vigilance.

Method

Participants

Participants were 229 patients taking part in a larger prospective cohort study examining the clinical course and correlates of NCCP. Patients seeking evaluation in the cardiology department of a large, university-affiliated medical center were eligible to participate if they presented with a chief complaint of chest pain or discomfort, completed a cardiac evaluation (i.e., general physical exam, exercise tolerance test) with results indicating no abnormalities, were at least 18 years of age, and were fluent in the English language. Exclusion criteria included having a current or lifetime cardiac diagnosis (e.g., congenital heart disease, myocardial infarction), or current or recent (last 6 months)

untreated medical or psychiatric illness (e.g., active psychosis, manic episode).

Measures

Anxiety Sensitivity Index (ASI)

The ASI is a self-report measure used to evaluate anxiety focused on panic-related bodily sensations, including beliefs about the dangerousness of physical anxiety symptoms (Reiss, 1980; Reiss, Peterson, Gursky, & McNally, 1986). The ASI consists of 16 items each on a 4-point Likert scale (0 = Very Little, 4 = Very Much). Typical items include "When I feel pain in my chest, I worry that I'm going to have a heart attack" and "When my throat feels tight, I worry that I could choke to death." Scores for each item are summed to produce a total score ranging from 0 to 64, with greater anxiety sensitivity being indicated by higher total scores. The scale comprises three lower-order factors (i.e., physical, cognitive, and social concerns) and one higher-order factor being anxiety sensitivity (Zinbarg, Brown, Barlow, & Rapee, 2001). The ASI has demonstrated good internal consistency ($r = .82$–$.91$) and convergent validity (Peterson & Reiss, 1992; Zinbarg, Brown, Barlow, & Rapee, 2001). Cronbach's alpha in this study was 0.90.

Body Vigilance Scale (BVS)

The BVS is a self-report measure of attention and sensitivity to interoceptive sensations. It has a total of 19 items each on a 10-point Likert scale. Four items assess total body vigilance, while others address the degree of attentional focus, perceived sensitivity to changes in bodily sensations, and average amount of time spent attending to sensation. Typical items include "I am very sensitive to changes in my internal bodily sensations" (0 = Not at all like me, 10 = Extremely like me). Additionally, there are composite ratings for 15 separate bodily sensations (e.g., numbness, tingling, vision changes, nausea). Participants are asked to "rate how much attention you pay to each of these sensations" (0 = None, 10 = Extreme). The BVS total scale has been shown to have good internal consistency ($\alpha = 0.82$) and adequate test–retest reliability ($r = .58$; Schmidt et al., 1997) and can be a useful indicator of treatment response for panic disorder (Deacon & Abramowitz, 2006a). Cronbach's alpha in this study was 0.81.

Kellner Illness Attitude Scale (IAS)

The IAS is a 29-item self-report measure that assesses the fears, attitudes, and beliefs associated with hypochondriasis. Each item is rated on a 5-point Likert scale ranging from "no" to "most of the time." After a few of the items were

removed due to questions surrounding their validity, the remaining 24-item scale was used to construct the five IAS dimensions (i.e., fear of illness and pain, symptom effects, treatment experience, disease conviction, and health habits; Hadjistavropoulos, Frombach, & Asmundson, 1999). This five-factor model is outlined by Hadjistavropoulos et al. (1999) and is the most appropriate factor structure for use in non-clinical samples. The 3-item IAS-Hypochondriacal Beliefs/Disease Conviction subscale is most relevant for this study. The items comprising the subscale are as follows: "Do you believe that you have a physical disease but the doctors have not diagnosed it correctly?"; "When your doctor tells you that you have no physical disease, do you refuse to believe them?"; and "When you have been told by a doctor what s/he found, do you soon begin to believe that you may have developed new illness?" The IAS-Disease Conviction subscale demonstrated acceptable internal consistency in this study (Cronbach's $\alpha = 0.68$).

Multidimensional Pain Inventory (MPI)

The MPI consists of 63 items assessing three separate domains of which perceived pain intensity and the impact of pain on different aspects of patient's lives being the most relevant for this study. This domain consists of five empirically derived subscales that measure (1) pain severity, (2) interference due to pain, (3) perceived control over pain and life events related to pain, (4) affective distress, and (5) support received from significant others. All items are rated on a 7-point Likert scale. Studies have demonstrated the MPI's adequate psychometric properties (i.e., internal consistency = 0.70–0.90, test–retest coefficients = 0.62–0.91; Kerns, Turk, & Rudy, 1985). MPI-Pain Severity and MPI-Pain Interference subscales were most relevant for this study. Typical items from the MPI-Pain Severity subscale include "Rate the level of your pain at the present moment" (0 = No Pain, 6 = Very intense pain) and "On average, how severe has your pain been during the last week?" (0 = Not at all, 6 = Extremely severe). Typical items from the MPI-Pain Interference subscale include "In general, how much does your pain interfere with your day-to-day activities?" (0 = No interference, 6 = Extreme interference) and "Since the time your pain began, how much has your pain changed your ability to work?" (0 = No Change, 6 = Extreme change). MPI-Pain Severity (Cronbach's $\alpha = 0.73$) and MPI-Pain Interference (Cronbach's $\alpha = 0.90$) subscales demonstrated good internal consistency in this study.

Procedure

The Institutional Review Boards at the University of Missouri–St. Louis, Boston University, and Beth Israel Deaconess Medical Center at Harvard Medical School approved this study. All patients presented with a chief complaint of chest pain and received a diagnosis of NCCP from their cardiologist after showing no evidence of electrocardiogram changes during an exercise tolerance test. A modified Bruce Protocol was used to conduct the exercise tolerance test. This test involves walking on a treadmill while the speed and inclination are adjusted over time, during which participants' hearts are monitored by electrocardiograph. The modified protocol was designed for individuals with cardiac issues and includes only minor adjusts in inclination. For testing guidelines, see Gibbons et al. (1997), and see Goldschlager, Selzer, and Cohn (1976) regarding reliability and validity information. After being informed that their testing showed no cardiac abnormalities, patients were invited to participate in a research study on medical and psychological factors that may be associated with chest pain. An eligibility screen based on the inclusion and exclusion criteria was conducted over the telephone for those who agreed to be contacted. Eligible patients were invited to participate in a longitudinal study that consisted of a clinical interview and self-report questionnaires at four time points. Although encouraged to participate in both the interview and the questionnaire portion of the study, patients were permitted to do either the interview or the questionnaire only, and were compensated $25 for participation at each time point. Trained clinical psychology doctoral students or a licensed psychologist conducted the structured clinical interviews (i.e., Anxiety Disorder Interview Schedule for DSM-IV; Di Nardo, Brown, & Barlow, 1994), and questionnaires were completed and returned to the research office via pre-paid envelopes.

Statistical Analysis

Two separate hierarchical multiple regression analyses predicting chest pain severity and chest pain interference were performed using anxiety sensitivity, body vigilance, and disease conviction as predictors, each being entered into a separate step in the model, respectively. Anxiety sensitivity was entered prior to body vigilance and disease conviction as it considered more of a stable trait-like factor (McNally, 1994). Likewise, body vigilance was entered prior to disease conviction because, from a theoretical perspective, attentional biases precede more complex cognitions, such as disease conviction. The independent variables used in the analyses were the total score of the ASI, the total score for the BVS, and the disease conviction subscale score for the IAS. The dependent variable in model 1 was the pain severity subscale score for the MPI, and the dependent variable in model 2 was the interference due to pain subscale score for the MPI. It was hypothesized that disease conviction will account for a significant amount of the variance in chest pain severity and interference after accounting for the variance contributed by anxiety sensitivity and body vigilance.

Relative weights analysis was also used to examine the relative contribution of each predictor variable (i.e., ASI, BV, DC) to the outcome measures (i.e., pain severity, pain interference) in order to measure the unique influence of each predictor while excluding any variance that is redundant among them in a model where they are entered simultaneously. Body vigilance was hypothesized to account for a substantial portion of the variance in both pain severity and interference in addition to anxiety sensitivity and body vigilance.

Results

Descriptive Statistics and Preliminary Analyses

A total of 229 participants participated in this study. However, 39 patients did not complete the necessary questionnaires and were excluded from the analyses ($N = 190$). The average age of participants was 50 years old ($SD = 10.4$; range = 27–78) and the sample was 56% female. Over half of the sample was married (56%), working full-time (58%), and well educated (63% with a bachelor's degree; 99% with a high school diploma). The majority of participants self-identified as Caucasian (85%), and most of the remaining participants identified as African-American (11%).

Demographic variables were examined in relation to each study variable. Outcome measures indicate no group differences with respect to relationship status, employment status, and ethnicity, p's > .05. Participants' age was found to have a moderate negative correlation with body vigilance scores $r(188) = -.278$, $p < .001$, but was not correlated with any other outcome measure. In addition, female participants ($M = 2.91$, $SD = 1.25$) reported a slightly higher level of pain severity compared to males ($M = 2.51$, $SD = 1.30$; $t(190) = -2.15$, $p < .05$; $d = 0.31$). No other differences were found regarding demographic variables. The influence of certain health behaviors on outcome measures was also examined. Chest pain severity and life interference were both found to be unrelated to participant's report of smoking behavior ($p > .05$), caffeine consumption ($p > .05$), and physical activity ($p > .05$); hence, these variables were not included in the regression analyses.

With regard to the participants' chest pain, approximately 40% had experienced chest pain for over a year, and 81% reported having chest pain for at least one month. Approximately half of participants (51%) experienced episodes of chest pain at least weekly with 29% experiencing chest pain on a daily basis. Only a small portion of participants (3%) reported continuous chest pain suggesting that most of the participants experience chest pain episodically. Most of the participants (51%) described their chest pain as being of at least moderate intensity, and 63% reported these episodes to last no more than 20 min each. The majority of participants (73%) reported only mild interference due to their chest pain, and MPI-subscale scores were found to be lower than those reported in chronic pain samples (Turk & Rudy, 1990). Both chest pain severity ($p < .05$) and life interference due to chest pain ($p < .001$) were significantly correlated with participants' number of medical visits in the past year. Overall, this sample of individuals with NCCP reported less pain severity and pain-related life interference when compared to other normative or chronic pain samples.

Given that the majority of participants reported mild chest pain severity and interference, it was not surprising that the mean anxiety sensitivity ($M = 16.7$, $SD = 10.96$) for the sample fell within the range typically found in non-clinical samples ($M = 14.2$–22.5; Peterson & Reiss, 1992). However, 20% of participants reported anxiety sensitivity scores similar to those reported by individuals diagnosed with a DSM Axis-I anxiety disorder (ASI > 24.9), and about 8% of the sample reported scores higher than those typically found in individuals with Panic Disorder (ASI > 36.6; Taylor, Koch, & McNally, 1992). Likewise, the mean body vigilance score ($M = 13.14$, $SD = 7.70$) was similar to scores found in other non-clinical samples ($M = 15.52$, $SD = 8.74$; Olatunji et al., 2007), but approximately 19% of the participants reported body vigilance scores higher than the average scores found in patients diagnosed with a primary Axis-I anxiety disorder (BVS > 20.57; Olatunji et al., 2007). The average disease conviction score ($M = 1.52$, $SD = 2.10$) was also found to be lower than that in Hypochondriacal populations ($M = 7.5$, $SD = 3.4$), but elevated compared to non-clinical samples ($M = 0.6$, $SD = 1.2$; Kellner, Abbott, Winslow, & Pathak, 1987).

Associations Between Predictor Variables and Chest Pain

To investigate the hypothesis that anxiety sensitivity, body vigilance, and disease conviction would each be positively related to life interference from chest pain and chest pain severity, correlations among these variables were examined. Anxiety sensitivity (ASI-total) was strongly associated with life interference due to chest pain (MPI-Interference), $r(185) = .488$, $p < .001$, and moderately associated with chest pain severity (MPI-Severity), $r(188) = .237$, $p < .001$. Body vigilance scores (BVS) also showed a moderate association with chest pain interference (MPI-Interference), $r(183) = .241$, $p < .001$, and with chest pain severity (MPI-Severity), $r(187) = .205$, $p < .01$. In addition, disease conviction (IAS-Disease Conviction) showed a moderate association with chest pain interference (MPI-Interference), $r(186) = .340$, $p < .001$, and a small correlation with chest pain severity (MPI-Severity), $r(190) = .148$, $p < .05$. Thus, anxiety sensitivity, body vigilance, and disease conviction

were all found to be associated with the pain-related outcome measures. Means, standard deviations, and zero-order correlations for all study variables are reported in Table 1.

Hierarchical Regression Analyses Predicting Pain Interference and Severity

A hierarchical regression analysis was conducted to examine the relationship between disease conviction and life interference due to chest pain, after accounting for anxiety sensitivity and body vigilance. In Step 1 of the regression, anxiety sensitivity accounted for 22% of the variance (R^2) in chest pain interference, $F(1,174) = 48.63$, $p < .001$. In Step 2, body vigilance predicted a non-significant amount of additional variance ($\Delta R^2 = 0.005$, $p = .30$). However, in Step 3, disease conviction accounted for a significant amount of additional variance ($\Delta R^2 = 0.03$, $p < .05$) in predicting chest pain interference. Overall, the model accounted for 25% of the variance (R^2) in life interference due to chest pain, $F(3,172) = 18.92$, $p < .001$. Additional details from this regression model can be found in Table 2.

Next, a hierarchical regression analysis was conducted to examine the relationship between disease conviction and chest pain severity, after accounting for anxiety sensitivity and body vigilance. Given that females demonstrated a slightly higher level of pain severity than males, gender was entered into Step 1 of the regression model. In Step 2, anxiety sensitivity accounted for 5% of the variance (R^2) in chest pain severity, $F(1,177) = 9.102$, $p < 0.01$. In Step 3,

body vigilance accounted for a significant amount of additional variance ($\Delta R^2 = 0.023$, $p < 0.05$). However, in Step 4, disease conviction did not predict a significant amount of additional variance ($\Delta R^2 = 0.004$, $p = 0.68$). Overall, the model accounted for 10.7% of the variance (R^2) in chest pain severity, $F(3,175) = 4.81$, $p < 0.01$. Additional details can be found in Table 3.

To further examine the degree to which anxiety sensitivity, body vigilance, and disease conviction contribute to pain severity and interference, a relative weights analysis (Johnson, 2000) was performed. Relative weights analysis enables the examination of each predictor's unique contribution to the total variance explained in the model and provides a statistical method for addressing any shared variance in the assessment approaches used. Of the total variance in pain interference explained by all three predictors ($R^2 = 0.248$), anxiety sensitivity accounts for 64% ($r^2 = 0.160$), body vigilance for 11% ($r^2 = 0.027$), and disease convictions explains 25% ($r^2 = 0.061$). In addition to the hierarchical regression analysis, this provides further evidence that anxiety sensitivity and disease conviction are significant contributors to pain interference. Of the total variance in pain severity explained by all three predictors ($R^2 = 0.076$), anxiety sensitivity accounts for 42% ($r^2 = 0.032$), body vigilance for 48% ($r^2 = .037$), and disease convictions explains 10% ($r^2 = .008$). Likewise, this provides further evidence that anxiety sensitivity and body vigilance are significant contributors to pain severity.

Table 1 Independent and dependent variable intercorrelations

Measure	Mean (SD)	CAQ				
		AS	BV	DC	Pain-I	Pain-S
Anxiety sensitivity	16.70 (10.96)	–	–	–	–	–
Body vigilance	13.14 (7.70)	.347***	–	–	–	–
Disease conviction	1.52 (2.10)	.411***	.171*	–	–	–
Pain interference	0.93 (1.16)	.488***	.241***	.340***	–	–
Pain severity	2.70 (1.30)	.237**	.205**	.148*	.400***	–

Anxiety sensitivity ASI-Total score, *Body vigilance* BVS-Total score, *Disease conviction* IAS-disease conviction subscale, *Pain interference* MPI-Interference subscale, *Pain severity* MPI-severity subscale

*$p < .05$; **$p < .01$; ***$p < .001$; ($N = 183$)

Table 2 Hierarchical regression analysis for pain interference

	B	$SE\ B$	β	t	R^2	ΔR^2	F-change
Step 1							
Anxiety sensitivity	.051	.007	.467	6.97***	.218	.218	48.63***
Step 2							
Body vigilance	.012	.011	.076	1.05	.223	.005	1.100
Step 3							
Disease conviction	.091	.038	.170	2.38*	.250	.025	5.66*

*$p < .05$; **$p < .01$; ***$p < .001$; ($N = 177$)

Table 3 Hierarchical regression analysis for pain severity controlling for gender

	B	$SE\ B$	β	t	R^2	ΔR^2	F-change
Step 1							
Gender	.469	.192	.182	2.44*	.033	.033	5.964*
Step 2							
Anxiety sensitivity	.027	.009	.219	3.02**	.081	.048	9.102**
Step 3							
Body vigilance	.028	.013	.164	2.12*	.104	.023	4.431*
Step 4							
Disease conviction	.039	.047	.064	.823	.107	.004	.678

$^*p < .05$; $^{**}p < .01$; $^{***}p < .001$; ($N = 180$)

Discussion

The purpose of this study was to evaluate the impact of disease conviction in the context of constructs considered integral to our understanding of NCCP, namely anxiety sensitivity and body vigilance. First, we sought to confirm the relationship between anxiety sensitivity and pain-related variables associated with NCCP. Anxiety sensitivity was indeed correlated with pain-related life interference and chest pain severity. This finding provides further empirical support that NCCP patients demonstrate an increased fear of physical sensations, and perhaps cardio-pulmonary sensations in particular (Eifert et al., 1996; Lipsitz et al., 2004; White et al., 2011). Second, we examined the relationship between body vigilance and chest pain severity and interference. Consistent with earlier findings (White et al., 2010), the degree of attention paid to internal sensations was positively associated with the level of life interference due to chest pain as well as the severity of chest pain experienced. We also assessed the relationship between disease conviction and pain-related variables associated with NCCP. The degree to which one believes they have a cardiac illness was positively related to the severity of chest pain and the degree to which it interferes in daily activities. This finding substantiates Eifert et al. (1996) research demonstrating that disease conviction is a construct relevant to those diagnosed with NCCP.

Next, hierarchical regression analysis revealed that anxiety sensitivity is a strong factor in life interference due to chest pain. However, contrary to our hypothesis, body vigilance did not significantly contribute to the variance in chest pain interference beyond what was explained by anxiety sensitivity. Despite this, disease conviction contributed significant additional variance beyond what was predicted by anxiety sensitivity and body vigilance. This suggests that when controlling for the fear of and attention paid to cardiac sensations, the degree to which one believes they have a cardiac condition has a significant impact on the level of life interference they experience. This echoes similar findings that NCCP patients who report their condition as more "serious" show greater levels of pain-related life interference (Israel, White, & Gervino, 2015). The second hierarchical regression analysis showed that anxiety sensitivity is also a significant predictor of NCCP patients' chest pain severity, and body vigilance contributed a significant amount of additional variance beyond that predicted by anxiety sensitivity. However, disease conviction did not explain a significant amount of additional variance after accounting for the other predictors. Relative weights analysis further corroborated these results. Interestingly, the findings regarding pain severity is opposite of what we might expect given each of the predictor's relationship to chest pain interference. One possible explanation for these findings might be that the amount of attention allocated to bodily sensations alters one's perception of pain, but only the degree to which one internalizes the meaning of those sensations (i.e., disease conviction) impacts subsequent behavior.

Together, these findings indicate that anxiety sensitivity, body vigilance, and disease conviction may all be important constructs in the etiology and maintenance of NCCP. The lack of a satisfactory biomedical explanation for NCCP has encouraged many researchers to examine the various psychological constructs that may be relevant to a biopsychosocial conceptualization of the disorder (White & Raffa, 2004). Our theoretical understanding of panic disorder and its similarity to NCCP symptomatology has been a catalyst for much of the research in this area. Much like panic disorder, cognitive misappraisals of innocuous bodily sensations are integral to our conceptualization of NCCP (Mayou, 1998; White & Raffa, 2004). Furthermore, empirical evidence supports the notion that interpreting cardiac sensations as harmful may not only intensify chest pain severity (Aikens et al., 2001), but lead to restrictions in daily activity for fear of exacerbating symptoms (White et al., 2011). This study provides further empirical support for the impact anxiety sensitivity has on chest pain severity and interference. Patients who report fearing the physical sensations related to anxiety may exhibit more severe chest pain and avoid activities that could potentially onset those sensations.

Likewise, theoretical models of NCCP have posited a central role for increased vigilance to cardio-pulmonary sensations in the etiology and maintenance of the syndrome (Eifert et al., 2000; White & Raffa, 2004). Laboratory experiments have not only demonstrated a link between the deployment of attentional resources and the level of pain experienced (Arntz, Dreessen, & Merckelbach, 1991), but recent findings have demonstrated a connection between body vigilance and chest pain within NCCP (White et al., 2010). Our findings expand this literature by demonstrating that body vigilance impacts chest pain severity, but not life interference, after accounting for fear of anxiety-related physical sensations. This suggests that increased awareness of cardiac sensations may impact one's level of pain, but not influence one's response to that pain (e.g., avoidance of physical activity) after fear of those sensations has been taken into account. This finding is not entirely surprising considering that fear of interoceptive cues has been shown to partially mediate the relationship between body vigilance and chest pain interference (White et al., 2010).

Rooted in a conceptual understanding of hypochondriasis, theoretical models of NCCP have also posited that interpreting minor physical sensations as evidence of a serious medical illness is a fundamental aspect of NCCP (Mayou, 1998). Although there is evidence to suggest that NCCP patients show an elevated tendency to attribute their chest pain to a physical condition (Eifert et al., 1996), the impact of disease conviction in relation to anxiety constructs that have already been shown to affect chest pain severity and interference is unknown. These findings suggest that the subjective severity of chest pain is not related to how much one believes they have a physical cardiac condition, after taking into account their fear of and vigilance for the very symptoms that would serve as evidence of a cardiac condition. However, despite not directly contributing to the subjective experience of chest pain, disease conviction appears to play an important role in the avoidance behaviors exhibited by those with NCCP. The degree to which one believes their benign cardiac sensations are evidence of a physical condition substantially contributes to their pain-related life interference, beyond fear of and attention to those sensations. As has been previously suggested, patients who attribute their chest pain to a physical cause may be more likely to attempt relieving their symptoms by modifying their behavior (White et al., 2011). This extends our current theoretical understanding of NCCP by elucidating the specific way in which these underlying constructs affect those with this impairing syndrome. These findings also have important practical implications for interventions designed to treat NCCP. For instance, disease conviction may be a particularly useful cognitive variable to target when patients have difficulty engaging in behavioral interventions aimed at reducing avoidance. Likewise, NCCP patients reporting greater pain severity may derive

increased benefit from interventions that focus on enhancing patients' ability to control attention through techniques such as mindfulness.

There are limitations to this study that warrant consideration. This study employed a within-subjects design which precludes comparison with a control group. As such, these findings may or may not be specific to patients with NCCP. The generalizability of this study is limited to patients who experience chronic cardiac symptoms, but receive no physical explanation after medical evaluation and an exercise tolerance test. The results from this study also relied on self-report data, and thus were subject to participant bias. These data were drawn from a single time point and bar causal conclusions to be drawn. In addition, this study employed the original 16-item version of the Anxiety Sensitivity Index rather than the updated 18-item ASI-3, which has demonstrated improved psychometrics over the original (Taylor et al., 2007); nevertheless, anxiety sensitivity was analyzed as a unified construct as part of this study circumventing some of the concerns regarding original measure. It should also be noted that constructs like anxiety sensitivity and body vigilance may exert influence on the experience of NCCP patients due to their relationship to higher-order personality factors, such as neuroticism. Although beyond the scope of this study, future research might investigate the role of personality on the experience of chest pain and life interference in NCCP. The influence of certain health behaviors (e.g., smoking, caffeine consumption, physically activity) on study outcomes was briefly examined, yet other heath indices related to heart disease or anxiety (e.g., BMI, sleep, substance use) or clinical factors associated with chest pain (e.g., medical illnesses such as GERD) were not controlled for in this study. Future research could improve upon these findings by replicating this study while controlling for such factors. Despite these limitations, this study employed reliable and validated measures of each construct being assessed. It is also the first study to examine the impact of disease conviction in a NCCP population without neglecting the influence of anxiety sensitivity and body vigilance.

Overall, NCCP is a common condition that has eluded medical explanation. Research into the psychological mechanisms that modulate the experience of chest pain have provided a framework for better understanding this syndrome. Anxiety sensitivity and body vigilance may both represent psychological vulnerabilities that increase one's risk for developing NCCP, and may exacerbate the experience of chest pain. In addition, anxiety sensitivity and disease conviction may contribute to the psychosocial impairment brought about by significant symptoms. Fortunately, all three of these mechanisms are potential targets for psychological intervention. In addition, these findings suggest cognitive variables, such as disease conviction, may be especially important to target in inventions due to their connection with

the functional impairment found in NCCP. It is crucial that research continue to explore the roles such psychological factors play in the etiology and maintenance of NCCP. A comprehensive description of the factors that contribute to this syndrome is imperative for developing interventions best suited to mitigate its impact on each individual.

Compliance with Ethical Standards

Conflict of interest Caleb M. Pardue, Kamila S. White, and Ernest V. Gervino declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Human and Animal Rights and Informed Consent This article does not contain any studies with animals performed by any of the authors.

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