BORDERLINE PERSONALITY DISORDER FEATURES AND PAIN:
THE MEDIATING ROLE OF ANXIETY SENSITIVITY
AMONG A COLLEGE STUDENT SAMPLE

By

RACHEL ELYSE JONES

A dissertation submitted in partial fulfillment of the
requirements for the degree of

DOCTOR OF PHILOSOPHY

WASHINGTON STATE UNIVERSITY
Department of Psychology

DECEMBER 2014

© Copyright by RACHEL ELYSE JONES, 2014
All Rights Reserved
To the Faculty of Washington State University:

The members of the Committee appointed to examine the dissertation of RACHEL ELYSE JONES find it satisfactory and recommend that it be accepted.

_________________________________
Sarah Tragesser, Ph.D., Chair

_________________________________
David Marcus, Ph.D.

_________________________________
Paul Kwon, Ph.D.
ACKNOWLEDGMENT

I would like to thank my committee chair, Dr. Sarah Tragesser, for her invaluable mentorship throughout the process of my dissertation. I am grateful for her investments of time and expertise that have helped me to develop my skills and abilities as a researcher. I would also like to acknowledge Dr. David Marcus and Dr. Paul Kwon for serving on my dissertation committee and to thank them for their efforts in helping me craft this project.
BORDERLINE PERSONALITY DISORDER FEATURES AND PAIN:
THE MEDIATING ROLE OF ANXIETY SENSITIVITY
AMONG A COLLEGE STUDENT SAMPLE

Abstract

by Rachel Elyse Jones, Ph.D.
Washington State University
December 2014

Chair: Sarah Tragesser

Chronic pain is a widespread phenomenon that results in significant burden in the United States and other industrialized countries. There is also evidence of elevated prevalence rates of personality disorders among chronic pain patients in comparison to prevalence rates of personality disorders among the general population. Further, the co-occurrence of chronic pain and personality disorders has been associated with a variety of negative outcomes, such as increased functional disability and health care utilization.

Recent findings have highlighted substantial rates of borderline personality disorder, in particular, among individuals experiencing chronic pain. Although it is clear that borderline personality disorder and chronic pain are associated, there is a lack of research regarding the particular relation between these two phenomena. As such, the purpose of the current study was to explore the connection between borderline personality disorder features and pain among a sample of college students. More specifically, the current study examined the role of negative
affect (i.e., anxiety sensitivity, depression, trait anxiety, and anger) in explaining the borderline personality disorder-pain connection.

Significant, positive associations between borderline personality disorder features and each measured pain-related variable – including pain severity, pain interference, and pain during activities – were found to be fully accounted for by indirect effects of negative affect. This suggests that the relation between borderline personality disorder features and pain can be explained by the mediating effect of negative affect, and that multiple negative affect variables, including anxiety sensitivity and depression, play a significant role in accounting for this association among the current sample.

Adding to the literature, the current investigation was the first to explore the role of anxiety sensitivity, a factor that has been shown to be significantly related to chronic pain and elevated in borderline personality disorder, in the association between borderline personality disorder features and chronic pain. The current findings also lend support for future investigations of the potential clinical utility of addressing particular aspects of negative affect in individuals presenting with both borderline personality disorder features and pain complaints.
**TABLE OF CONTENTS**

ACKNOWLEDGMENT .................................................................................................................. iii

ABSTRACT ...................................................................................................................................... iv-v

LIST OF TABLES ............................................................................................................................. viii

LIST OF FIGURES ........................................................................................................................... ix

CHAPTER

1. INTRODUCTION ....................................................................................................................... 1
   Chronic Pain and Personality Disorders ...................................................................................... 1
     Impact of Personality Disorders on Chronic Pain and Health .................................................... 2
     Purported Theoretical Relationship ............................................................................................ 3
     Chronic Pain and Borderline Personality Disorder ................................................................. 4
   Chronic Pain and Negative Affect .................................................................................................. 7
   Chronic Pain and Depression ......................................................................................................... 7
   Chronic Pain and Anxiety ............................................................................................................... 8
   Chronic Pain and Anger .................................................................................................................. 9
   Borderline Personality Disorder and Negative Affect ................................................................. 10
   Chronic Pain and Anxiety Sensitivity ............................................................................................ 11

2. CURRENT STUDY ..................................................................................................................... 13

3. METHOD ..................................................................................................................................... 15
   Participants ................................................................................................................................. 15
   Measures ....................................................................................................................................... 15
     Borderline Personality Disorder Features ............................................................................... 15
     Anxiety Sensitivity ...................................................................................................................... 16
Depression................................................................. ................................................... ................16
Trait Anxiety................................................................. ................................................... ........17
Anger.................................................................................................................. ............ 17
Pain Severity and Interference................................................................. ........................................... 17
Pain During Activities............................................................................................... ..............18
Procedure ..................................................................................................................................19
4. RESULTS ..........................................................................................................................19
Descriptive Statistics ................................................................................................. ........19
Path Analyses .................................................................................................................. 19
Pain Severity.................................................................................................................. ........20
Pain Interference............................................................................................................. ........20
Pain During Activities ................................................................................................. ........21
5. DISCUSSION ................................................................................................................... .......................22
REFERENCES ................................................................................................................... ..........................28
LIST OF TABLES

1. Table 1; Descriptive Statistics .................................................................41
2. Table 2; Bivariate Correlations .................................................................42
3. Table 3; Direct Effect of Negative Affect on Pain ....................................43
4. Table 4; Effect Decomposition Table .......................................................44
5. Table 5; Specific Indirect Effects from Borderline Personality Disorder Features to Pain through Negative Affect ..................................................45
LIST OF FIGURES

1. Figure 1; Proposed Path Analyses .................................................................46
Dedication

This dissertation is dedicated to my parents, sister, and fiancé, each of whom have graciously provided me with unconditional support throughout my graduate career. Without them, this pursuit would not have been possible.
CHAPTER ONE: INTRODUCTION

Chronic pain is a widespread phenomenon that results in significant burden in the United States and other industrialized countries. In a nationally representative sample of 27,000 U.S. adults, approximately 30% of people reported currently experiencing some type of chronic pain, with previous estimates ranging from 14% to 64% (Johannes, Le, Zhou, Johnston, & Dworkin, 2010). Other recent statistics demonstrate that more adults in the U.S. are directly affected by chronic pain than by diabetes, cancer, and heart disease combined (American Academy of Pain Medicine; AAPM; 2012). The annual financial cost of chronic pain in the U.S. is estimated to be $635 billion, measured in terms of both medical costs and loss of productivity (Institute of Medicine; IOM; 2011). There is also a wealth of evidence demonstrating a positive association between chronic pain and lowered quality of life (QOL), with some studies estimating that chronic pain patients (CPPs) demonstrate the lowest QOL ratings of any medical condition (Becker et al., 1997).

The current study aims to further explore the topic of chronic pain by focusing on the relation between pain and borderline personality disorder (BPD) features. First, the current literature examining chronic pain, personality disorders, and negative affect will be reviewed. In the present study, the association between BPD features and pain are hypothesized to be fully explained by negative affect variables. Study findings, including implications of results and ideas for future directions, will also be presented.

CHRONIC PAIN AND PERSONALITY DISORDERS

Chronic pain and personality disorders (PDs) frequently co-occur (Dersh, Polatin, & Gatchel, 2002). Between 24% to 81% of CPPs meet criteria for an Axis II disorder (Burton, Polatin, & Gatchel, 1997; Gatchel, Polatin, & Kinney, 1995; Reich, Tupin, &
Abramowitz, 1983). These rates are significantly higher than prevalence rates of personality disorders (PDs) among the general population (i.e., 5.9% to 13.5%; Dersh et al., 2002; Gatchel, 1996). Although a number of studies have established a relationship between PDs and pain (Weisberg, Gallagher, & Gorin, 1996; Weisberg & Keefe, 1997), the few studies that have examined which PDs occur most often in CPP populations have produced mixed results (Dersh et al., 2002). Gatchel, Polatin, Mayer, and Garcey (1994) found that paranoid PD was the most frequent PD diagnosis in CPPs, whereas Fishbain, Goldberg, Meagher, Steele, and Rosomoff (1986) reported that dependent PD was most common. In other CPP samples, Reich, Rosenblatt, and Tupin (1983) found that histrionic PD diagnoses were most frequent, and Sansone, Whitecar, Meier, and Murry (2001) found that borderline PD diagnoses occurred most often.

Impact of PDs on Chronic Pain and Health

There is emerging evidence that the co-occurrence of PDs and pain is predictive of a variety of negative outcomes. For instance, Powers and Oltmanns (2013) reported that PD features have been associated with increased negative self-perceptions of health status, and the presence of a PD in adolescence has been prospectively linked with increased odds of experiencing pain, physical ailments, and poor physical health in adulthood (Chen et al., 2009). Those with comorbid PD and Axis I diagnoses demonstrated even higher rates of negative health outcomes over time compared to individuals with PDs only (Chen et al., 2009). Further, PD status in acute low back pain (LBP) patients was predictive of non-return to work six months post-assessment (Gatchel et al., 1995). After controlling for current health status, depression levels, and health behaviors, PDs were associated with decreased physical functioning, increased fatigue, increased functional disability, and increased pain symptoms at
baseline and six-month follow-up in a sample of community members (Powers & Oltmanns, 2012). Additionally, the presence of a PD was prospectively linked with increased medication use and health care utilization over time (Powers & Oltmanns, 2012). Among CPPs undergoing outpatient treatment, the presence of a PD diagnosis was related to increased levels of pain and distress at both the beginning and end of treatment compared to non-PD patients (Elliott, Jackson, Layfield, & Kendall, 1996).

**Purported Theoretical Relationship**

Weisberg and Keefe (1997) put forth a diathesis-stress model to explain the relationship between PDs and pain. They conceptualized the presence of persistent pain as a stressor that causes significant burden on coping resources. As pain continues to tax the individual in multiple ways, coping resources are thought to diminish; this may result in manifestation of a PD among individuals with underlying personality traits associated with maladaptive coping styles. Conrad and colleagues (2007) reported that a temperamental dimension of harm avoidance, or sensitivity to fear cues and related inhibition, was elevated among CPPs who met PD criteria compared to healthy controls. This finding suggests that those with comorbid chronic pain and PDs may be particularly prone to hypervigilance toward pain cues, which may promote avoidance and escape-related behaviors. These specific coping strategies for pain are hypothesized to exacerbate the experience of pain and related disability (Vlaeyen & Linton, 2000).

Weisberg and Keefe’s (1997) model is supported by research conducted among a group of chronic LBP patients receiving pain treatment (Vittengl, Clark, Owen-Salters, & Gatchel, 1999). From pre- to post-treatment, there was a significant decrease in overall rates of PD diagnoses. Some have sought to explain these findings by suggesting that post-treatment,
coping resources were increased and thus the manifestation of personality symptoms, although still present, decreased such that fewer participants met DSM, dimensional criteria for a PD (Dersh et al., 2002). Results from a national, 20-year investigation of the effects of psychiatric and personality diagnoses on health outcomes suggest a directional relationship between PDs and pain that is opposite to Weisberg and Keefe’s (1997) model (Chen et al., 2009). Specifically, the authors reported that adolescents with PD diagnoses demonstrated significantly higher odds ratios of developing pain-related conditions over time (Chen et al., 2009).

**Chronic Pain and Borderline Personality Disorder**

Recent findings suggest that researchers should specifically investigate the relation between BPD and pain. BPD is a psychiatric disorder composed of symptoms including mood instability, impulsivity, self-harm, interpersonal problems, and identity disturbances (APA, 2000). Results from a national survey indicate that BPD is directly related to increased presence of physical diseases, even after controlling for Axis I and other Axis II disorders (El-Gabalawy, Katz, & Sareen, 2010). Further, the co-occurrence of BPD and chronic illness was associated with greater odds of poor QOL and suicide attempts compared to the effects of BPD alone (El-Gabalawy et al., 2010). Additionally, Bender and colleagues (2001) demonstrated that BPD patients tend to overuse medical resources.

The association between BPD and pain is particularly important in light of the current popularity of opioid therapy for the treatment of chronic pain (Ballantyne & Shin, 2008). Tragesser, Jones, Robinson, Stutler, and Stewart (2013) reported that BPD features were associated with increased quantity and frequency of prescription opioid use, risk for pain medication misuse, as well as opioid consequences and dependence features in a sample of
young adults. Although opioid therapy is a common treatment intervention for CPPs, the analgesic effects of opioids are not necessarily sustained across time (Ballantyne & Shin, 2008), and long-term opioid use is associated with a variety of negative psychosocial and physical health outcomes (Gruber, Silveri, & Yurgelun-Todd, 2007; Savage, 1996; Sullivan, Von Korff, Banta-Green, Merrill, Saunders, 2010). Further, in BPD patients, there is emerging evidence that opioid-based pain medications may be associated with increased likelihood of dissociative symptoms (Saper & Lake, 2002).

Although Sansone, Pole, Dakroub, and Butler (2006) did not find an association between BPD features and pain, there are a number of studies that do report a relation between BPD or BPD features and pain complaints (e.g., Braden & Sullivan, 2008; Dersh, Gatchel, Mayer, Polatin, & Temple, 2006; Sansone, Sinclair, & Wiederman, 2010). In a review of eight studies of CPPs that assessed for BPD, Sansone and Sansone (2012) found that rates of BPD diagnoses ranged from 9.4 to 58%, with an average prevalence rate of 30%. Compared to the general population, in which 1.4% of individuals meet BPD criteria, the rate of BPD among CPPs is staggering. The eight studies of BPD and pain conducted to date have included investigations of both clinical and community samples (Sansone & Sansone, 2012), using diagnostic interviews (Dersh et al., 2006; Fischer-Kern et al., 2011; Gatchel, et al., 1994; Sansone et al., 2001) and self-report measures (Braden & Sullivan, 2008; Manchikanti, Pampati, Beyer, & Damron, 2002; Sansone, Mueller, Mercer, & Wiederman, 2010; Workman, Hubbard, & Felker, 2002) to assess for the presence of BPD. Conrad and colleagues (2007) found that among CPPs who met criteria for a Cluster B PDs, PD symptoms were strongly correlated with high novelty seeking, a facet of impulsivity which is related to BPD (Tragesser & Robinson, 2009; Whiteside & Lynam, 2001). In a large, survey-based U.S. population study, McWilliams and Higgins (2013)
found that BPD features were positively related to chronic pain conditions, such as arthritis, chronic spinal pain, and severe headaches, even after adjusting for demographics and other forms of psychopathology.

BPD is positively associated with increased severity of current, 30-day, and 12-month pain complaints in both CPPs (Tragesser, Bruns, & Disorbio, 2010) and internal medicine patients (Sansone, Mueller, et al., 2010). Older BPD patients reported increased pain experiences compared to younger BPD patients (Blum et al., 2008), and the presence of persistent BPD features was also predictive of increased prevalence of pain syndromes (Frankenberg & Zanarini, 2004). Powers and Oltmanns (2012) showed that BPD symptoms were significantly linked to measures of physical functioning. Additionally, first-degree relatives of BPD patients have demonstrated increased rates of somatic pain disorder diagnoses versus relatives of non-BPD patients (Weisberg, 2000).

Current conceptualizations of BPD assert that two core features – affective instability and impulsivity – account for symptoms of the disorder (New & Siever, 2002; Siever & Davis, 1991). In fact, Tragesser and Robinson (2009) found that both affective instability and impulsivity uniquely contributed to total BPD scores, and that the combination of the two factors accounted for 60% of the variance in BPD scores. Further, Tragesser, Bruns, and Disorbio (2010) assessed for the presence of BPD features among a CPP population and reported that the significant association between BPD and pain was fully mediated by negative affect, including measures of depression, anxiety, and hostility. This finding suggests that among those with BPD features, underlying negative affect is particularly important in explaining the co-occurrence of chronic pain.
CHRONIC PAIN AND NEGATIVE AFFECT

The finding that negative affect accounted for the relationship between BPD features and pain complaints is consistent with research showing that, among CPPs in general, negative affect has also been linked to the experience of pain (Dersh et al., 2002). Williams, Urban, Keefe, Shutty, and France (1995) found that CPPs endorsed elevated levels of depression, anxiety, and somatization on the Symptom Checklist 90-Revised (Derogatis, 1983), whereas other reports demonstrate that elevated levels of depression, anxiety, and anger are frequently seen in CPPs (Kreitler & Niv, 2007).

Chronic Pain and Depression

Bair, Robinson, Katon, and Kroenke (2003) reviewed 42 studies conducted among chronic pain patients and reported a mean prevalence rate of Major Depressive Disorder between 18% and 85%. Reported current prevalence rates of major depressive disorder (MDD) among those with chronic pain range between 30% and 54%, whereas reported lifetime prevalence rates range from 32% to 57% (Banks & Kearns, 1996; Katon, Egan, & Miller, 1985; Walker et al., 1995). Both current and lifetime estimates of MDD among chronic pain patients are higher than MDD estimates among the general population (5% and 17%) and other medical diagnoses, such as heart disease (14% and 27%) (Banks & Kearns, 1996).

Additionally, the presence of pain symptoms is associated with a two to three-fold increased risk for depression (Kroenke & Price, 1993; Magni, Moresche, Merskey, & Luchini, 1993). The likelihood of developing depression also increases as a function of number of pain symptoms in CPPs (Dworkin & Gitlin, 1991), primary care patients (Kroenke et al., 1994), and community members (Von Korff, Dworkin, LeResche, & Kruger, 1988), with the presence of two pain complaints predicting a six-fold increased risk and the presence
of three or more pain complaints predicting an eight-fold increased risk (Katon & Sullivan, 1990). The likelihood of experiencing depression increases as a function of increased pain severity (Carroll, Cassidy, & Cote, 2000; Lamb et al., 2000; Moldin et al., 1993), increased frequency of pain symptoms (Wang et al., 1999), and increased duration of pain complaints (Gureje, Von Korff, Simon, & Gater, 1998). As depressive symptoms increase in severity, individuals report experiencing pain symptoms more frequently (Von Korff et al., 1988).

**Chronic Pain and Anxiety**

Studies conducted among community members in the U.S. and internationally indicate an increased prevalence rate of anxiety disorders among those suffering from pain complaints versus the general population (35% versus 17%) (McWilliams, Cox, & Enns, 2003; McWilliams, Goodwin, & Cox, 2004). Overall, community members who endorse chronic pain are two-to-three times more likely to meet criteria for a current anxiety disorder (Demyttenaere et al., 2007), whereas women with fibromyalgia demonstrate a four-to-five-fold increase in lifetime prevalence rates of generalized anxiety disorder, posttraumatic stress disorder, and obsessive-compulsive disorder (Raphael, Janal, Nayak, Schwartz, & Gallagher, 2006).

Among clinical pain populations, current prevalence estimates of anxiety disorders range from 7% to 29% (Asmundson & Katz, 2009; Reme, Tange, Moe, & Eriksen, 2011), with some studies reporting current prevalence rates (25-29%) higher than those in the general population (18%; Atkinson, Slater, Patterson, Grant, & Garfin, 1991; Kinney, Gatchel, Polatin, Fogarty, & Mayer, 1993). There is also evidence that lifetime prevalence rates of anxiety disorders among CPPs are higher than prevalence rates for healthy controls (Kinney et al., 1993; Polatin, Kinney, Gatchel, Lillo, & Mayer, 1993) and the general population (Atkinson et al., 1991). McCracken, Gross, Aikens, and Carnrike (1996) also reported that anxiety accounted for 16-
54% of variance in the report of pain severity, pain-related behaviors, and disability among a treatment-seeking sample of CPPs.

**Chronic Pain and Anger**

Reports suggest that anger and pain are related, with researchers reporting that between 69% and 88% of CPPs endorse experiencing anger (Corbishley, Hendrickson, & Butler, 1990; Okifuji, Turk, & Curran, 1999). Even after adjusting for potential elevation of anger among patients in general, Fishbain and colleagues (2010) reported that levels of anger and chronic anger were elevated among CPPs compared to community members with medical disorders. A positive correlation between anger and pain has been established in studies using a variety of clinical pain populations, such as tension headaches (Hatch et al., 1991), cancer pain (Glover, Dibble, Dodd, & Miaskowski, 1995; Sela, Bruera, Cooner-Spady, Cumming, & Walker, 2002), spinal cord injuries (Conant, 1998; Summers, Rapoff, Varghese, Porter, & Palmer, 1992), back pain (Nisenzon et al., 2014; Okifuji et al., 1999), and chest pain (Tsouna-Hadjis et al., 1998). Conant (1998) reported that among spinal cord injury CPPs, anger was significantly associated with pain perception, and levels of hostility among those with coronary artery disease also predicted pain intensity ratings (Tsouna-Hadjis et al., 1998).

Research investigating the facets of anger, including anger-in and anger-out, in relation to pain, have produced mixed results (Greenwood, Thurston, Rumble, Waters, & Keefe, 2003). Anger out, or the inclination to externally express anger, has been associated with pain severity, and pain responsiveness in previous investigations of CPPs (Bruehl, Burns, Chung, & Chont, 2009; Burns, 1997; Lombardo, Tan, Jensen, & Anderson, 2005). Anger out is also predictive of increased pain tolerance (Stephens, Atkins, & Kingston, 2009) as well as increased perceptions of control over pain (Graham, Lobel, Glass, & Lokshena, 2008). Anger-in, another facet of
global anger, can be described as suppressed manifestations of anger-based experiences, and has been related to increased pain (Burns et al., 2008), pain interference (Kerns, Rosenberg, & Jacob, 1994), and self-reported depressive symptoms (Achterberg-Lawlis, 1982). Duckro, Chibnall, and Tomazic (1995) found that among chronic posttraumatic headache patients, anger-in and anger out were both directly related to depression, but were only indirectly related to perceived disability via earlier effects on depression. This finding suggests that anger may promote depressive experiences, and that depression is more salient in relation to negative pain outcomes versus anger facets.

BORDERLINE PERSONALITY DISORDER AND NEGATIVE AFFECT

Numerous studies report an association between BPD and depression (Comtois, Cowley, Dunner, & Roy-Byrne, 1999; Perry, 1985; Rogers, Widiger, & Krupp, 1995). Lieb, Zanarini, Schmahl, Linehan, and Bohus (2004) reported that between 41% to 83% of BPD patients endorsed a history of major depression. Comtois and colleagues (1999) found that BPD was significantly related to depression scores among BPD inpatients, and Perry (1985) reported that BPD patients endorsed elevated rates of chronic depression compared to antisocial personality disorder and bipolar I individuals. Comtois and colleagues (1999) found that, compared to individuals with other PD diagnoses, BPD patients demonstrated the greatest levels of self-reported depression. Further, BPD patients who did not meet criteria for a mood disorder rated themselves as more severely depressed compared to non-BPD patients who met criteria for a different PD, major depressive disorder patients, and healthy controls (Comtois et al., 1999).

A relationship between BPD and anxiety has also been demonstrated in the literature. Comtois and colleagues (1999) found that self-reported anxiety was significantly higher among BPD patients compared to patients with other PDs and healthy controls. Snyder and Pitts
(1988) reported that among individuals in inpatient treatment, BPD patients scored higher on measures of anxiety compared to patients meeting diagnostic criteria for dysthymia. Nurnberg, Raskin, Levine, and Polack (1989) also reported a significant association between BPD and anxiety, and found that the combination of BPD and an anxiety disorder resulted in poorer treatment outcomes compared to outpatients meeting criteria for BPD or anxiety disorders only.

BPD and anger are also related, evidenced by the fact that the experience of intense anger is one DSM diagnostic criteria for BPD (APA, 2000). Further, BPD patients report a tendency to experience anger and hostility; anger experiences in such patients were distinct from depressive experiences (Gardner, Leibenluft, O’Leary, & Cowdry, 1991). Baer and Sauer (2011) also reported that BPD features were predictive of anger rumination after controlling for depression, anxiety, and general stress levels.

In addition to depression, anxiety, and anger, anxiety sensitivity (AS) may also be important to the BPD-pain relationship. To date, anxiety sensitivity (AS), a variable significantly related to a variety of pain complaints and outcomes (Ocanez, McHugh, & Otto, 2010), and to BPD (Gratz, Tull, & Gunderson, 2008), has been neglected in research examining the BPD-pain association. Not only has AS been associated with pain, but there is also emerging evidence that BPD patients demonstrate elevated levels of AS (Gratz et al., 2008). More specifically, Gratz and colleagues (2008) found that, among a group of psychiatric outpatients, those who met BPD criteria reported elevated AS levels compared to non-PD patients, even after controlling for the influence of two core features of BPD (i.e., emotional intensity and impulsivity). This relationship was mediated by experiential avoidance.

**CHRONIC PAIN AND ANXIETY SENSITIVITY**

AS is defined as fear of “anxiety-related bodily sensations” as a result of beliefs that
these sensations will result in psychological, social, and physical consequences (Reiss & McNally, 1985). Anxiety sensitivity can be discriminated from trait anxiety, although the two factors are moderately correlated ($r = .30 - .50$; Lilienfeld et al., 1989). This finding is supported by evidence that AS accounts for variance above and beyond trait anxiety in regard to fearful responding (McNally, 1996; McNally & Eke, 1996; Sandin, Chorot, & McNally, 2001) and clinical manifestations of anxiety (Taylor, Koch, & McNally, 1992).

According to the fear-avoidance model of pain (Vlaeyen & Linton, 2000), individuals high in AS will interpret pain signals and potential consequences of those signals as particularly negative (Asmundson, Vlaeyen, & Crombez, 2004). These negative cognitions maintain the experience of pain by promoting hypervigilance toward pain cues and maladaptive pain-related behaviors, such as avoidance of physical activity (Norton & Asmundson, 2004; Vlaeyen & Linton, 2000). These negative pain behaviors are said to result in increased pain-related disability (e.g., decreased ability to walk), which further promotes the experience of negative cognitions regarding pain experiences (Vlaeyen & Linton, 2000). AS has been investigated in both clinical and nonclinical populations (Ocanez et al., 2010). Among clinical pain populations, including those experiencing back pain (Asmundson & Norton, 1995), work-related injuries (Asmundson, Frombach, & Hadjistavropoulos, 1998), headaches (Asmundson, Wright, Norton, & Veloso, 2001; Carranza, 2001; Norton & Asmundson, 2004), non-cardiac chest pain (Raffa, 2006), and mixed pain (Asmundson & Taylor, 1996; Dehghani, Sharp, & Nicholas, 2003; Greenberg & Burns, 2003; Hadjistavropoulos, Asmundons, & Kowalyk, 2004; McCracken, Zayfert, & Gross, 1992; Plehn, Peterson, & Williams, 1998; Zvolensky, Goodie, McNeil, Sperry, & Sorrell, 2000), AS was significantly related to fear of pain ($r = .50$), negative affect ($r = .44$), pain severity ($r =
.24), and disability ($r = .22$; Ocanez et al., 2010). Additionally, Esteve, Ramirez-Maestre, and Lopez-Martinez, (2012) found that AS was related to pain-related fear and avoidance. Among those experiencing non-cardiac chest pain, White, McDonnell, and Gervino (2011) reported that AS was associated with pain-related disability and, for women only, increased health care utilization.

Similar effect sizes were reported for studies conducted with nonclinical pain samples, such as dental patients (Klages, Kianifar, Ulusoy, & Wehrbein, 2006; Sorrell, 2003, women in labor (Lang, Sorrell, Rodgers, & Lebeck, 2006), women with menstrual pain (Sigmon, Dorhofer, Rohan, & Boulard, 2000), and healthy controls who participated in experimental manipulations of pain (Esteve & Camacho, 2008; Ochsner et al, 2006; Thompson, Keogh, French, & Davis, 2007; Vancleef, Peters, Roelofs, & Admudson, 2006). Effect sizes reported for the association between AS and pain-related variables/outcomes among nonclinical samples are as follows: fear of pain, $r = .44$; negative affect, $r = .31$, and pain severity, $r = .18$ (Ocanez et al., 2010). Although mean AS scores are generally slightly higher for female than male samples, gender did not moderate the above relationships (Ocanez et al., 2010; Peterson & Reiss, 1992).

Taken together, the above evidence suggests that AS is an individual vulnerability factor related to the experience of pain and pain-related problems. Additionally, AS contains unique variance above and beyond trait anxiety, suggesting that the association between BPD, AS, and pain and must be tested independently from the effects of trait anxiety on this relationship.

CHAPTER TWO: CURRENT STUDY

Although BPD and pain are associated, there is a lack of research regarding specific, underlying mechanisms that may explain the connection these two phenomena. To date, only
one study has tested what accounts for the BPD-pain association. Tragesser and colleagues (2010) found that negative affect mediated the relation between BPD features and pain complaints. Their measures of negative affect included reports of depression features, trait anxiety, and hostility. The purpose of the current study is to expand on these initial findings regarding BPD, negative affect, and pain.

The current study will be the first investigation of the role of AS in the association between BPD features and chronic pain. The inclusion of AS in examining the relations between BPD, negative affect, and pain is particularly important given the significant association between AS and pain reports and consequences (Ocanez et al., 2010), as well as initial evidence that AS is elevated in BPD patients even when controlling for underlying features of the disorder (Gratz et al., 2008). If AS is a significant predictor of pain outcomes in those with high levels of BPD features, this association may inform treatment of comorbid BPD and chronic pain. Preliminary studies have demonstrated that cognitive-behavioral interventions can reduce AS levels, which is associated with decreased pain outcomes (Broman-Fulks & Storey, 2008; Tull, Schulzinger, Schmidt, Zvolensky, & Lejuez, 2007).

In the current study, it is hypothesized that BPD features and pain-related variables will be related (Dersh et al., 2006; Workman et al., 2002). However, this association will no longer be significant when accounting for negative affect variables. It is also hypothesized that BPD features will be directly associated with measures of negative affect. This hypothesis is consistent with previous studies that have indicated a relation between BPD features and negative affect variables (Comtois et al., 1999; Gardner et al., 1991; Gratz et al., 2008; Perry, 1985). Higher levels of BPD features are predicted to be associated with higher levels of negative affect variables. Negative affect variables, including depression, anger, trait
anxiety, and AS, are also predicted to be show a positive, direct association with pain complaints, pain interference, and pain during activities, even while controlling for other negative affect variables.

CHAPTER THREE: METHOD

PARTICIPANTS

Participants were undergraduate students enrolled in psychology courses at Washington State University; they received course credit for their participation. The sample was composed of 921 (62% female) participants, with an average age of $M(SD) = 19.9 (3.6)$ years old. The sample was primarily White/Non-Hispanic (68.7%); other participant ethnicities were as follows: Asian American = 10.6%; Hispanic American = 9.8%; Other = 6.8%; African American = 3.0%; and Native American = 1.1%. The majority (56.7%) of the sample described their current relationship status as “single.” The study was approved by the Washington State University Institutional Review Board.

MEASURES

Borderline Personality Disorder Features

BPD features were measured using the Personality Assessment Inventory – Borderline Features Scale (PAI-BOR; Morey, 1991). The PAI-BOR contains 24 items that are rated on a scale from 0 (false, not at all true) to 3 (very true). Item responses were summed to create a total score. Sample items from the PAI-BOR include the following: “My mood can shift quite suddenly” and “I worry a lot about other people leaving me.” Morey’s (1991) initial study on the PAI-BOR found that a higher than threshold score (i.e., 37) on the PAI-BOR was associated with clinically significant borderline features; this finding provides support for the criterion-related validity of this measure. In the current study, 12.3% of participants scored above
threshold on the PAI-BOR. This percentage is similar to estimates reported in previous examinations of BPD features among college students (Trull, 1995). Additionally, the PAI-BOR has demonstrated good psychometric properties across multiple investigations (Gardner & Qualter, 2009; Morey, 1991; Trull, 1995). According to George and Mallery’s (2003) guidelines for interpretation of Cronbach’s alpha coefficients, the internal consistency estimate for the PAI-BOR ($\alpha = 0.87$) in the current study can be described as “good.”

**Anxiety Sensitivity**

AS was measured using the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI contains 16 items that are scored on a scale ranging from 0 (very little) to 4 (very much). The ASI has acceptable psychometric properties, with high levels of internal consistency (i.e., $\alpha = .82 - .91$) and test-retest reliability (Peterson & Plehn, 1999). The 16 items (e.g., “It scares me when I feel shaky” and “Unusual body sensations scare me”) were added to calculate a total score. Cronbach’s alpha ($\alpha = 0.88$) for the ASI in the current study fell within the “good” range (George & Mallery, 2003).

**Depression**

The Beck Depression Inventory-II (BDI-II: Beck, Steer, & Brown, 1996) was used as a measure of depressive symptoms. The BDI–II consists of 21 items that assess the intensity of depression in clinically depressed and normal individuals. Each item is a list of four statements (scored from 0 – 3) that are arranged in increasing severity. Sample items include “I do not feel sad,” and “I don’t feel disappointed in myself.” The BDI-II has demonstrated adequate psychometric properties, including a Cronbach’s alpha = .92 (Beck et al., 1996). In the current study, scores for the BDI-II were obtained by summing responses to each of the 21 items; the internal consistency estimate for the BDI-II ($\alpha = 0.89$) fell in the range described as
“good” by George and Mallery (2003).

**Trait Anxiety**

Anxiety was measured using the State-Trait Anxiety Inventory-Trait Scale (STAI; Spielberger, Gorsuch, & Lushene, 1970). This scale contains 20 items that are answered using a 4-point Likert scale, with responses ranging from “almost never” to “almost always.” Participants were asked to respond to items in regard to how they “generally feel.” Sample items include statements such as “I feel nervous” and “I am tense.” The STAI has a reported average $\alpha = .89$, and the STAI-Trait scale demonstrated an average test-retest reliability estimate equal to .88 (Barnes, Harp, & Jung, 2000). In the present study, all items were summed together to create a total score. Current internal consistency ($\alpha = 0.90$) fell within the “good” range (George & Mallery, 2003).

**Anger**

The Behavior Anger Response Questionnaire (BARQ; Linden et al., 2003) was used as a measure of anger. It contains 37 items that are answered using a 5-point Likert scale ranging from “rare” to “very frequently.” Sample responses include items such as “I just keep busy hoping to work off my anger,” and “I hit or push the person who angered me.” Linden and colleagues (2003) reported good consistency ($\alpha = .73 - .75$) and test-reliability ($r = .61 - .85$) estimates for the BARQ. To obtain a BARQ total score, all 37 items were summed together. Cronbach’s alpha ($\alpha = 0.78$) for the BARQ fell within the “adequate” range in the current study (George & Mallery, 2003).

**Pain Severity and Interference**

The Brief Pain Inventory (BPI; Cleeland, 1991) was used to measure pain severity as well as pain-related interference with mood and other aspects of daily functioning. There are four
items that comprise the pain severity score, including: pain at its worst, pain at its least (in the past 3 months), average pain, and current pain (right now). Each of these four items are rated using a 0 (no pain) to 10 (pain as bad as you can imagine); the mean response to the four items was used to derive the overall pain severity score. Pain interference was measured by averaging responses to 7 items rated on a 0 (does not interfere) to 10 (completely interferes) scale. These questions direct participants to rate the extent to which pain has interfered with their functioning in multiple domains. Specific areas of functioning include general activity, walking ability, sleep, mood, relationships, work (housework and work outside of the home), and life enjoyment. Keller and colleagues (2004) reported that among non-cancer pain patients, the BPI demonstrated adequate levels of internal consistency ($\alpha = .73$ - .75) and test-retest reliability ($r = .82$ - .95).

The BPI is also significantly correlated with other measures of pain (Keller et al., 2004). In the present study, internal reliability estimates for the two BPI scales were as follows: pain severity ($\alpha = 0.73$, adequate); and pain interference ($\alpha = 0.88$, good; George & Mallery, 2003).

**Pain During Activities**

A subset of 12 questions of The Pain Standard Evaluation Questionnaire (SEQ Pain; Müller et al., 2008) was used as measure of pain during various activities, such as ascending stairs and walking on a flat surface. Each of the 12 questions are rated using a 7-point scale ranging from “no pain” to “intolerable pain.” Item responses were averaged to derive a composite score. The SEQ Pain demonstrated adequate psychometric properties in a large population-based study of adults in Switzerland (Muller et al., 2008). Cronbach’s alpha ($\alpha = 0.91$) for the SEQ Pain scale in the current study fell within the “excellent”
range (George & Mallery, 2003).

**PROCEDURE**

Participants completed measures in paper-and-pencil format, including a demographic questionnaire, BARQ, BDI-II, STAI, ASI, as well as selected questions from the BPI and SEQ Pain. Study measures were presented to participants in the same order and were embedded into a larger packet of questionnaires.

**CHAPTER FOUR: RESULTS**

**DESCRIPTIVE STATISTICS**

Descriptive statistics were computed using SPSS Version 21 (IBM Corp, 2012). Means, standard deviations, minimum and maximum scores, Cronbach’s alphas, and skew and kurtosis estimates for each measure are provided in Table 1. Bivariate correlations were also computed between measure variables. As reported in Table 2, all correlations were significant ($p < .01$), ranging from $r = 0.19$ to 0.68.

**PATH ANALYSES**

Path analyses were conducted using Mplus Version 6.12 (Muthen & Muthen, 2010). MLR, an estimation procedure that accounts for non-normality, was utilized in each path analysis due to high skew and kurtosis of depression and pain scores (see Table 1; Muthen & Muthen, 2010). Sex was also controlled for in the path analyses. Three path analyses were conducted in which: 1) a single pain variable (i.e., severity, interference, during activities) was regressed onto BPD features; 2) negative affect variables (i.e., AS, depression, anxiety, anger) were regressed onto BPD features; and 3) the pain variable was regressed onto each of the negative affect variables. Additionally, the effects of BPD features on the pain variable through negative affect (i.e., indirect effects) were measured via use of the Mplus model
indirect command in each of the three path analyses (Muthen & Muthen, 2010). See Figure 1 for a pictorial representation of the path analyses.

**Pain Severity**

First, the direct association between BPD features and pain severity was tested. For every one standard deviation (SD) increase on BPD features, pain severity increased by 0.25 SDs, $SE = .01, p < .001$. Next, direct associations between BPD features and negative affect were examined. BPD features demonstrated significant, direct effects with each negative affect variable. For every one SD increase in BPD features: AS increased by 0.38 SDs, $SE = .03, p < .001$; depression increased by 0.63 SDs, $SE = .02, p < .001$; anxiety increased by 0.56 SDs, $SE = .03, p < .001$; and anger increased by 0.27 SDs, $SE = .03, p < .001$.

Then, direct associations between negative affect variables and pain severity were analyzed (see Table 3). AS and depression demonstrated significant, direct associations with pain severity. Direct effects from anxiety to pain severity and anger to pain severity were non-significant.

Given these findings, the effects of BPD features on pain severity through negative affect variables were tested. As reported in Table 4, the direct effect from BPD features to pain severity was no longer significant; the indirect path from BPD features to pain severity through negative affect was significant. Specific, significant indirect effects were detected for AS and depression, but not for anxiety or anger (see Table 5).

**Pain Interference**

First, the direct association between BPD features and pain interference was tested. For every one standard deviation (SD) increase on BPD features, pain interference increased by 0.33 SDs, $SE = .01, p > .001$. Direct associations between BPD features and negative affect
variables are reported above (see Pain Severity). Next, direct associations between, negative affect variables and pain interference were analyzed. Of the negative affect variables measured, AS and depression demonstrated significant, direct associations with pain interference (see Table 3). Direct paths from anxiety to pain interference and from anger to pain interference were non-significant.

Given these findings, the effects of BPD features on pain interference through negative affect were tested. The direct effect from BPD features to pain interference was no longer significant (see Table 4). A significant, indirect effect from BPD features to pain interference was found. Significant, specific indirect effects were found for AS and depression, but not for anxiety or anger (see Table 5).

**Pain During Activities**

First, the direct association between BPD features and pain during activities was tested. For every one standard deviation (SD) increase on BPD features, pain during activities increased by 0.26 SDs, $SE = .00, p < .001$. Direct associations between BPD features and negative affect variables are reported above (see Pain Severity). Next, direct effects from negative affect variables to pain during activities were analyzed. AS, depression, and anger demonstrated significant, direct associations with pain during activities (see Table 3). Anxiety did not demonstrate a significant, direct association with pain during activities.

Given these findings, the effects of BPD features on pain during activities through negative affect variables were tested. The direct effect from BPD features to pain during activities was no longer significant (see Table 4). A significant, indirect effect of BPD features on pain during activities through negative affect was found (see Table 4). Significant, specific indirect effects were found for AS and depression; indirect effects for anxiety and anger were
non-significant (see Table 5).

CHAPTER FIVE: DISCUSSION

As hypothesized, BPD features were significantly and positively related to each negative affect factor, including AS, depression, anxiety, and anger. Two negative affect variables – AS and depression – evidenced significant, direct associations with pain-related factors, even when accounting for effects of the remaining negative affect variables. Although BPD features demonstrated significant, direct effects to pain variables, these effects were no longer significant when indirect effects from BPD features to pain variables through negative affect were considered. Thus, the significant, positive associations between BPD features and each of the pain-related variables – including pain severity, pain interference, and pain during activities – were accounted for by negative affect. That is, the current results demonstrate that the shared variance between BPD features and pain variables was fully explained by negative affect. More specifically, AS and depression – but not trait anxiety or anger – demonstrated significant, indirect effects from BPD features to pain variables, even when the influence of the other negative affect variables was considered. Thus, it can be concluded that AS and depression play a significant role in accounting for the association between BPD features and pain among the current sample.

These findings are consistent with previous literature that has shown a relation between BPD and BPD features and pain variables among both community and clinical samples (e.g., Dersh et al., 2006; Sansone & Sansone, 2012; Workman et al., 2002). The results of the current study provide further evidence of this previously reported BPD-pain association, extended to a non-clinical sample. These findings suggest that conceptualizing BPD from a dimensional perspective may be particularly important, as the BPD-pain relation does not appear to be
unique to individuals who meet (categorical) diagnostic criteria for BPD. The current results provide evidence for the possibility that those with elevated levels of BPD features report increased levels of pain and pain-related interference as a result of experiencing negative affect. More specifically, BPD features may be promoting or exacerbating the presence of negative affect in such individuals, which in turn amplifies the perception of pain.

The association between BPD features and pain was no longer significant when accounting for negative affect variables; this finding replicates Tragesser and colleagues’ (2010) results, in which negative affect explained the BPD-pain association among a sample of pain patients. Further, the current findings are consistent with theoretical perspectives and research evidence demonstrating the importance of affective experiences as a core component of BPD (Linehan, 1993; New & Siever, 2002; Siever & Davis, 1991; Tragesser & Robinson, 2009). The current results suggest that negative affect components, specifically AS and depression, may potentially be important treatment targets in populations with both significant BPD features and chronic pain.

Adding to Tragesser and colleagues’ (2010) findings in which depression, anxiety, and anger explained the BPD-pain relation, the present study was the first to investigate the effect of AS in the association between BPD features and pain-related variables. For each pain-related factor, including pain severity, pain interference, and pain during activities, AS demonstrated significant, indirect effects in explaining the relation between these factors and BPD features. AS standardized indirect path coefficients ranged from 0.05 to 0.08, $p < .001$, showing an overall significant (albeit small in magnitude) effect of AS in the BPD-pain relation, above and beyond the influence of other negative affect variables. In contrast, trait anxiety did not demonstrate significant direct effects on pain variables or indirect effects in any of the BPD-
pain relations. Although trait anxiety and AS are related constructs (e.g., Lilienfeld et al., 1989), they appear to be distinct factors, of which AS appears to be more important in understanding the BPD-anxiety-pain association.

The role of AS in BPD has been previously explored (Gratz et al., 2008); specifically, elevated levels of AS were demonstrated among BPD outpatients versus non-PD outpatients. This association was mediated by experiential avoidance. AS has been conceptualized as being related to increased pain via efforts to escape and avoid the experience of pain symptoms. Individual high in AS often interpret pain and pain-related cues as particularly negative and harmful (fear-avoidance model of pain; Vlaeyen & Linton, 2000). Such escape and avoidance techniques (e.g., stopping physical activity) not only contribute to increased pain itself (e.g., muscle atrophy) but also reinforce catastrophic perceptions of pain, promoting continued escape and avoidance. There is evidence that BPD is specifically characterized by experiential avoidance (see Chapman, Dixon-Gordon, & Walters, 2011 for a review), potentially due to underlying emotional vulnerabilities, including difficulties managing emotions and intolerance of distress. Thus, it could be that AS is reflective of this emotion-related diathesis in BPD, in which anxiety-related experiences are both prominent and managed maladaptively (e.g., avoided).

The association between BPD and substance use is well established (Grant et al., 2008; Trull, Jahng, Tomko, Wood, & Sher, 2010; Trull, Sher, Minks-Brown, Durbin, & Burr, 2000), and evidence of use of psychoactive substances as an avoidant coping strategy in BPD has been demonstrated (Chapman & Cellucci, 2007; Kruekelbach, McCormick, Schulz, & Grueneich, 1993). More specifically, BPD features have been associated with a number of prescription opioid-related variables, including increased quantity, frequency, risk for misuse,
as well as consequences and dependence features (Tragesser et al., 2013). One possibility is that, among individuals demonstrating significant levels of BPD features, prescription opioids may be misused as a means to avoid the experience of pain (versus as a way to manage pain symptoms). Future studies are needed to test such a hypothesis.

Similar to trait anxiety, anger did not demonstrate significant direct effects on pain variables or indirect effects in any of the BPD-pain relations. This null result may be reflective of mixed findings regarding the association between anger and pain when anger subfacets (e.g., anger-in, anger-out) are considered (Greenwood et al., 2003), as well as evidence of the mediating effect of depression in explaining the relation between anger and pain-related disability (Duckro et al., 1995). As such, it may be that anger (as well as trait anxiety) are potentially less important components to target in BPD-pain populations, at least compared to AS and depression.

Consistent with study hypotheses, BPD features were directly associated with each measured negative affect factor. Standardized path coefficients from BPD features to negative affect variables were all significant and substantial, ranging from 0.27 (anger) to 0.63 (depression), $p < .001$. Such findings are in line with previous research demonstrating a significant relation between AS, depression, anxiety, and anger with BPD (Baer & Sauer, 2011; Comtois et al., 1999; Gratz et al., 2008) as well as theoretical and empirical evidence of affective experiences as core components of BPD (Linehan, 1993; New & Siever, 2002; Siever & Davis, 1991; Tragesser & Robinson, 2009). Depression evidenced the most substantial direct path coefficient from BPD features to negative affect, which is consistent with high comorbidity rates (i.e., between 41% to 83%) of BPD and major depression (Lieb et al., 2004).

The current study has a variety of strengths and limitations. Although it was found that
the shared association between BPD features and pain could be fully explained by negative affect, the cross-sectional design of the study precludes interpretations of causality between study variables. For instance, the current study cannot determine if BPD features casually predict pain-related outcomes or vice versa. Additional components, including longitudinal study designs, replication of findings across independent samples, and manipulation of study variables, should be incorporated into future studies of BPD and pain in order to flesh out the directionality of the associations between BPD features and pain. The current study’s large sample size is a strength. In contrast, the majority of participants were Caucasian females and the sample was composed exclusively of college students. Use of more diverse (and clinical) samples in future investigations will help to determine if the current pattern of findings are generalizable to other populations.

Although negative affect fully explained the variance in BPD-pain associations, the associations between BPD features and pain variables were small in magnitude among the current sample. It may be that larger associations between BPD and pain may be found in clinical populations, such as those seeking treatment for chronic pain. The current findings also lend support for future investigations of the potential clinical utility of addressing negative affect (e.g., teaching skills to facilitate adaptive coping with emotional experiences) in individuals presenting with both BPD features and pain complaints.

In sum, among a sample of college students, significant, positive associations between BPD features and each measured pain-related variable – including pain severity, pain interference, and pain during activities – were fully accounted for by indirect effects of negative affect. Negative affect variables, including AS and depression– but not trait anxiety or anger – demonstrated significant, indirect effects from BPD to pain variables above and
beyond the influence of the other negative affect variables. Study findings are consistent with existing literature, further adding to the current empirical evidence of BPD-pain relations by expanding such findings to a non-clinical sample and by examining particular effects of AS in the relation between BPD features and pain experiences.
REFERENCES


36


<table>
<thead>
<tr>
<th>Construct/Measure</th>
<th>M(SD)</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Cronbach’s α</th>
<th>Skew(SE)</th>
<th>Kurtosis(SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Borderline Personality Disorder</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD Features (PAI-BOR)</td>
<td>24.1 (10.6)</td>
<td>3.0</td>
<td>60.0</td>
<td>0.87</td>
<td>0.54 (.08)</td>
<td>-0.04 (.16)</td>
</tr>
<tr>
<td><strong>Negative Affect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety Sensitivity (ASI)</td>
<td>20.5 (10.8)</td>
<td>0.0</td>
<td>64.0</td>
<td>0.88</td>
<td>0.69 (.08)</td>
<td>0.05 (.16)</td>
</tr>
<tr>
<td>Depression (BDI-II)</td>
<td>9.1 (7.7)</td>
<td>0.0</td>
<td>49.0</td>
<td>0.89</td>
<td>1.38 (.08)</td>
<td>2.29 (.17)</td>
</tr>
<tr>
<td>Anxiety (STAI)</td>
<td>37.7 (11.2)</td>
<td>20.0</td>
<td>76.0</td>
<td>0.90</td>
<td>0.52 (.08)</td>
<td>-0.12 (.17)</td>
</tr>
<tr>
<td>Anger (BARQ)</td>
<td>105.4 (14.8)</td>
<td>60.0</td>
<td>148.0</td>
<td>0.78</td>
<td>-0.02 (.08)</td>
<td>0.09 (.16)</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity (BPI)</td>
<td>2.8 (1.6)</td>
<td>0.0</td>
<td>10.0</td>
<td>0.73</td>
<td>0.63 (.08)</td>
<td>0.91 (.16)</td>
</tr>
<tr>
<td>Interference (BPI)</td>
<td>2.7 (2.3)</td>
<td>0.0</td>
<td>16.3</td>
<td>0.88</td>
<td>1.08 (.08)</td>
<td>1.59 (.16)</td>
</tr>
<tr>
<td>During Activities (SEQ Pain)</td>
<td>2.2 (1.1)</td>
<td>0.0</td>
<td>8.8</td>
<td>0.91</td>
<td>1.42 (.08)</td>
<td>2.65 (.16)</td>
</tr>
</tbody>
</table>

*Note. n = 921; PAI-BOR = Personality Assessment Inventory-Borderline Features Scale (Morey, 1991); ASI = Anxiety Sensitivity Index (Peterson & Reiss, 1992); BDI-II = The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); STAI = State-Trait Anxiety Inventory-Trait Scale (Spielberger, Gorsuch, & Lushene, 1970); BARQ = Behavior Anger Response Questionnaire (Linden et al., 2003); BPI = The Brief Pain Inventory (Cleeland, 1991); SEQ Pain = The Pain Standard Evaluation Questionnaire (Müller et al., 2008).*
Table 2

Bivariate Correlations

<table>
<thead>
<tr>
<th></th>
<th>PAI-BOR</th>
<th>ASI</th>
<th>BDI-II</th>
<th>STAI</th>
<th>BARQ</th>
<th>BPI (S)</th>
<th>BPI (I)</th>
<th>SEQ Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAI-BOR</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td><strong>0.38</strong></td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td><strong>0.63</strong></td>
<td><strong>0.46</strong></td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAI</td>
<td><strong>0.56</strong></td>
<td><strong>0.43</strong></td>
<td><strong>0.68</strong></td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BARQ</td>
<td><strong>0.27</strong></td>
<td><strong>0.36</strong></td>
<td><strong>0.27</strong></td>
<td><strong>0.22</strong></td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPI (Severity)</td>
<td><strong>0.25</strong></td>
<td><strong>0.31</strong></td>
<td><strong>0.35</strong></td>
<td><strong>0.28</strong></td>
<td><strong>0.19</strong></td>
<td>--</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPI (Interference)</td>
<td><strong>0.32</strong></td>
<td><strong>0.35</strong></td>
<td><strong>0.42</strong></td>
<td><strong>0.32</strong></td>
<td><strong>0.20</strong></td>
<td><strong>0.57</strong></td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>SEQ Pain</td>
<td><strong>0.26</strong></td>
<td><strong>0.27</strong></td>
<td><strong>0.34</strong></td>
<td><strong>0.22</strong></td>
<td><strong>0.20</strong></td>
<td><strong>0.51</strong></td>
<td><strong>0.54</strong></td>
<td>--</td>
</tr>
</tbody>
</table>

Note. \( n = 921 \); PAI-BOR = Personality Assessment Inventory-Borderline Features Scale (Morey, 1991); ASI = Anxiety Sensitivity Index (Peterson & Reiss, 1992); BDI-II = The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); STAI = State-Trait Anxiety Inventory-Trait Scale (Spielberger, Gorsuch, & Lushene, 1970); BARQ = Behavior Anger Response Questionnaire (Linden et al., 2003); BPI = The Brief Pain Inventory (Cleeland, 1991); SEQ Pain = The Pain Standard Evaluation Questionnaire (Müller et al., 2008).

* \( p < .05 \).  ** \( p < .01 \).  *** \( p < .001 \).
Table 3

Direct Effects of Negative Affect on Pain

<table>
<thead>
<tr>
<th></th>
<th>BPI Severity</th>
<th>BPI Interference</th>
<th>SEQ Pain During Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASI</td>
<td>0.17 (.04)</td>
<td>0.21 (.04)</td>
<td>0.15 (.04)</td>
</tr>
<tr>
<td>BDI-II</td>
<td>0.22 (.06)</td>
<td>0.28 (.05)</td>
<td>0.20 (.06)</td>
</tr>
<tr>
<td>STAI</td>
<td>0.04 (.05)</td>
<td>0.02 (.05)</td>
<td>-0.01 (.05)</td>
</tr>
<tr>
<td>BARQ</td>
<td>0.06 (.04)</td>
<td>0.04 (.04)</td>
<td>0.08 (.04)</td>
</tr>
</tbody>
</table>

Note. n = 921; ASI = Anxiety Sensitivity Index (Peterson & Reiss, 1992); BDI-II = The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); STAI = State-Trait Anxiety Inventory-Trait Scale (Spielberger, Gorsuch, & Lushene, 1970); BARQ = Behavior Anger Response Questionnaire (Linden et al., 2003); BPI = The Brief Pain Inventory (Cleeland, 1991). SEQ Pain = The Pain Standard Evaluation Questionnaire (Müller et al., 2008). Path loadings are completely standardized. Standard error estimates are in parentheses. *p < .05. **p < .01. ***p < .001.
Table 4

*Effect Decomposition Table*

<table>
<thead>
<tr>
<th></th>
<th>BPI Severity</th>
<th>BPI Interference</th>
<th>SEQ Pain During Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PAI-BOR</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct Effect</td>
<td>0.01 [-.08 - .10]</td>
<td>0.07 [-.01 - .15]</td>
<td>0.07 [-.01 - .16]</td>
</tr>
<tr>
<td>Indirect Effect</td>
<td>0.24 [.17 - .31]***</td>
<td>0.27 [.21 - .33]***</td>
<td>0.19 [.13 - .26]***</td>
</tr>
<tr>
<td>Total Effect</td>
<td>0.25 [.19 - .31]***</td>
<td>0.34 [.28 - .40]***</td>
<td>0.26 [.20 - .32]***</td>
</tr>
</tbody>
</table>

*Note. n = 921; PAI-BOR = Personality Assessment Inventory-Borderline Features Scale (Morey, 1991); ASI = Anxiety Sensitivity Index (Peterson & Reiss, 1992); BDI-II = The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); STAI = State-Trait Anxiety Inventory-Trait Scale (Spielberger, Gorsuch, & Lushene, 1970); BARQ = Behavior Anger Response Questionnaire (Linden et al., 2003); BPI = The Brief Pain Inventory (Cleeland, 1991). Path loadings are completely standardized. Bootstrap 95% confidence intervals are in brackets. *p < .05. **p < .01. ***p < .001.*
### Table 5

*Specific Indirect Effects from Borderline Personality Disorder Features to Pain Through Negative Affect*

<table>
<thead>
<tr>
<th></th>
<th>BPI Severity</th>
<th>BPI Interference</th>
<th>SEQ Pain During Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASI</td>
<td>0.06 [.03 - .10]</td>
<td>0.08 [.05 - .11]</td>
<td>0.05 [.02 - .09]</td>
</tr>
<tr>
<td>BDI-II</td>
<td>0.14 [.07 - .21]</td>
<td>0.17 [.11 - .24]</td>
<td>0.13 [.05 - .20]</td>
</tr>
<tr>
<td>STAI</td>
<td>0.02 [-.03 - .07]</td>
<td>0.01 [-.05 - .06]</td>
<td>-0.01 [-.07 - .05]</td>
</tr>
<tr>
<td>BARQ</td>
<td>0.02 [.00 - .04]</td>
<td>0.01 [-.01 - .03]</td>
<td>0.02 [.00 - .04]</td>
</tr>
</tbody>
</table>

*Note. n = 921; ASI = Anxiety Sensitivity Index (Peterson & Reiss, 1992); BDI-II = The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); STAI = State-Trait Anxiety Inventory-Trait Scale (Spielberger, Gorsuch, & Lushene, 1970); BARQ = Behavior Anger Response Questionnaire (Linden et al., 2003); BPI = The Brief Pain Inventory (Cleeland, 1991). SEQ Pain = The Pain Standard Evaluation Questionnaire (Müller et al., 2008). Path loadings are completely standardized. Bootstrap 95% confidence intervals are in brackets. *p < .05. **p < .01. ***p < .001.*
Figure 1. Proposed path analyses. For each analysis, a single pain variable was regressed onto BPD features. Each negative affect variable was also regressed onto BPD features. The pain variable was regressed onto each negative affect variable. Indirect effects of BPD features on the pain variable through negative affect variables were tested.