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Examining Emotional Pain among Individuals with Chronic Physical Pain: nomothetic and idiographic approaches

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Examining Emotional Pain among Individuals with Chronic Physical Pain: nomothetic and idiographic approaches

by

Madelyn R. Frumkin

A thesis presented to
The Graduate School
of Washington University in
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Washington University in St. Louis

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ABSTRACT OF THE THESIS

Examining Emotional Pain among Individuals with Chronic Physical Pain: nomothetic and idiographic approaches

by

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Masters of Arts in Psychology

Washington University in St. Louis, 2019

Associate Professor Thomas Rodebaugh, Chair

Pain is often conceptualized as an experience that is both physical and emotional. These two components are often difficult to distinguish, which may contribute to the experience of chronic physical pain without an apparent physical cause. In the current two studies, I sought to examine whether emotional pain is associated with physical pain severity for individuals with chronic pain. Emotional pain and the more specific experiences of psychological and social pain have been defined as the experience of pain affect in response to non-physical stimuli (i.e., thwarted belongingness, loss, social rejection). In Study 1, I found that emotional and psychological pain were significantly positively correlated with physical pain intensity among individuals with chronic pain. In Study 2, I used an idiographic approach to examine whether emotional and physical pain can be conceptualized as distinct constructs that prospectively predict one another for individuals with chronic pain. In two individuals, I found that emotional and physical pain did represent distinct factors; however, neither predicted the other over three-hour lags. Implications for further idiographic and nomothetic research are discussed.
Introduction

According to the International Association for the Study of Pain, “[Pain] is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience” (Merskey & Bogduk, 1986, p. S217). This definition captures the distinction between two components of pain: the sensory component (i.e., the location, quality, and intensity of the physical experience) and the affective component (i.e., the unpleasant emotional response; Price, 2000). If two people are subject to the same stimulus and only the second person experiences an unpleasant emotional response, it can be said that only the second person experienced pain. On the other hand, consider a person who experiences an unpleasant emotional response without an apparent sensory cause. When the affective component is present, the individual is experiencing pain, though perhaps not of a physical nature. As Engel (1969, p. 45) proposed, “The idea of injury as well as the need to suffer may lead to pain, just as may a real lesion or injury.”

Nearly one-third of individuals in the United States report chronic pain, or persistent pain that lasts three to six months or more (Johannes, Le, Zhou, Johnston, & Dworkin, 2010; Treede et al., 2015). A primary difficulty in treating chronic pain is that the cause of persistent physical pain is often unclear. Approximately one-third of individuals with chronic pain report no diagnosed pain condition, and some of the most common diagnoses (i.e., lower back pain, migraine headache) offer a label for the symptoms rather than an etiological explanation (Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2005). It is due to this common absence of “real lesion or injury” (Engel, 1969, p. 45) that I suggest another source of chronic pain: emotional pain.
Emotional pain has been defined as the presence of pain affect in response to emotional rather than physical experiences (MacDonald, 2009). One subset of emotional pain known as social pain refers to emotional responses specific to damaged social relationships (i.e., rejection, death of a loved one, the breakup of an important relationship; MacDonald & Leary, 2005). Social pain has recently received significant attention, particularly in the neuroimaging field. Multiple studies have shown that eliciting social exclusion is associated with activation in the dorsal anterior cingulate cortex and anterior insula, regions also implicated in sensory pain perception (Eisenberger & Lieberman, 2004a; Fisher, Brown, Aron, Strong, & Mashek, 2010; Kross, Berman, Mischel, Smith, & Wager, 2011). As a result of these shared biological mechanisms, it has been posited that pain affect evolved to signal threats to one’s social connectedness, in addition to potential sources of physical injury (Eisenberger & Lieberman, 2004b; Macdonald & Leary, 2005). From an evolutionary perspective, avoiding social exclusion and seeking important partnerships is perhaps as adaptive as avoiding physical harm.

Psychological pain is a second subset of emotional pain that has received significant research attention, particularly as it is related to suicidality. Through case studies and observations, suicide researcher Edwin Shneidman developed the concept of psychological pain, or psychache, to describe intense affective experiences of “hurt, anguish, or ache that takes hold of the mind” (Shneidman, 1998, p. 13). Shneidman posited that psychological pain arises from unmet psychological needs, and that suicidal thoughts and behavior emerge when death is seen as a means of escape from unbearable psychological pain (Shneidman, 1993, 1998). Consistent with this theory, self-reported psychological pain is more strongly associated with suicidal thoughts, motivation, preparation, and attempter status than are other well-established suicide risk factors, such as depression, hopelessness, and perfectionism (Flamenbaum & Holden, 2007;
Holden, Mehta, Cunningham, & McLeod, 2001; Troister & Holden, 2010). Furthermore, change in psychological pain over time is associated with change in suicidal thoughts and preparation for suicide (Troister, Davis, Lowndes, & Holden, 2013).

In the chronic pain field, the constructs of emotional pain, social pain, and psychological pain have received little attention. This is perhaps surprising given not only the overlap in terminology, but also the fact that suicide risk is at least doubled in chronic pain patients (Tang & Crane, 2006). The Multidimensional Affect and Pain Survey (MAPS; Knotkova et al., 2006) includes an emotional pain subscale; however, this subscale includes a range of affective experiences such as depressed mood, self-blame, anger, anxiety and fear. I argue that emotional pain as defined in the prior literature is more specific. Here, I define emotional pain, social pain, and psychological pain as affective experiences of pain stemming from social or psychological causes. Indeed, prior factor analyses have shown that psychological pain is distinct from depression, including amongst undergraduates with chronic pain (Troister & Holden, 2013; Frumkin et al., under review). Given the increased risk of suicide and biological overlaps in physical and emotional pain experiences, an investigation of these specific constructs is warranted among individuals with chronic pain.

In this article, I examine emotional pain among individuals seeking treatment for chronic pain. In Study 1, I examine the relationship between severity of physical pain and self-reported emotional pain, psychological pain, and rejection sensitivity at the group level. In Study 2, I take a personalized medicine approach to examining the role of emotional pain in chronic pain syndromes. Using intensive longitudinal data and idiographic modeling techniques, I examine the correlational and prospective relationships among physical pain, emotional pain, depression, and social connectedness for individual patients with chronic pain.
Study 1: a nomothetic approach

In Study 1, I sought to gain a better understanding of how emotional pain is related to chronic physical pain and related symptoms. As mentioned previously, the only measure of emotional pain that has been used repeatedly in the chronic pain literature refers broadly to negative emotional experiences, rather than the specific experience of pain affect in response to emotional stimuli. Thus, I sought to assess whether tendency to experience pain affect in response to emotional stimuli is associated with physical pain severity, tendency to catastrophize about pain, psychosocial illness impact, and rejection sensitivity.

Based on my theory that individuals with chronic pain may be experiencing pain affect in response to both emotional and physical stimuli, I hypothesized that emotional and psychological pain would be positively correlated with physical pain severity, as well as with psychosocial illness impact. Furthermore, due to previous theories that the social pain subset of emotional pain is related to social rejection, I also hypothesized that rejection sensitivity would be positively correlated with emotional and psychological pain severity. I thought this relationship would be stronger than a potential positive relationship between rejection sensitivity and physical pain severity.

Finally, I collected a measure of pain catastrophizing, which has been shown to be one of the most robust psychological predictors of increased pain and impairment (Gatchel, Peng, Peters, Fuchs, & Turk, 2007; Sullivan, Bishop, & Pivik, 1995). I hypothesized that pain catastrophizing would be positively correlated with emotional and psychological pain severity in addition to physical pain severity. I also tested pain catastrophizing as a moderator of the relationship between emotional or psychological pain and physical pain intensity. I hypothesized
that individuals with a greater tendency to catastrophize about pain would exhibit a stronger relationship between physical and emotional pain.

Method

Participants

Participants were recruited from the Washington University Pain Center. For a four-month period, all individuals with scheduled appointments were contacted via their contact information listed in the electronic medical record. Individuals with a listed email address were emailed, and those without an email address were contacted by phone. In order to be eligible for participation, individuals were required to be over the age of 18, report chronic physical pain for at least the past 3 months, and report pain on more days than not with an average intensity of 3 or greater on a 0-10 scale. These inclusion criteria were included as screening items at the beginning of the online survey. Potential participants were also excluded for any linguistic or cognitive issue that prevented full participation in the study and completion of study measures.

The sample consisted of 65 individuals with a mean age of 56.84 years (SD = 13.72). The sample was primarily female (66%, n = 43) and White (89%, n = 58). The sample had low diversity, with 7 participants (11% of the sample) identifying as Black or African American and 1 individual identifying as Hispanic or Latinx. The majority of participants (55%, n = 36) had completed 4-year college or above and were married (62%, n = 40).

Procedure

All research activities were approved by the Washington University in St. Louis Institutional Review Board. Individuals who were contacted via email received a link to a Qualtrics survey in the email. After viewing a consent letter, individuals could choose whether or not to continue to the survey. Individuals who were contacted via phone were asked for an email
address in order to receive the same survey link. All study measures were completed online via Qualtrics. No compensation was provided.

Measures

**Physical and emotional pain severity.** Pain severity was assessed using the Patient-Reported Outcomes Measurement Information System (PROMIS) Pain Intensity scale – short form (Cella et al., 2010). PROMIS measures were developed by the National Institutes of Health to precisely and efficiently measure patient-reported outcomes. The Pain Intensity scale – short form includes three items assessing worst pain intensity in the past week, average pain intensity in the past week, and current pain intensity on a scale from 1 (*Had no pain*) to 5 (*Very severe*).

In the current study, I assessed both physical and emotional pain using this scale. Prior to completing the physical pain and emotional pain scales, participants viewed a paragraph instructing them to consider physical experiences (i.e., headache, tooth pain, joint or muscle pain) or emotional experiences (i.e., feeling rejected, lonely, or socially isolated), respectively. A raw score for each was then calculated by summing responses to the three items. Internal consistency in the current study was good (α = .81 and .89 for physical and emotional pain, respectively).

**Psychological pain.** Psychological pain was assessed using the Psychache Scale (PAS; Holden, Mehta, Cunningham, & McLeod, 2001). The PAS assesses 9 items related to frequency of psychologically painful experiences (i.e., *I seem to ache inside*) using a 5-point Likert scale ranging from 1 (*Never*) to 5 (*Always*) and 4 items related to severity of psychological pain (i.e., *I can’t take my pain any more*) on a scale from 1 (*Strongly disagree*) to 5 (*Strongly agree*). The PAS has indicating excellent internal consistency and convergent validity with measures of suicidality and self-injury (Troister, Davis, Lowndes, & Holden, 2013; Troister & Holden, 2013).
The PAS has also demonstrated an invariant factor structure across undergraduates with and without chronic pain, indicating its potential utility as a measure of psychological pain among individuals with chronic pain (Frumkin et al, under review). Internal consistency in the current study was excellent ($\alpha = .93$).

**Psychosocial illness impact.** Psychosocial illness impact was also assessed using a PROMIS scale. The Psychosocial Illness Impact-Negative–Short Form 4a asks participants to rate to what degree they feel worthless, disconnected from others, worried about the future, and like their life lacks meaning. For each question, participants rate how much they felt these things before the illness and now, since the illness on a scale from 1 (*Not at all*) to 5 (*Very much*). A raw score was calculated by summing all responses for each participant. Internal consistency in the current study was acceptable ($\alpha = .77$).

**Pain catastrophizing.** The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) was used to assess the nature of thoughts about physical pain. The PCS was developed to assess three related components of catastrophizing: rumination (i.e., *I can’t stop thinking about how much it hurts*), magnification (i.e., *I worry that something serious may happen*), and helplessness (i.e., *There is nothing I can do to reduce the intensity of the pain*). Participants rate each item on a 5-point Likert scale ranging from 0 (*Not at all*) to 4 (*All the time*). The PCS has been used in several studies assessing perceptions of pain and has demonstrated excellent internal consistency in community and pain outpatient samples (Osman et al., 1997, 2000). Internal consistency in the current study was excellent ($\alpha = .95$).

**Rejection sensitivity.** The Rejection Sensitivity-Adult Questionnaire (RSQ; Berenson et al., 2009) was used to assess sensitivity to rejection across a range of hypothetical social situations. For 9 scenarios, participants rated on two domains: a) how concerned or anxious they
would be on a scale from 1 (Very unconcerned) to 7 (Very concerned), and b) how likely it is that the other person would be receptive on a scale from 1 (Very unlikely) to 7 (Very likely)\(^1\). For example, one scenario is asking a family member or friend for a loan. The participant would rate how concerned or anxious they would be that their family would not want to help them, as well as how likely they think it is that their family would help them. A score for each scenario is created by reverse-scoring Part B and multiplying this score by the rating for Part A. Then, a total score is generated by taking the mean of the resulting 9 scores for each participant. The RSQ has demonstrated acceptable internal consistency and test-retest reliability (Berenson et al., 2009; Downey & Feldman, 1996). Internal consistency in the current study was acceptable (\(\alpha = .76\)).

**Missing data**

Multiple imputation was conducted using the mice package in R version 3.5.1 (Buuren & Groothuis-Oudshoorn, 2015). The primary source of missing data was the RSQ, which was completed in full by 66\% of participants (\(n = 43\)).

**Results**

**Pain characteristics**

Duration of chronic pain in the current sample ranged from 5 months to 62 years (\(M = 12.58\) years, \(SD = 11.87\) years). Most participants (92\%) reported experiencing pain 7 days per week on average. On a scale of 0 (No pain) to 10 (Worst pain imaginable), the average pain severity in the past week was 6 (\(SD = 2\)). The most commonly reported pain locations were lower back (71\%), neck (43\%), knees (37\%), and hips (37\%). Most participants reported pain in more than one location (\(n = 59; 91\%)\).

\(^1\) The RSQ is intended to be assessed on a 1-6 scale. An extra response option was inadvertently added in this study. Therefore, means should not be compared to previous studies.
Relationships among study variables

Means, standard deviations, and relationships among study variables are presented in Table 1. Consistent with our hypotheses, the modified PROMIS form for emotional pain intensity and the PAS were both significantly positively correlated with physical pain intensity ($r's = .51$ and $.47$, respectively, $p's < .001$). Psychosocial illness impact was significantly positively correlated with psychache ($r = .67$, $p < .0001$) and emotional pain intensity ($r = .27$, $p = .041$), but not physical pain intensity ($r = .20$, $p = .122$). Rejection sensitivity was significantly positively correlated with psychache ($r = .32$, $p = .015$), but not emotional pain intensity ($r = .20$, $p = .209$) or physical pain intensity ($r = .13$, $p = .348$).

Consistent with previous studies, pain catastrophizing was significantly positively correlated with physical pain severity ($r = .47$, $p < .001$). Pain catastrophizing was also significantly positively correlated with emotional pain severity ($r = .36$, $p = .007$) and psychache ($r = .76$, $p < .0001$). I then tested pain catastrophizing as a moderator of the relationship between emotional and physical pain severity. In a multiple regression analysis, emotional pain severity and pain catastrophizing were both significant predictors of physical pain severity [$\beta = .26$ and $.04$, respectively; $F(3, 55) = 11.16$, $p < .001$, $R^2 = .38$]. However, the interaction between emotional pain severity and pain catastrophizing was not significant ($\beta = .006$, $p = .208$). Thus, it appears that individuals with a greater tendency to catastrophize reported significantly more physical pain on average, controlling for emotional pain severity. Although individuals with a greater tendency to catastrophize appeared to have a stronger relationship between emotional and physical pain than those with a lower tendency to catastrophize (see Figure 1), pain catastrophizing was not a significant moderator of the relationship between emotional and physical pain.
Discussion

In the current study, I found that emotional and psychological pain were significantly positively correlated with physical pain intensity among individuals with chronic pain. Although no firm conclusions can be drawn from these cross-sectional results, our finding suggests that emotional and psychological pain may be part of chronic pain syndromes. What remains to be tested is whether pain affect in response to emotional experiences is interpreted as physical pain affect, thereby exacerbating the experience of chronic pain.

Importantly, psychache was significantly strongly correlated with psychosocial illness impact, whereas physical pain intensity was not. One might expect that more severe physical pain would be associated with greater psychosocial impacts; however, previous studies have shown that beliefs about pain tend to impact functioning more strongly than the severity of physical pain (Jensen, Turner, Romano, & Karoly, 1991; Turk & Okifuji, 2002). For example, acceptance of pain predicts better functioning, even after controlling for perceived pain intensity (McCracken, 1998). Our finding that psychological pain may also predict psychosocial functioning more strongly than intensity of physical pain adds to these findings, perhaps suggesting an additional target for the psychological treatment of chronic pain.

Pain catastrophizing has also been repeatedly found to exacerbate physical pain among chronic pain patients (Gatchel et al., 2007; Sullivan et al., 1995). In the current study, I found that the tendency to catastrophize about pain is also associated with greater emotional and psychological pain. This suggests that similar mechanisms may perpetuate both physical and emotional pain, namely the tendency to catastrophize both types of pain affect. Furthermore, pain catastrophizing has previously been found to be a significant predictor of suicidality among individuals with chronic pain (Edwards, Smith, Kudel, & Haythornthwaite, 2006; Tang & Crane,
Although psychological pain has also been strongly linked with suicide (Verrocchio et al., 2016), this relationship has not yet been tested among individuals with chronic pain. Given that suicide risk is increased two-fold among chronic pain patients, a more thorough investigation of the relationships among pain catastrophizing, emotional or psychological pain, and suicidality is warranted. For instance, based on the trends in the current data, pain catastrophizing may have a small to moderate effect on the relationship between physical and emotional pain, which may be detected with a larger sample size.

In addition to examining emotional and psychological pain, I also sought to test theories of social pain through associations between rejection sensitivity and physical, emotional, and psychological pain. Consistent with our hypothesis, rejection sensitivity was significantly positively correlated with psychache, but not physical pain intensity. Notably, the relationship between rejection sensitivity and emotional pain intensity was also not significant. I consider these findings preliminary, primarily because the RSQ had the most cases of missing data, which required multiple imputation. Missing data likely occurred because the RSQ includes items geared towards college students, such as bringing up the issue of sexual protection with your significant other. Many of the participants in the current study skipped one or more items of the RSQ, presumably because these scenarios did not apply to them. Thus, the associations between rejection sensitivity and physical and emotional pain should be examined using a more suitable measure of rejection sensitivity or social pain.

Study 1 provided a broad overview of the relationship between emotional or psychological pain and physical pain among chronic pain patients. A strength of the current study is the clinical sample of individuals seeking treatment for chronic pain, as these research questions have not previously been addressed in this population. However, a significant
The weakness of the current study is a lack of diversity in our sample. Although all patients at the Pain Center were invited to participate, it is possible, although not known to be true, that participants were more likely to respond to the survey request if they were white. Reasons for non-participation among diverse individuals should be investigated and addressed for more representative research.

Study 1 also faces several statistical limitations. Correlation matrices appear to be a simple explanation of how various measures are related to one another; however, they can be highly impacted by factors such as measurement overlap and measurement error. More broadly, cross-sectional research faces the limitation of only yielding results in reference to the group on average, rather than individual patients. There has been a growing realization in the field of psychology that what is true at the group level may not be true for most or even any individuals (Fisher, Medaglia, & Jeronimus, 2018; Molenaar, 2004). For these reasons, I now turn to statistical tools that allow us to better assess the relationship between emotional and physical pain for individual patients.

**Study 2: an idiographic approach**

In Study 1, I took a nomothetic approach to examining the relationship between emotional or psychological pain severity and physical pain severity among chronic patients. Nomothetic approaches are useful because they provide information about a group on average. I found that greater emotional or psychological pain is associated with greater physical pain, a finding that I would expect to generalize to other groups of chronic pain patients. The vast majority of modern psychological research uses a nomothetic approach, as this approach allows researchers to draw conclusions about large groups of people with relatively little data from each individual person.
Although nomothetic research is intended to answer questions about groups of people, findings are often assumed to apply to individual patients. For example, I might assume that because emotional and physical pain had a significant positive relationship in Study 1, the same should be true within individuals over time. However, Fisher et al. (2018) and others have shown that group-level relationships often do not generalize to individual participants. This problem represents the ecological fallacy: the false notion that conclusions drawn about groups should be assumed to generalize to individuals (Robinson, 2009). In Hamaker’s (2012) example, there is a negative group-level correlation between typing speed and typos; that is, more experienced typists make fewer errors. However, at the individual level, people make more errors when they type faster. Thus, there is a fundamental problem with the assumption that nomothetic or group-level findings also apply to individual patients.

These concerns about the validity of nomothetic findings have motivated calls for idiographic research (Barlow & Nock, 2009; Hamaker & Wichers, 2017; Molenaar, 2004). In contrast to nomothetic research, which draws conclusions about groups of people, idiographic research focuses on the individual. Idiographic approaches have a long history in psychological research, from the founders of experimental psychology to Skinner’s intensive studies of single animals (Barlow & Nock, 2009). These approaches have also been applied to clinical settings, beginning with Shapiro (1961, 1966) and continuing to researchers interested in single-case designs today (i.e., Kazdin, 2011). Indeed, idiographic approaches seem especially fitting for questions related to clinical psychology, a field that aims to alleviate symptoms for individual patients.

The field of psychology has long lacked statistical means of illustrating how an individual’s symptoms relate over time, forcing clinicians to rely heavily on clinical intuition.
However, several recent advances in data collection and analysis techniques have fostered an increase in idiographic research with intensive longitudinal designs. Significant developments in smartphone technology have led to increasing popularity of ecological momentary assessment (EMA), in which participants answer surveys multiple times per day over several days via their smartphone or other mobile device. In addition to providing a better understanding of how symptoms such as physical pain and mood fluctuate in patients’ day-to-day lives (May, Junghaenel, Ono, Stone, & Schneider, 2018), EMA allows for the collection of large amounts of data per participant over a relatively short amount of time (i.e., weeks). Once a large data set is obtained from a participant, idiographic data analysis techniques can be used to show how an individual’s symptoms relate to one another over time.

A growing number of statistical approaches have been extended to model individual processes. For a more exhaustive list, see Piccirillo et al. (2019). In the current study, I use dynamic structural equation modeling (DSEM; Hamaker & Wichers, 2017; Muthén & Muthén, 2017). Most idiographic techniques (i.e., graphical vector autoregression; Wild et al., 2010) are equipped to model relationships between observed variables, both contemporaneously and over time. An advantage of DSEM is that structural equation modeling allows one to model latent variables, therefore correctly quantifying the uncertainty associated with measurement error. Furthermore, although other methods rely on the assumption that processes are stationary or can be de-trended, DSEM is able to partially handle non-stationarity by modeling the effects of day and time on each observed variable. Due to these statistical advantages, DSEM was deemed an appropriate tool for the questions at hand.

The primary aim of Study 2 was to assess the relationships between physical pain, emotional pain, social connectedness, and depression for individual patients with chronic pain.
Prior to the start of data collection, three hypotheses were preregistered on Open Science Framework (see https://osf.io/5rtvx/ for full hypotheses and methods). First, I sought to examine whether physical pain and emotional pain load onto distinct latent factors for each individual. Second, I sought to examine whether depression and emotional pain load onto distinct latent factors for each individual. For these first two tests, I hypothesized that the results would vary between participants, such that neither a one-factor nor a two-factor model would provide better fit for all individuals. Lastly, I sought to examine whether emotional and physical pain prospectively predict one another for each individual. I hypothesized that prediction would vary between participants.

Given that idiographic research is relatively new and has never before been conducted with chronic pain patients, I also had several secondary aims related to evaluating the validity and utility of these novel methods. First, I sought to develop a protocol for comparing DSEM models, so as to avoid undue influence of the researchers on the statistical findings. I also sought to construct full DSEM models for each participant based on all of the symptoms assessed, including careful consideration of statistical assumptions and differential outcomes. In sum, I intended Study 2 to both offer insight into individuals with chronic pain and contribute to the ongoing development of idiographic research.

Method

Participants

Participants were recruited from Study 1. Those who completed Study 1 were given the option to provide contact information in order to be contacted about future studies. The same eligibility requirements were maintained from Study 1, with the additional inclusion criterion of owning an iPhone to complete EMA. Participants were excluded from Study 2 who had an active
substance use disorder within the past three months, or any acute psychiatric illness that impaired safety or prevented the participant from completing study tasks (e.g., active psychosis, mania, or suicidality).

**Procedure**

All research activities were approved by the Washington University in St. Louis Institutional Review Board. After confirming inclusion criteria over the phone, potential participants were invited to attend an in-person screening visit. At the in-person visit, I obtained informed consent and conducted a structured clinical interview to obtain psychological diagnoses and assess exclusion criteria. If eligible, participants also completed self-report questionnaires. Finally, participants downloaded a free application to their smartphone for the delivery of EMA. The EMA protocol then lasted for four weeks, in which participants received notifications on their smartphone four times per day. Notifications were delivered at the same times per day, four hours apart. For example, participants could receive notifications at 9am, 1pm, 5pm, and 9pm, or the times could be shifted to fit the participant’s schedule (i.e., 10am, 2pm, 6pm, 10pm). Participants received two reminder notifications if they did not complete the prompt within ten or twenty minutes respectively, and the survey closed after thirty minutes.

Participants could receive up to $35 for completing Study 2. Participants received $10 for completing the in-person screening visit, plus an additional $25 for completing 90% or more of the EMA prompts each week. If participants did not complete 90% or more of the prompts each week, they were given an option to extend the EMA period for up to 14 days. Participants who extended the EMA period also received the full $35.

**Measures**
**Diagnostic interview.** The MINI-International Neuropsychiatric Interview for DSM-5 (MINI; Sheehan, 2014) was administered to assess for exclusion criteria and obtain DSM-5 diagnoses. All interviews were conducted by the first author and reviewed by the last author for diagnostic agreement. Full diagnostic agreement was reached for all participants.

**Baseline measures.** I collected several baseline measures to be used in potential future multi-level analyses. At present, these measures are presented only as descriptives, given the small sample size of this idiographic study. Baseline measures included the BDI-II, PCS, the difficulty identifying feelings subscale of the Toronto Alexithymia Scale (TAS; Bagby, Parker, & Taylor, 1994), and the clarity subscale of the Trait Meta-Mood Scale (TMMS; Salovey, Mayer, Goldman, Turvey, & Palfai, 1995).

**EMA.** During the EMA portion of the study, participants received the same 13 questions four times per day in randomized order (see Appendix A). The EMA items were designed to assess four factors. The physical pain factor was intended to include three items related to the intensity, interference, and noticeability of physical pain. The emotional pain factor was also intended to include three items related to the intensity, interference, and noticeability of emotional pain. The depression factor was intended to include three items related to hopelessness, helplessness, and depressed mood. Finally, the social connectedness factor was intended to include three items related to loneliness, worry about relationships, and feeling connected to people. Tiredness was also assessed at each prompt. All items were rated on a scale from 0 (*Not at all*) to 10 (*A great deal*).

**Data Analytic Procedure**

I assessed my hypotheses using DSEM. DSEM is a modified version of structural equation modeling (SEM) that includes time-series modeling and time-varying effects modeling.
in addition to allowing for estimation of latent variables. DSEM allows one to regress any observed or latent variable not only on other variables at a single time point, but also on itself and other variables at previous time points (Asparouhov, Hamaker, & Muthén, 2018). DSEM analyses were conducted in Mplus Version 8.2 using the Bayes estimator (Muthén & Muthén, 2017). Full code and data will be made available at https://osf.io/5rtvx/.

**Assessment of pre-registered hypotheses.** First, I developed two competing models to test our pre-registered hypotheses. For Hypothesis 1, Model 1a included two distinct factors for physical pain (indicated by the three physical pain items) and emotional pain (indicated by the three emotional pain items). One lag was included, such that emotional pain was allowed to predict itself and physical pain, and physical pain was allowed to predict itself an emotional pain four hours later. Each item was also regressed on day (i.e., which day of the study) and time (i.e., which survey of the day). Model 1b included all emotional and physical pain items on a single factor, which was allowed to predict itself over one lag.

For Hypothesis 2, Model 2a included two distinct factors for emotional pain (indicated by the three emotional pain items) and depression (indicated by the hopeless, helpless, and depressed items). Similar lags were included as described above. Model 2b included all emotional pain and depression items on a single factor.

Hypothesis 3 was tested using Model 1a, the model which included separate physical and emotional pain factors that were allowed to predict each other and themselves over one lag.

**Prepare data set for each participant.** Next, a data set was prepared for each participant from the EMA. In order to achieve equal spacing between assessments, overnight assessment points were added and data was coded as missing (see Piccirillo et al., 2019). At this point, variance of the individual items was also assessed for each participant. Ideally, variables used in
DSEM should be dimensional with a range of at least seven. Although variables with less range can be treated as categorical, Mplus does not currently have the capability to include fit statistics for DSEM models using categorical variables. Therefore, testing my hypotheses requires data that at least approximates dimensional. For example, if a participant does not endorse an item at all, or if there are only very few non-zero endorsements, treating these variables as dimensional would violate the assumptions of DSEM.

**Run the hypothesized models.** For participants with sufficient variability in their data, I then ran the hypothesized model. In order to compare models, it is important that the Proportional Scale Reduction (PSR) factor settles close to 1 (Muthén, 2010). If the PSR does not settle close to 1, then the deviance information criterion (DIC) will vary across random seeds, making model comparison difficult. Thus, I assessed stability of both the PSR and DIC for hypotheses that required modeling comparison. Stability can generally be accomplished by increasing the number of iterations, unless there is a significant problem with the model. I tested all models with at least 10,000 iterations and a thin of 100 across three random seeds. If the PSR or DIC were unstable, I continued to increase the number of iterations.

**Compare the hypothesized models.** Once the PSR and DIC were stable, I compared the DIC across the two models for each hypothesis. The model with a lower DIC indicated better fit (Asparouhov et al., 2018).

**Exploratory DSEM models.** After testing my hypotheses, I also developed full DSEM models for each participant, including all measured variables. The EMA items were chosen based on a hypothesized model, in which emotional pain, physical pain, depression, and social connectedness represented distinct factors that could correlate with each other, as well as with the observed variable of tiredness. All factors and the observed variable “tired” were also
allowed to prospectively predict one another over one lag (4 hours). All items were regressed on the variables day (i.e., which day of the study) and time (i.e., which survey of the day).

Full DSEM models were initially run treating all data as dimensional. Where fit issues arose, the DIC was used to test whether alterations to the hypothesized model improved fit. As above, I aimed for a stable PSR near 1 and a stable DIC across multiple random seeds before comparing models. After settling on the best fitting model according to the DIC, items that did not have a range of at least seven were specified as categorical. Additional iterations were added if the PSR did not appear stable and low.

Results

Three participants provided enough EMA data to be analyzed using DSEM. All three participants were White, older than 55, and reported a chronic pain duration of 15 years or more. One participant was male and two were female. All three met criteria for past, recurrent MDD. Current depressive symptoms were reported as moderate by Participant 1 and minimal by Participants 2 and 3. Participant 2 additionally met criteria for current Generalized Anxiety Disorder (GAD). Compared to the Study 1 mean for pain catastrophizing (PCS = 23.37), all participants were below average (PCS = 13, 19, and 3, respectively). Average pain severity in the past week on a 0-10 scale was reported as 5 for Participant 1, 7 for Participant 2, and 3 for Participant 3.

Hypothesis 1

Hypothesis 1 tested whether emotional and physical pain should be conceptualized as two latent factors, or as one general pain factor for each participant. Participant 3 only dichotomously endorsed the emotional pain items, with five or fewer non-zero instances of each item. As such, there was not enough variability to treat this participant’s data as dimensional for the purposes of
Hypothesis 1. However, this lack of variability in one domain but not the other would suggest two distinct factors.

As can be seen in Table 2, the two-factor model was preferable for Participants 1 and 2, as is indicated by a lower DIC. Across three random seeds, the correlation between the emotional and physical pain factors was .95-.96 for Participant 1 and .68 for Participant 2.

**Hypothesis 2**

Hypothesis 2 tested whether emotional pain and depression should be conceptualized as two latent factors, or as one general negative affect factor for each participant. Participant 3 did not endorse any depressive symptoms; thus, this participant was excluded from Hypothesis 2 analyses.

As can be seen in Table 2, the two-factor model was preferable for Participants 1 and 2, as is indicated by a lower DIC. The correlation between the emotional pain and depression factors was .61 for Participant 1 and .99 for Participant 2.

**Hypothesis 3**

Hypothesis 3 tested whether emotional and physical pain prospectively predict one another for each individual. Based on Model 1a, emotional and physical pain did not prospectively predict one another for either Participant 1 or Participant 2.

**Full idiographic models**

**Participant 1.** Participant 1 reported the most item-level variability of the three participants. When treating this individual’s data as dimensional, the hypothesized model did not reveal any significant fit issues. Three items were then specified as categorical: hopeless, helpless, and worry about relationships. When specifying items as categorical, it can help convergence to collapse categories so that a reasonable number of observations (at least 5) are
included in each category. This yielded 3 hopelessness categories, 4 helplessness categories, and 4 worry about relationships categories.

As can be seen in Figure 2, many factors were significantly positively correlated with one another and with the observed variable “tired” for Participant 1. Physical pain and social connectedness each appeared to predict themselves over the 4-hour lag. Of note, whereas the correlation between the emotional and physical pain factors was .95-.96 for this participant in Hypothesis 1, the correlation between the two factors in the full model was .75. The emotional pain and depression factors were also significantly correlated at .57, as compared to .61 in Hypothesis 2.

**Participant 2.** Participant 2 only endorsed a non-zero amount of worry about relationships at three time points. As such, this item did not have enough variability to be included on the social factor. The remaining two social connectedness items, loneliness and connectedness, were composited. With the composited social connectedness items, the dimensional model converged appropriately. Next, 7 items were specified as categorical: the composited social connectedness item, all three emotional pain items, and all three depression items. After running 60,000 iterations with a thin of 100, the PSR was further from 1 than desired (final PSR = 1.052), indicating the model may not be converging properly. Only day and time effects reached significance. It was surprising that the correlations between factors were no longer significant; for example, the correlation between the emotional pain and depression factor was .70, but with a 95% credible interval including zero (-.19-.96). This correlation was .97 in the dimensional model (95% credible interval: .91-.99) and .99 in hypothesis 2.

Of note, the depression items had little variance for Participant 2. After collapsing categories, depression had 4 categories with 45, 6, 27, and 6 endorsements, hopelessness had 3
categories with 68, 5, and 11 endorsements, and helplessness had only 2 categories with 68 and 16 endorsements. For these reasons, I removed the depression factor to assess whether the model appeared to converge more appropriately. Without the depression factor, the PSR settled at 1.003 with 10,000 iterations and a thin of 100. As can be seen in Figure 3, emotional pain, physical pain, and social connectedness were significantly positively correlated once the depression factor was removed. Emotional pain was also significantly positively correlated with tiredness.

Participant 3. Participant 3 provided data that was all non-dimensional. The emotional pain, worry about relationships, and tired variables were endorsed dichotomously with few non-zero observations. While I initially attempted to include these variables in the model, they produced a large number of problematic iterations. Ultimately, I included only the physical pain and social connectedness items, which allowed the model to terminate normally. The model showed only time effects.

Discussion

In Study 2, I took an idiographic approach to examining the relationship between emotional and physical pain for individuals with chronic pain. My results suggest that for at least two individuals with chronic pain, emotional pain is distinct from both physical pain and depression. Furthermore, I found that emotional pain tends to be moderately to strongly positively correlated with both physical pain and depression for individual patients. These findings suggest that emotional pain may be an important part of chronic pain syndromes, at least for the two individuals whose data I examined here.

Contrary to Hypothesis 3, emotional and physical pain did not appear to predict one another for either participant. This finding has several potential explanations. First, it is possible that, despite the significant positive correlations between these two constructs, they simply do
not predict one another over time. However, other possibilities should be ruled out before accepting that this is the case. For example, perhaps emotional and physical pain do predict one another, but over a longer or shorter span of time. It is also possible that with more variability in the data, I would have greater ability to detect such predictive effects. For this and other reasons, I suggest considering a 0-100 scale in lieu of the 1-10 scale used in the current study. This would allow for further variability in the data, which would increase the likelihood of the data satisfying assumptions inherent to DSEM.

In addition to evaluating our pre-registered hypotheses, I sought to develop a framework for building and comparing idiographic models in DSEM. A key takeaway from this portion of Study 2 is that these methods are complex and require a great deal of attention to possible methodological concerns. I believe that DSEM offers several advantages over other idiographic techniques, including its ability to estimate latent variables and model day and time effects on each observed variable. However, DSEM also has several limitations, which became evident as I constructed full idiographic models for each individual. Notably, these limitations are also present in other idiographic analysis techniques, I simply consider them here in the context of DSEM. A primary limitation is the handling of non-dimensional variables. Variables can be specified as categorical in Mplus, but fit statistics (i.e., DIC) cannot be procured from such an analysis. Thus, if one wishes to compare models, all variables must be treated as dimensional. In the current study, I arrived at a best-fitting model by treating variables as dimensional, and then testing that model with the appropriate variables specified as categorical. I recognize that this approach is somewhat backwards, but at present it is the best we have. While these tools continue to be developed in Mplus, I suggest making other efforts to measure variables dimensionally (i.e., using a larger response scale).
DSEM and SEM approaches in general have been criticized as incurring a high number of *researcher degrees of freedom* (Simmons, Nelson, & Simonsohn, 2011). As was evident in our construction of full idiographic models, these models often require constraints and multiple model comparisons. I fully acknowledge that this element of human influence on the data analysis process provides opportunity for error as well as p-hacking, when researchers continuously cherry-pick data or analyses until results become significant (Head, Holman, Lanfear, Kahn, & Jennions, 2015). However, I also acknowledge that failing to model the data appropriately can lead to spurious and misleading findings. Thus, altering models as I have done here is a necessary step. In addition to the steps described in this article, I will post code and guidelines that will hopefully provide a framework for building and comparing idiographic models (see https://osf.io/5rtvx/). I hope that as the field of idiographic research continues to develop, researchers will be transparent about what problems arise in their models and how they choose to handle them.

Taking these limitations into account, I am cautiously optimistic about the utility of idiographic models in clinical practice. Idiographic methods have often been touted as tools for determining treatment targets, and I continue to hope that these types of models will one day tell us which of several co-occurring symptoms is the most suitable target for treatment. For example, if I did see that emotional pain or depression predicts physical pain for a patient, this could be an empirical indication that depression treatment is warranted. Conversely, if it is physical pain that predicts the emotional outcomes for a patient, treatment of the physical condition may be more suitable. In the current study, I failed to find associations of this nature. It is becoming increasingly clear across many methods of idiographic analysis that idiographic models often reveal fewer meaningful predictive effects than we hope to find (i.e., Wright et al,
Rather, many models reveal only correlational, autoregressive, and time-related effects. I contend that these effects are still interesting, because they reveal dependencies among an individual’s symptoms that we would have otherwise been unable to detect using traditional means of nomothetic analysis. Furthermore, it is possible that adjusting which variables we measure and how we measure them may improve our ability to detect meaningful relationships between symptoms.

Summary

Chronic pain is often conceptualized as having a strong emotional component. Not only is chronic pain highly comorbid with psychological disorders such as MDD, but the experience of pain itself is defined as including both a sensory and an affective component. In Study 1, I found that emotional and psychological pain are related to physical pain severity and the impact of chronic pain on one’s psychosocial functioning. In Study 2, I found that for at least two individuals with chronic pain, emotional pain is distinct from and positively related to both physical pain and depression. These findings suggest that emotional pain may be a reasonable treatment target for individuals with chronic pain, perhaps above and beyond currently developed interventions focusing on physical pain and depression. Further research is still necessary to understand how physical and emotional pain impact one another over time for individual patients.
Appendix A: EMA questions

At the screening visit, the following instructions were used to clarify what I mean when I ask about physical pain vs. emotional pain:

*People often report that they experience physical pain, including headache, tooth pain, joint or muscle pain. When I ask you about your physical pain, please consider only these types of **physical** experiences (not limited to those listed).*

*People also report that they experience pain from emotional events or states. Some common sources of emotional pain are feeling rejected, lonely, or socially isolated. When I ask you about your emotional pain, please consider only these types of **emotional** experiences (not limited to those listed).*

During the EMA period, the following 13 questions were presented at each prompt in randomized order. Participants responded using a sliding scale from 0 (*Not at all*) to 10 (*A great deal*).

1 [Phys1]. How much physical pain do you have right now?
2 [Phys2]. How much is your physical pain interfering with your current activities?
3 [Phys3]. How noticeable is your physical pain?
4 [Emo1]. How much emotional pain do you have right now?
5 [Emo2]. How much is your emotional pain interfering with your current activities?
6 [Emo3]. How noticeable is your emotional pain?
7 [Dep]. I feel depressed
8 [Hope]. I feel hopeless
9 [Help]. I feel helpless
10 [Tired]. I feel tired
11 [Lonely]. I feel lonely
12 [WryRel]. I feel worried about my relationships with people
13 [Conn]. I feel connected to people
References


https://doi.org/10.1073/pnas.1102693108


https://doi.org/10.1037/0033-2909.131.2.202


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Figures and Tables

Table 1. Means, standard deviations, and correlations among study variables.

<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>SD</th>
<th>Phys</th>
<th>Emo</th>
<th>PAS</th>
<th>RSQ</th>
<th>PCS</th>
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<tbody>
<tr>
<td>1. Physical pain</td>
<td>9.91</td>
<td>2.10</td>
<td>--</td>
<td></td>
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<tr>
<td>2. Emotional pain</td>
<td>6.88</td>
<td>3.06</td>
<td>.51*</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PAS</td>
<td>23.67</td>
<td>9.63</td>
<td>.47*</td>
<td>.51*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. RSQ</td>
<td>9.85</td>
<td>6.09</td>
<td>.13</td>
<td>.20</td>
<td>.32*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. PCS</td>
<td>22.86</td>
<td>13.39</td>
<td>.47*</td>
<td>.36*</td>
<td>.76*</td>
<td>.10</td>
<td>--</td>
</tr>
<tr>
<td>6. PII</td>
<td>16.91</td>
<td>5.69</td>
<td>.20</td>
<td>.27*</td>
<td>.67*</td>
<td>.27</td>
<td>.57*</td>
</tr>
</tbody>
</table>

Note: Phys = PROMIS Physical pain; Emo = PROMIS Emotional pain; PAS = Psychache Scale; RSQ = Rejection Sensitivity Questionnaire; PCS = Pain Catastrophizing Scale; PII = Psychosocial Illness Impact

* p < .05
Table 2. Results of model comparisons for Hypotheses 1 & 2.

<table>
<thead>
<tr>
<th></th>
<th>Two-Factor</th>
<th>One-Factor</th>
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<tr>
<td></td>
<td>DIC</td>
<td>Factor Correlation</td>
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<tr>
<td>Participant 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H1: emotional &amp;</td>
<td>1077.498-1311.925</td>
<td>.95-.96*</td>
</tr>
<tr>
<td>physical pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H2: emotional pain &amp; depression</td>
<td>1597.63-1604.77</td>
<td>.61*</td>
</tr>
<tr>
<td>Participant 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H1: emotional &amp;</td>
<td>815.08-820.95</td>
<td>.68*</td>
</tr>
<tr>
<td>physical pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H2: emotional pain &amp; depression</td>
<td>982.52-983.58</td>
<td>.99*</td>
</tr>
</tbody>
</table>

Note. For both participants, the two-factor models provided better fit than one-factor models.

*Significance as indicated by a 95% credible interval that does not contain zero.
Figure 1. Relationship between Emotional and Physical Pain Moderated by Pain Catastrophizing
Figure 2. Full idiographic model for Participant 1

Note. All paths represented were significant, as indicated by a 95% credible interval that did not contain zero. Green indicates positive relationships; red indicates negative relationships. Double-sided arrows represent contemporaneous relationships; single-headed green arrows represent lagged relationships. Single-headed black arrows represent factor loadings. Relationships with Day (i.e., day of the study) and Time (i.e., survey of the day) are contemporaneous.
Figure 3. Idiographic model for Participant 2

Emotional Pain

Emo1 → Emo2 → Emo3

Physical Pain

Social Connected

Time

Tired

Day

Time

Phys1

Phys2

Phys3

Time
Figure 4. Idiographic model for Participant 3