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Individual Differences in the Cognitive-Behavioral Model of Chronic Back Pain in Patients
Receiving Spine Surgery: Towards Precision Phenotypes

by
Madelyn R. Frumkin, M.A.

A dissertation presented to
Washington University in St. Louis
in partial fulfillment of the
requirements for the degree
of Doctor of Philosophy

August 2024
St. Louis, Missouri

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Table of Contents

List of Figures	iii
List of Tables	iv
Acknowledgments.....	v
Abstract.....	vi
Introduction.....	1
Conceptualization and Treatment of Chronic Back Pain: A historical perspective	1
Towards Precision Pain Medicine	5
The Current Study.....	6
Method	10
Participants	10
Presurgical Measures	13
Postsurgical Measures	16
Data Analysis.....	16
Power	19
Results.....	19
Construct Validity.....	20
Average Within-Person Relationships between Self-Reported Pain, Depressed Mood, and Catastrophizing	21
Hypothesis 1: Prospective Relationships will Significantly Vary across Individuals.....	25
Hypothesis 2: Individual-level paths will vary based on demographic and clinical moderators.	29
Exploratory Aim 1: Relationships between Activity and EMA	33
Exploratory Aim 2: Associations with Surgical Outcomes.....	36
Discussion.....	38
Appendix A. EMA Items	45
References Cited	46

List of Figures

Figure 1: Distributions of composite Ecological Momentary Assessment (EMA) constructs.....	21
Figure 2: Group-level contemporaneous (A) and lagged (B) relationships between constructs...	25
Figure 3: Variability in individual-level parameter estimates.....	28
Figure 4. Pairwise correlations between individual-level estimates and demographic and clinical characteristics.....	31
Figure 5: Association between number of significant paths from individual-level estimates and demographic and clinical characteristics.....	32
Figure 6: Time series of Fitbit step count and subsequent Ecological Momentary Assessment (EMA) pain reports for three exemplar participants.....	35
Figure 7: Associations between dynamic effects involving consequences of activity and postoperative improvement in pain interference.....	37

List of Tables

Table 1: Demographics and clinical characteristics.....	12
Table 2: Variability in individual-level paths.....	27

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Madelyn Frumkin

Washington University in St. Louis

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ABSTRACT OF THE DISSERTATION

Individual Differences in the Cognitive-Behavioral Model of Chronic Back Pain in Patients

Receiving Spine Surgery: Towards Precision Phenotypes

by

Madelyn Frumkin

Doctor of Philosophy in Clinical Science

Department of Psychological and Brain Sciences

Washington University in St. Louis, 2024

Professor Thomas Rodebaugh, Chair

Chronic back pain impacts over 20 million adults in the United States and represents the costliest condition in our healthcare system. Treatments for chronic back pain range from cognitive-behavioral therapy to opioid pain medications and spine surgery. At present, it is extremely difficult to determine which treatments are likely to maximize benefit and minimize risk for a given patient. As such, the goal of this study was to examine whether individual differences in the cognitive-behavioral model of chronic back pain were associated with baseline characteristics or surgical outcomes. Patients ($N = 95$) with chronic back pain who were receiving spine surgery completed baseline self-report questionnaires and three weeks of mobile health monitoring, which included ecological momentary assessment and activity tracking. Multilevel dynamic structural equation models were used to examine individual differences in dynamic relationships among physical, emotional, and cognitive symptoms. Results indicated substantial between-person variability, such that no lagged relationships were statistically significant for $> 28\%$ of participants. Although there was evidence that many dynamic

relationships between subjective experiences (i.e., pain and depressed mood) were weakly associated with symptom severity at baseline, they were not associated with surgical outcomes. However, dynamic relationships between activity and subsequent subjective reports emerged as larger and more robust predictors of postoperative improvement. Specifically, I observed greater postoperative improvements in pain interference among patients for whom increased activity was more strongly associated with subsequent pain interference ($r = -.40, p < .001$), pain severity ($r = -.39, p < .001$), and catastrophizing ($r = -.36, p = .003$) preoperatively. These findings suggest there is significant potential for dynamic relationships, particularly those involving objective monitoring, to be harnessed to develop and test precision interventions for chronic back pain.

Introduction

Chronic back pain (CBP) is the costliest condition in the United States healthcare system, with an estimated \$134.5 billion in spending from 1996 to 2016 (Dieleman et al., 2020). In 2019, over 20 million adults in the United States reported significant impairment due to CBP (Yong et al., 2022). In addition to interfering with physical functioning and day-to-day behavior, CBP is associated with increased risk of depression, suicide, and substance addiction (Currie & Wang, 2004; Ilgen et al., 2013; Vowles et al., 2015). Importantly, back pain is the most common reason for prescription of long-term opioids, which remain the leading cause of unintentional drug overdoses in the United States (Boudreau et al., 2009; Centers for Disease Control and Prevention (CDC), 2011; Okie, 2010). Given the rising prevalence of CBP and the worsening opioid epidemic, it is critical to improve understanding and treatment of CBP.

Conceptualization and Treatment of Chronic Back Pain: A historical perspective

The field of medicine was historically governed by the biomedical model, which conceptualized the mind and body as distinct and independent entities (Engel, 1977; Gatchel et al., 2007). These ideas were inspired by Renee Descarte's *mind-body dualism*, which he introduced in opposition to many ancient Greek and Roman philosophers before him who viewed pain as being intertwined with emotional experience (Rey, 1995). Under the biomedical model, pain was treated as problem that was purely physical. Several theories of pain were developed and tested under this model, including some that suggested pain resulted from the stimulation of specific receptors, and contrasting theories that the receptors were non-specific, but the patterns of neural firings were what differentiated pain from other sensations (Moayed & Davis, 2013; Rey, 1995).

In the early 1800s, morphine was isolated from opium, and by the mid-1800s was used widely for acute and chronic pain. The emphasis on pharmacological treatment of pain continued to grow with increasing numbers of disabled veterans returning from World War II and Vietnam (Bernard et al., 2018). However, by the 1950s, physicians and researchers were beginning to sound alarms. Biomedical theories of pain were failing to account for many phenomena including pain in absence of noxious stimuli, phantom limb pain, and the variable relationship between tissue damage and pain (Gatchel et al., 2007). Furthermore, treatments being used for pain relief were not systematically having the intended effects (Beecher, 1959; Rey, 1995). It was apparent that something was missing.

From Biomedical to Biopsychosocial Models of Pain. The field of pain moved dramatically away from biomedical models with Melzack and Wall's (1965) seminal gate control theory of pain. Gate control theory was one of the first major theories to propose a mechanism by which other systems could inhibit or facilitate the pain experience. In this way, gate control theory could account for the variable relationship between pain and tissue damage, as well as variable responses to pain medications (Gatchel et al., 2007). The theory was also multidimensional in that it recognized the role of both biological and psychosocial factors (e.g., emotion, cognition) in inhibiting or facilitating pain perception.

Chronic pain has now been recognized and treated as a biopsychosocial phenomenon for several decades (Gatchel et al., 2007; Lumley et al., 2011; Turk & Monarch, 2002). This model has shifted both our understanding of why individuals experience pain and our conceptualization of chronic pain conditions. Of significant importance to the biopsychosocial model is the distinction between *disease* and *illness*. Disease is defined as “an objective biological event,” whereas illness reflects “subjective experience or self-attribution” regarding the disease (Turk &

Monarch, 2002). Similarly, *nociception* refers to the direct stimulation of nerves that trigger pain pathways to the pain, whereas *pain* reflects the subjective perception of unpleasant sensory experiences (Gatchel, 2004; Gatchel et al., 2007). Reflecting this distinction, the International Association for the Study of Pain has defined pain as: “An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage” (Merskey & Bogduk, 1986; Treede, 2018).

Cognitive-Behavioral Theory. While the medical community was shifting towards a biopsychosocial model of pain, the psychological community was shifting towards behavioral and cognitive-behavioral models of psychopathology. These changes soon merged, with American psychologist Wilbert Fordyce publishing one of the first descriptions of behavioral treatment for chronic pain (Fordyce, 1976). Fordyce primarily applied principles of operant condition to target pain behaviors. Soon after, Turk and colleagues (1983) added cognitive components to this model, describing ways that thoughts and beliefs about pain may influence behavior. For example, it became widely accepted that pain-related fear played a significant role in the onset and maintenance of CBP (Crombez et al., 1999, 2012). By the 1990s, evidence supporting the efficacy of cognitive-behavioral approaches to treating chronic pain was rapidly accumulating (Ehde et al., 2014; Hoffman et al., 2007; Jensen & Turk, 2014; Morley et al., 1999).

Ongoing Tensions between Biomedical and Biopsychosocial Models of CBP

Treatment. As evidence for psychologically mediated treatment of CBP was mounting, the opioid epidemic was just beginning. In 1995, OxyContin was approved by the Food and Drug Administration, who at the time believed the drug had lower abuse potential than existing formulas given its slower absorption (Center for Drug Evaluation and Research, 2023).

Misjudgments about the risks associated with OxyContin in combination with misleading advertising from the drug's maker, Purdue Pharma, led to a nearly tenfold increase in OxyContin prescriptions for chronic non-cancer pain from 1997 to 2002 (Cicero et al., 2005; Van Zee, 2009). This began the first wave of the opioid epidemic. From 1999 to 2006, the number of overdose deaths involving opioid analgesics more than tripled from 4,000 to 13,800 (Warner et al., 2009). Despite limited evidence of efficacy and high risks, opioids remain a common treatment for CBP (Boudreau et al., 2009; Deyo et al., 2009, 2011).

During this time, spine surgery has also become an increasingly common procedure for CBP. Rates of spine surgery increased by 55% in the 1980s and have continued to increase, contributing to rising healthcare expenditures related to spine problems (Deyo & Mirza, 2006; Marquez-Lara et al., 2014; Martin et al., 2009). Spine surgery is intended to correct anatomical problems believed to be causing CBP. In some cases, the cause of pain may be evident, such as a tumor pressing on the spine or a trauma to the spine. However, in the vast majority of cases, it is difficult to determine if anatomical abnormalities identified via imaging are the *cause* of CBP, as half or more of pain-free adults 30 and older will have degenerative spine imaging findings (Brinjikji et al., 2015). Consequently, updated guidelines have cautioned against routine imaging of patients with CBP (Hall et al., 2021). Despite this, healthcare expenditures for spine care including imaging continued to increase. Critically, 10-40% of patients who undergo spine surgery will experience *Failed Back Surgery Syndrome* (FBSS), which is defined as persistent or recurring CBP following one or more spine procedures (Baber & Erdek, 2016; Chan & Peng, 2011; Hall et al., 2021). It is also common for patients to continue using opioids after spine surgery, and analgesic poisoning is the leading cause of death three years after spine surgery

(Juratli et al., 2009; Uhrbrand et al., 2021). It is therefore important to consider the pros and cons of CBP interventions, especially relative to individual patient characteristics.

Towards Precision Pain Medicine

Precision medicine aims to provide an answer to the question of what treatment or treatments are likely to maximize benefit and minimize risk for a given patient (Ashley, 2015; Collins & Varmus, 2015). Precision medicine approaches have generally focused on genetic or other biological factors that can be used to match patients with the medications most likely to work (Emery & Akil, 2020; Fernandes et al., 2017; Gandal et al., 2016). In pain medicine, there has been some progress towards identifying genetic variants predictive of *who* is likely to experience greater pain and pain-related problems (Niculescu et al., 2019). However, these biomarkers are highly overlapping with other psychiatric disorders, including depression and suicidality (Le-Niculescu et al., 2013). Brain imaging is also a popular method in the search for precision pain biomarkers. There has been some progress towards identifying neural signatures associated with pain (Lee et al., 2021; Tracey et al., 2019; Wager et al., 2013) and possible mechanisms for treatment response (Edwards et al., 2023; Tu et al., 2019). However, at present, there are limited actionable treatment decisions that can be made based on biological factors alone (Nour et al., 2022).

Given the lack of clear and actionable biomarkers, as well as the emphasis on conceptualizing pain as a biopsychosocial phenomenon, increasing efforts are being made to identify psychosocial factors that could aid in precision pain medicine. For example, several studies have attempted to empirically test the question “What works for whom?” by assessing moderators of treatment response to CBT for chronic pain, including demographics, disease characteristics, coping styles, maladaptive beliefs around pain, and treatment expectancies

(Broderick et al., 2016; Chen et al., 2023; Gurung et al., 2015; Turner et al., 2007; Wertli et al., 2014). However, no clear and robust moderators of psychosocial treatments for chronic pain have emerged (Murillo et al., 2022). Consequently, matching patients to treatments based on baseline characteristics has had mixed success (Turk, 2005; Vlaeyen & Morley, 2005).

While psychological research has largely focused on explaining individual differences in response to CBP treatment, the field of medicine has focused more on prediction of who is at risk for poor outcomes. For example, several studies have attempted to uncover who is at risk for outcomes including problematic opioid use, spine surgery complications, and failed back surgery syndrome. Psychosocial factors such as depression have emerged as risk factors for poor outcomes across all of these medical interventions (Chan & Peng, 2011; Grattan et al., 2012; Khor et al., 2018). Similarly to CBP, depression is a highly heterogeneous condition (Fried & Nesse, 2015). Depression is also so highly comorbid with chronic pain, and the directionality of this relationship likely varies across individuals (Arnow et al., 2006; Bair et al., 2008; Currie & Wang, 2004; Magni et al., 1994). Thus, it remains unclear how to act on this information.

I argue that to improve understanding of what works for whom, we must move towards identifying *precision phenotypes* based on biopsychosocial mechanisms that may predict treatment response. This effort is more analogous to the search for biomarkers than the search for patient clusters, in that I seek to identify individual differences in specific processes hypothesized to be involved in treatment response.

The Current Study

The overarching aim of the current study is to empirically test the cognitive-behavioral model of CBP in patients receiving spine surgery. Importantly, the existing theories are largely

non-specific, suggesting that thoughts, emotions, situations, and behaviors are all bidirectionally related to one another in the perpetuation of CBP (Ehde et al., 2014; Gatchel et al., 2007; Turner et al., 2007). The limited tests of these theories have often been conducted using methods that are inappropriate for the within-person nature of the question at hand. For example, the cognitive-behavioral mediation model suggests that internal factors such as perceived life interference, perceived control, and maladaptive thoughts (i.e., catastrophizing about pain) mediate the association between pain and depression (Rudy et al., 1988; Turk et al., 1995; Wood et al., 2013). However, these findings are based on cross-sectional data, which is insufficient for drawing conclusions about causality (Cole & Maxwell, 2003; Maxwell et al., 2011; Maxwell & Cole, 2007; McNeish & Mackinnon, 2022; Preacher, 2015). Thus, I will assess dynamic, within-person relationships between physical, emotional, and cognitive symptoms using EMA data and Dynamic Structural Equation Modeling (DSEM; Asparouhov et al., 2018a).

The following specific hypotheses were pre-registered on Open Science Framework prior to data collection (<https://osf.io/qmt89>). My first specific aim was to examine between-person variability in dynamic, within-person relationships between physical, emotional, and cognitive symptoms. Multiple studies have shown that within-person relationships between factors such as pain and affect range from negative or non-significant to strong and positive across different individuals (Frumkin & Rodebaugh, 2021; Mak & Schneider, 2020; van Middendorp et al., 2010; Vendrig & Lousberg, 1997). I sought to extend these findings by examining individual differences in the cognitive-behavioral model of CBP. I hypothesized (H1) that prospective relationships would significantly vary across individuals, as opposed to the alternative hypothesis that physical, emotional, and cognitive symptoms are related to the same degree across

individuals. Variability in these relationships across individuals is necessary to examine associations between individual differences and treatment outcomes.

Next, I examined moderators of between-person variability to facilitate understanding of these processes in terms of variables most often used to categorize CBP patients. In contrast to the data-driven methods that have been used in prior studies (Beneciuk et al., 2015; Viniol et al., 2013), I used hypothesis-driven methods to test theorized moderators of pain-related processes. Given that greater depressive symptom severity is related to denser temporal relationships among symptoms (Pe et al., 2015), and pain severity is associated with depressive symptom severity (Bair et al., 2008), I hypothesized (H2a) that individuals experiencing more severe depression or pain would exhibit stronger bidirectional temporal relationships between depressed mood and pain in personalized models. Because depression is more common among women and younger individuals (Fiske et al., 2009; Piccinelli & Wilkinson, 2000), I also hypothesized (H2b) that women and younger individuals will experience stronger bidirectional relationships in personalized models. Finally, I hypothesized (H2c) that individuals who had more severe difficulties with emotion regulation, anxiety, and pain catastrophizing would experience stronger bidirectional relationships in personalized models. If substantiated, these hypotheses would facilitate understanding and prediction of personalized models based on demographic and clinical characteristics.

I also had two exploratory aims. First, I incorporated physiological data collected via Fitbit wrist-worn sensors. Physical activity is a common antecedent to pain for individuals with and without chronic pain. It is a natural response to avoid activities that induce pain. However, a pattern of fear and avoidance around physical activity can contribute to chronic pain through a combination of biological (e.g., deconditioning) and psychosocial (e.g., loss of pleasurable

activities, sustained anxiety) pathways (Crombez et al., 2012; Meulders, 2019). Consequently, reduction of physical activity is considered a maladaptive pain behavior, and safely increasing activity is a key component of CBT for chronic pain (Fordyce, 1976; Turk et al., 1983). Tests of the theory that activity is associated with pain and psychosocial factors have been overwhelmingly cross-sectional (Greenberg et al., 2023; Heneweer et al., 2011). Thus, as an exploratory aim, I sought to dynamically test associations between activity, pain, depressed mood, and catastrophizing. Similarly to my primary aims, I hypothesized (H3) that these relationships would vary across individuals and be associated with demographic and clinical characteristics.

The above hypotheses focus on dynamic relationships between pain, catastrophizing, mood, and activity as correlates of presurgical symptom severity. However, my primary objective in conducting this study was to determine whether such dynamic relationships can suggest precision phenotypes that would be indicative of treatment outcomes. Thus, my final exploratory aim focused on determining whether any dynamic relationships were associated with postsurgical changes in pain interference. I hypothesized (H4a) that stronger dynamic relationships among pain, catastrophizing, and depressed mood would be associated with *worse* surgical outcomes, as surgery is not designed to address these biopsychosocial mechanisms of CBP. I also hypothesized (H4b) that stronger dynamic relationships from subjective variables to activity (e.g., increased pain leading to decreased activity) would be associated with worse surgical outcomes. By contrast, I hypothesized (H4c) that stronger dynamic relationships between activity and subsequent subjective variables (e.g., increased activity leading to increased pain) would be associated with *better* surgical outcomes, as surgery is intended to resolve pain that is resulting from anatomical problems, which would be exacerbated with activity.

Method

Participants

Participants ($N = 95$) included in this study were patients with degenerative spine disease being evaluated for possible spine fusion at Washington University School of Medicine (WUSM). Participants were recruited as part of a larger study with investigators at WUSM examining predictors of response to spine fusion surgery. Inclusion criteria included English-speaking adults aged 21 to 85 years old who owned a smartphone, had at least 1 week to complete assessments prior to surgery, and reported a numeric rating scale pain score of at least 3 out of 10 during the previous week. Patients who were undergoing surgery for infection, malignancy, or trauma, those undergoing isolated thoracic fusion, and those undergoing another major surgery within 3 months of data collection were excluded (see Greenberg et al., 2022 for further details). The study was approved by the institutional review board (IRB# 202012139), and all patients provided informed consent. Demographics and clinical characteristics are presented in Table 1.

Procedure

Participants were recruited over the phone following a recent appointment with a WUSM neurosurgery or orthopedic spine surgeon. A research coordinator contacted the patient, explained the study, and solicited interest in further participation. If participants indicated interest in the study, then the research coordinator verbally reviewed the purpose and procedures of the study, the voluntary nature of participation, access to protected health information, and study compensation. If patients indicated they would like to participate, they then provided informed consent and were given instructions to download the LifeData application (LifeData LLC) to complete Ecological Momentary Assessment (EMA) on their personal smartphone.

Participants specified a 12-hour period in which they preferred to receive surveys every 3 hours (i.e., 9am to 9pm).

Table 1. Demographics and clinical characteristics

Variable	<i>M (SD)</i>
Age	59.15 (12.52)
PROMIS Pain Interference	67.07 (5.06)
PROMIS Pain Intensity	66.15 (6.64)
PROMIS Physical Function	33.42 (4.98)
Pain Catastrophizing Scale	18.75 (13.00)
PHQ-9	7.66 (5.98)
PROMIS Anxiety	56.78 (8.66)
ERQ Cognitive Reappraisal	5.26 (1.06)
ERQ Expressive Suppression	3.42 (1.30)
	<i>n (%)</i>
Gender (Male)	44 (46%)
Race	
White	89 (94%)
African American/Black	4 (4%)
Other	2 (2%)
Ethnicity	
Hispanic or Latine	4 (4%)
Not Hispanic or Latine	90 (95%)
Education	
Did not graduate high school or obtain a GED	2 (2%)
High school degree	27 (28%)
College degree	35 (37%)
Graduate or professional school degree	31 (33%)
Employment Status	
Actively working	33 (35%)
Homemaker	4 (4%)
Unemployed	2 (2%)
Retired	38 (40%)
On disability	18 (19%)

Note. PROMIS = Patient-Reported Outcomes Measurement Information System (these are reported as t-scores); ERQ = Emotion Regulation Questionnaire; PHQ-9 = Patient Health Questionnaire-9

After agreeing to participate in the study, participants completed several self-report questionnaires via REDCap. Participants were also mailed a Fitbit Inspire 2 with instructions to wear the tracker as much as possible but at least during the 12-hour EMA period. Participants received 5 EMAs daily for approximately 3 weeks, or until their surgery. Participants could choose the time at which their surveys started. However, all surveys were administered every 3 hours. Participants had 30 minutes to respond to each survey and were sent 2 reminders at 15-minute increments. Participants were paid \$1 per completed EMA survey (up to \$105), and \$20 for using the Fitbit for any duration. For further details on study procedures and recruitment, see (Greenberg et al., 2022).

Presurgical Measures

Baseline Self-Report. The National Institutes of Health's (NIH) Patient-Reported Outcomes Measurement Information System (PROMIS) was used to assess several baseline characteristics. PROMIS measures were developed to improve measurement of patient-reported outcomes and have demonstrated sound psychometric properties, including in spine surgery populations (Ader, 2007; Steinhaus et al., 2019; Stone et al., 2016). Pain intensity was assessed using the PROMIS Pain Intensity Short Form. The three-item measure assesses current pain, as well as worst pain and average pain over the past week, on a scale from 1 (Had no pain) to 5 (Very severe). Pain interference, physical function, and anxiety were additionally assessed using the PROMIS computer adaptive testing feature. All PROMIS measures were scored as t-scores, such that a score of 50 represents the population mean, with a standard deviation of 10. For all measures except physician functioning, higher scores represent more severe symptoms. For physical functioning, lower scores represent worse physical function.

Depressive symptom severity was assessed using the depression module of the Patient Health Questionnaire (PHQ-9). This 9-item measure assesses each DSM-IV criteria for depression on a scale from 0 (Not at all) to 3 (Nearly every day). The PHQ-9 has shown strong evidence of validity and reliability (Kroenke et al., 2001).

Tendency to catastrophize about pain was assessed using the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995). 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 rated 13 items 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).

Ability to regulate emotions was assessed using the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). Participants rated 10 items regarding tendency to use cognitive reappraisal and expressive suppression to regulate emotions. Items are rated on a scale from 1 (Strongly disagree) to 7 (Strongly agree). The ERQ has strong psychometric properties in general community samples (Preece et al., 2020).

Ecological Momentary Assessment (EMA). Data for dynamic models were collected via EMA. At each prompt, participants responded to 12 EMA items assessing physical, emotional, and cognitive symptoms in randomized order (see Appendix A). Participants were instructed to respond based on how they were feeling right before they received the notification. All items were answered on a sliding scale from 0 (none) to 100 (worst possible).

In accordance with the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) recommendations, we used numeric rating scales (NRS) to assess pain intensity at each prompt (Dworkin et al., 2005). Given the clinical population, we chose to assess intensity of back pain, leg pain, and overall pain. Second, we assessed pain interference using three items related to the degree to which pain was interfering with activities, concentration, and enjoyment of life. These items were adapted from the PROMIS Pain Interference Short Form, which contains six items. We chose the three items that were most general, as some items that referred to specific contexts (e.g., getting groceries, socializing, recreational activities) may be difficult to answer in momentary assessments. Third, we assessed pain catastrophizing using three items adapted from the Daily Pain Catastrophizing Scale (Darnall et al., 2017). The three items assess the rumination (“I keep thinking about how much I hurt”, magnification (“My pain overwhelms me”), and helplessness (“I am afraid that my pain will get worse”) facets of catastrophizing. Prior work including a subset of the current sample showed that these three items showed good within- and between-person reliability, construct validity, and prognostic utility in surgical contexts (Frumkin et al., 2023). Finally, we assessed depressed mood using three items adapted from the PROMIS Emotional Distress-Depression Short Form. The short form contains four items related to feeling depressed, worthless, hopeless, and helpless. We omitted the “helpless” item given that helplessness is part of the assessment of catastrophizing. Between- and within-person reliability of all EMA constructs are reported in the results.

Ambulatory Physiological Assessment. Activity data were collected via a wrist-worn activity tracker. Participants were provided with a Fitbit Inspire 2 following enrollment and were asked to wear the Fitbit for the remainder of the study. Validity or reliability of Fitbits have been

measured in at least 144 studies, and results of a systematic review suggest that Fitbits accurately measure steps (Fuller et al., 2020).

Postsurgical Measures

Participants repeated the PROMIS Pain Interference computer adaptive test approximately 1-month post-surgery. This score was used to evaluate pre-post changes in pain interference.

Data Analysis

An analysis plan was pre-registered on Open Science Framework prior to data collection (<https://osf.io/qmt89>). Data were analyzed using R version 4.2.1 and Mplus version 8.9 (Muthén & Muthén, 2017).

Confirmatory Factor Analysis. I hypothesized a priori that the EMA items (see Appendix A) would represent 3 latent factors: pain (indicated by EMA items 1-6), depressed mood (indicated by EMA items 7-9), and pain catastrophizing (indicated by EMA items 10-12). A multi-level confirmatory factor analysis was conducted to confirm that items loaded onto the expected factors. If fit was below accepted cutoffs ($CFI \geq .90$, $TLI \geq .90$, and $RMSEA < 0.08$), then alternative models were compared with items removed or moved to different factors. Reliability of the resulting factors was assessed via between- and within-person omega coefficients (Lai, 2021).

All hypotheses were assessed using Dynamic Structural Equation Modeling (DSEM; Asparouhov et al., 2018a). The planned ML-DSEM models included autoregressive, contemporaneous, and cross-lagged relationships among pain, depressed mood, and catastrophizing. In other words, each construct would be allowed to predict itself and the other

constructs, both at the same point in time (contemporaneous) and over one lag (i.e., 3 hours). To satisfy stationarity assumptions, time variables (i.e., day of the study, survey of the day) were also allowed to predict each construct. A 95% Credible Interval (CI) that did not contain zero indicated a significant relationship between two constructs, which was also expressed by a Bayesian one-tailed p -value corresponding to the proportion of the posterior distribution that had a sign in the opposite direction from the posterior mean. All parameters would be specified as random, such that they could vary between individuals. Model convergence was assessed via the Proportional Scale Reduction (PSR). A PSR close to one suggested little variation across chains, indicating a properly converged model (Hamaker et al., 2018). By default, Mplus identifies models with $PSR < 1.1$ as converged, although a strict convergence criterion of $PSR < 1.01$ is preferred.

I intended to include all constructs in a single model. However, given the number of random effects and parameters being estimated, it is common for these models to have difficulty with convergence (Asparouhov & Muthen, 2022). In this case, I followed guidance from the developers of Mplus to estimate a series of two-variable models.

The preregistered analysis plan specified that these models would be used to assess my first hypothesis regarding between-person variability in dynamic relationships. Specifically, a between-person variance estimate with a 95% CI that did not contain zero would indicate significant between-person variability in within-person relationships. Notably, however, the default prior distributions in Mplus constrain between-level variance estimates to be positive, so it is not possible to obtain a non-significant result (Asparouhov & Muthen, 2022). I therefore relied on guidance from the developers of Mplus to consider a variance estimate meaningful if it was at least three times as large as its posterior standard deviation (Asparouhov & Muthen,

2022). Because the standard deviation will be influenced by sample size (e.g., it will decrease as sample size increases), I also examined individual-level estimates estimated in ML-DSEM for a more detailed understanding of variability across the sample. Pairwise correlation and linear regression analyses were then used to assess group-level moderators of individual-level prospective paths (H2).

This procedure was repeated to assess variability and moderators of dynamic relationships between activity and EMA variables (H3). Given that activity was continuously assessed via a wrist-worn Fitbit (versus EMA data collected approximately every 3 hours), I binned Fitbit data into 1-hour increments. To assess relationships between activity and subsequent self-reported pain, interference, catastrophizing, and depressed mood, I used Fitbit data collected in the hour prior to the EMA survey. Minute-level step count readings within each 1-hour increment were summed for each individual and divided by the individual's wear time in that hour (to account for occasions in which participants may not have worn the Fitbit for the full 60-minute increment). EMA response (e.g., pain) was then regressed on pre-EMA step count at the within-person level in ML-DSEM. Similarly, to assess relationships between self-reported EMA response and subsequent activity, the same procedure was followed for step count readings in the hour *following* EMA responses. Post-EMA step count was then regressed on EMA response (e.g., pain) at the within-person level. These ML-DSEM models were used to examine group-level effects and variability across individuals. Pairwise correlation and linear regression analyses were used to assess moderators of individual-level relationships between activity and participant characteristics.

Finally, estimates generated from the models described above were used to examine predictors of postsurgical pain interference. A change score was calculated for each participant

by subtracting presurgical pain interference from postsurgical pain interference (e.g., more negative scores indicate more improvement). Pairwise correlations and linear regression analyses were used to examine associations between improvement in pain interference and hypothesized within-person mechanisms (e.g., relationships between pain and mood, pain and activity, etc.).

Power

Power to uncover effects in ML-DSEM is based on a combination of the number of participants (N), number of observations (T), complexity of the model, and strength of the effects. Without prior work from which to estimate the expected effect sizes, I was unable to conduct exact power analyses. However, a prior simulation study suggests a tradeoff between N and T , such that measuring 150 subjects 25 times, 75 subjects 75 times, or 50 subjects 150 times yields sufficient power for somewhat simpler models (Schultzberg & Muthén, 2018). Thus, with 95 participants, I aimed to collect at least 50-75 observations per person for sufficient power to uncover within- and between-person effects.

Results

Across all 95 participants, 7192 of 8472 possible EMA prompts were completed. Participants completed an average of 76 surveys each ($SD = 26$, Range: 9-135), for an average adherence rate of 84% ($SD = 13\%$, Range: 40-100%). Age was positively associated with adherence rate ($r = .31$, $p = .002$), whereas depression symptom severity was weakly negatively associated with adherence rate ($r = -.23$, $p = .029$). Adherence rate was also marginally higher among men ($M = 87\%$) compared to women ($M = 82\%$), $t(82.56) = 2.03$, $p = .045$.

The majority of participants ($n = 79$, 83%) also wore the Fitbit during the preoperative EMA period. There was no evidence that those who wore the Fitbit systematically differed from those who did not on demographic or clinical characteristics. Fitbit data were available for 4527

of 7192 total time points (63%). On average, each participant had approximately 57 EMA surveys with Fitbit data available ($SD = 29$, Range: 1-103).

Construct Validity

With the residual variance for the hopeless item constrained to 0 (to be non-negative), fit of the hypothesized 3-factor model was acceptable but not excellent (CFI = 0.882, TLI = 0.849, RMSEA = 0.038). I tested a 4-factor model separating pain intensity from pain interference, and fit improved (CFI = 0.944, TLI = 0.924, RMSEA = 0.027). I therefore retained the 4-factor model for subsequent analyses. The pain severity, pain interference, pain catastrophizing, and depressed mood constructs each showed acceptable to good within-person reliability ($\omega_{\text{within}} = .73-.86$) and excellent between-person reliability ($\omega_{\text{between}} = .90-.99$). As shown in Figure 1, pain severity, pain interference, and catastrophizing composite variables were approximately normally distributed (skewness = -0.09-0.48). However, the depressed mood items were highly positively skewed (skewness = 2.17). As such, I log transformed only the depressed mood composite by first adding 1 to each value to remove zeros and then performing the base 10 logarithm. This transformation improved normality of the depressed mood composite (skewness = 0.36).

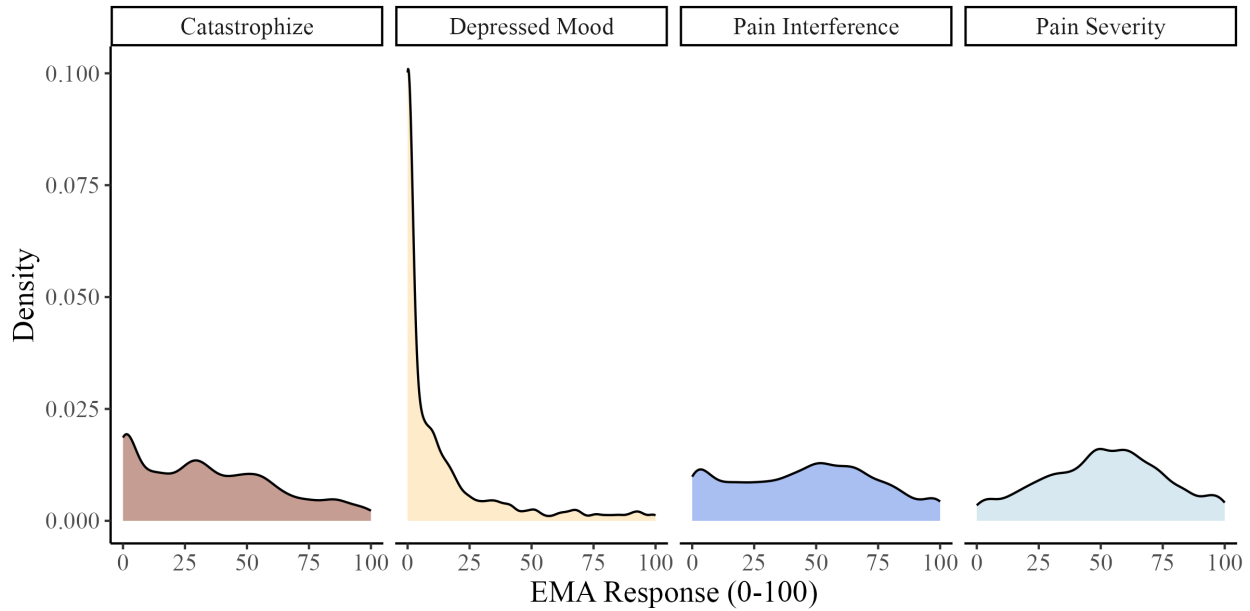


Figure 1. Distributions of composite Ecological Momentary Assessment (EMA) constructs

Average Within-Person Relationships between Self-Reported Pain, Depressed Mood, and Catastrophizing

Next, I intended to assess autoregressive, contemporaneous, and cross-lagged relationships among pain severity, pain interference, pain catastrophizing, and depressed mood constructs in a single model. However, this model would not converge ($PSR > 1.9$), likely due to its complexity. I therefore followed guidance from the developers of Mplus to estimate a series of two-variable models (Asparouhov & Muthen, 2022). I first report the within-level standardized estimates averaged over clusters (i.e., fixed effects) before testing my primary hypotheses related to between-person variability. The significant contemporaneous (i.e., same occasion) and lagged (i.e., next occasion) effects are visualized in Figure 2A and 2B, respectively. Significance was indicated by a 95% CI that did not contain zero.

Pain Severity and Pain Interference. Notably, I had planned to include both pain severity and pain interference in a single pain construct. However, given that the ML-CFA suggested separate severity and interference factors, I have analyzed them separately. Pain severity tended to increase over the course of each day ($\beta = 0.029, p < .01$) and over the course of the study ($\beta = 0.128, p < .001$). These time effects are partialled out of remaining effects.

Both pain severity and interference had a tendency to predict themselves or persist over time, as evidenced by significant lag-1 autoregressive coefficients ($\beta = 0.309$ for pain severity, $\beta = 0.306$ for interference, $ps < .001$). As shown in Figure 2A, a two-variable model assessing dynamic relationships between pain severity and pain interference suggests a significant positive covariance between pain severity and pain interference ($\beta = 0.566, p < .001$). As shown in Figure 2B, pain severity was also a significant predictor of next occasion interference ($\beta = 0.095, p < .001$), and interference was a significant predictor of next occasion pain severity ($\beta = 0.121, p < .001$).

Pain Severity and Depressed Mood. This model between pain severity and log transformed depressed mood converged well (PSR < 1.01). Depressed mood also had a tendency to predict itself or persist over time, as evidenced by a significant lag-1 autoregressive coefficient ($\beta = 0.308, p < .001$). As shown in Figure 2A, there was a significant positive covariance between pain severity and depressed mood ($\beta = 0.229, p < .001$). Regarding cross-lagged effects, pain severity was a small predictor of next occasion depressed mood ($\beta = 0.024, p = .043$). However, the 95% CI [-0.002 - 0.053] contained zero. Depressed mood was not a significant predictor of next occasion pain severity ($\beta = 0.016, p = .086$).

Pain Interference and Depressed Mood. Initially, this model did not converge as well. To help with convergence, I constrained the prospective path of interference predicting depressed mood and the autocorrelation of depressed mood to be fixed across individuals. I also removed individuals without any variance in depressed mood ($n = 6$). With these alterations, the PSR was below the convergence criterion of 1.1, but above the preferred criterion of 1.01 (PSR = 1.020). As shown in Figure 2A, there was a significant positive covariance between pain interference and depressed mood ($\beta = 0.277, p < .001$). Regarding cross-lagged effects, depressed mood was a small but significant predictor of next occasion pain interference ($\beta = 0.041, p = .004$), and pain interference was a small but significant predictor of next occasion depressed mood ($\beta = 0.024, p = .019$).

Pain Severity and Catastrophizing. Next, I examined relationships between pain constructs and the cognitive construct of catastrophizing. This model converged well (PSR < 1.01). Whereas pain severity tended to increase over the course of each day and over the course of the study, catastrophizing tended to decrease over the course of each day ($\beta = -0.026, p = .002$) and remain stable over the course of the study ($\beta = 0.021, p = .080$). Catastrophizing also had a tendency to predict itself or persist over time, as evidenced by a significant lag-1 autoregressive coefficient ($\beta = 0.296, p < .001$). There was a significant positive covariance between pain severity and catastrophizing ($\beta = 0.476, p < .001$). Regarding cross-lagged effects, catastrophizing was a significant predictor of next occasion pain severity ($\beta = 0.116, p < .001$), and pain severity was a significant predictor of next occasion catastrophizing ($\beta = 0.066, p < .001$).

Pain Interference and Catastrophizing. Similarly, there was a significant positive covariance between pain interference and catastrophizing ($\beta = 0.480, p < .001$). Regarding cross-

lagged effects, catastrophizing was a significant predictor of next occasion pain interference ($\beta = 0.140, p < .001$), and pain interference was a significant predictor of next occasion catastrophizing ($\beta = 0.127, p < .001$). This model also showed excellent convergence (PSR < 1.01).

Depressed Mood and Catastrophizing. Finally, to complete the pairwise comparisons, I examined dynamic relationships between depressed mood and catastrophizing. Initially, this model would not converge (PSR > 1.5). Examination of the trace plots suggested that the most problematic parameters were the covariance between depressed mood and catastrophizing and the lagged relationship of depressed mood predicting catastrophizing. To ease estimation of these parameters, I constrained the between-person variance of these parameters to zero. I also removed individuals without any variance in depressed mood ($n = 6$). With these alterations, the PSR was below the preferred criterion of 1.01 (PSR = 1.009).

There was a significant positive covariance between depressed mood and catastrophizing ($\beta = 0.346, p < .001$). Regarding cross-lagged effects, depressed mood was a significant predictor of next occasion catastrophizing ($\beta = 0.059, p < .001$), and catastrophizing was a significant predictor of next occasion depressed mood ($\beta = 0.073, p < .001$).

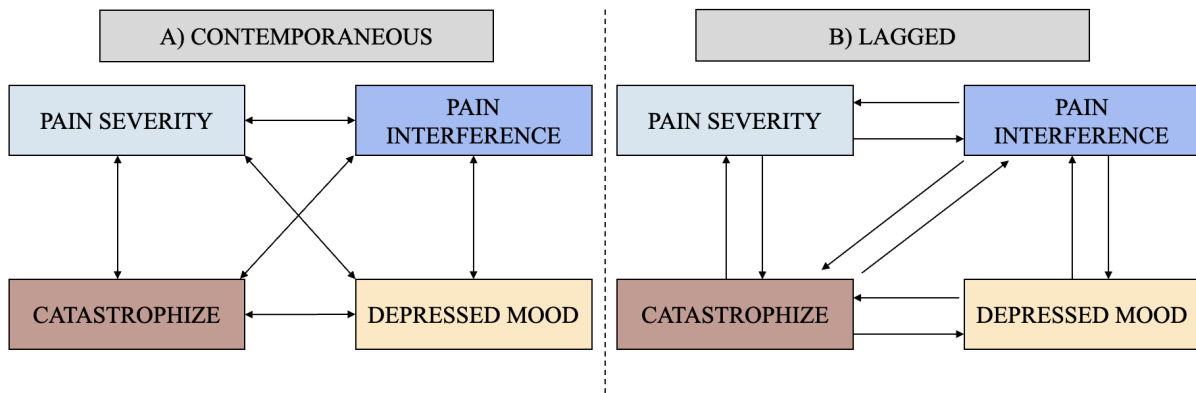


Figure 2. Group-level contemporaneous (A) and lagged (B) relationships between constructs. All paths shown were significant, as indicated by a 95% credible interval that did not contain zero. All paths were also in a positive direction, suggesting increases in one construct corresponds to increase in the other construct. Lagged relationships were assessed over 3 hours.

Hypothesis 1: Prospective Relationships will Significantly Vary across Individuals

Next, I used the two-variable models to examine variability in dynamic relationships between physical, emotional, and cognitive symptoms. For each model, I first report the variance estimates, which are considered meaningful if they are at least three times as large as their posterior standard deviation (Asparouhov & Muthen, 2022). I also used individual-level estimates (i.e., random effects) from the ML-DSEM model to visualize variability and report the proportion of participants with a significant estimate (95% CI does not contain zero).

As reported in Table 2 and visualized in Figure 3, all covariances showed substantial between-person variability. Most participants ($n = 87, 92\%$) showed a significant positive covariance between pain severity and pain interference. The majority of participants also exhibited significant positive covariances between pain variables (severity and interference) and catastrophizing ($n = 74-76, 78-80\%$). However, only about half of participants exhibited a

significant positive covariance of depressed mood with pain severity ($n = 50, 53\%$) or interference ($n = 48, 54\%$).

Few participants exhibited significant lagged relationships between pain and depressed mood. Pain severity positively predicted depressed mood for 3 participants and negatively predicted depressed mood for 1 participant. There was greater variability for lagged relationships with catastrophizing. For example, lag-1 pain interference significantly predicted catastrophizing and vice versa for 24% of participants ($n = 23$). Notably however, only 10 participants had a significant path in both directions. Lag-1 catastrophizing also significantly predicted pain severity for 18 participants and depressed mood for 14 participants. On average, each participant had 7 significant paths ($SD = 2.7$, Range: 1-16).

Table 2. Variability in individual-level paths

Parameter	Variance Estimate	Posterior <i>SD</i>	95% Credible Interval		<i>n</i>	%
Pain/Interference Covariance	0.133	0.023	0.097 - 0.188	*	87	91.58
Interference → Pain	0.022	0.008	0.011 - 0.042		24	25.26
Pain → Interference	0.015	0.005	0.008 - 0.028	*	12	12.63
Pain/Depressed Mood Covariance	0.040	0.009	0.026 - 0.062	*	50	52.63
Depressed Mood → Pain	0.028	0.012	0.011 - 0.059		2	2.11
Pain → Depressed Mood	0.003	0.002	0.001 - 0.007		3	3.16
Interference/Depressed Mood Covariance	0.092	0.018	0.065 - 0.133	*	48	53.93
Depressed Mood → Interference	0.049	0.014	0.028 - 0.084	*	6	6.74
Interference → Depressed Mood ^a	0.001	0.000	0.001 - 0.001		--	--
Pain/Catastrophize Covariance	0.135	0.023	0.097 - 0.189	*	76	80.00
Catastrophize → Pain	0.024	0.008	0.012 - 0.043	*	18	18.95
Pain → Catastrophize	0.011	0.004	0.006 - 0.02		10	10.53
Interference/Catastrophize Covariance	0.150	0.026	0.109 - 0.209	*	74	77.89
Catastrophize → Interference	0.027	0.007	0.016 - 0.043	*	23	24.21
Interference → Catastrophize	0.033	0.008	0.021 - 0.051	*	23	24.21
Depressed Mood/Catastrophize Covariance ^a	0.001	0.000	0.001-0.001		--	--
Catastrophize → Depressed Mood	0.012	0.003	0.008-0.019	*	14	15.73
Depressed Mood → Catastrophize ^a	0.001	0.000	0.001-0.001		--	--

n = number significant in hypothesized (positive) direction; % = percent significant in hypothesized (positive) direction.

^aThese paths were constrained to be fixed across participants to improve convergence.

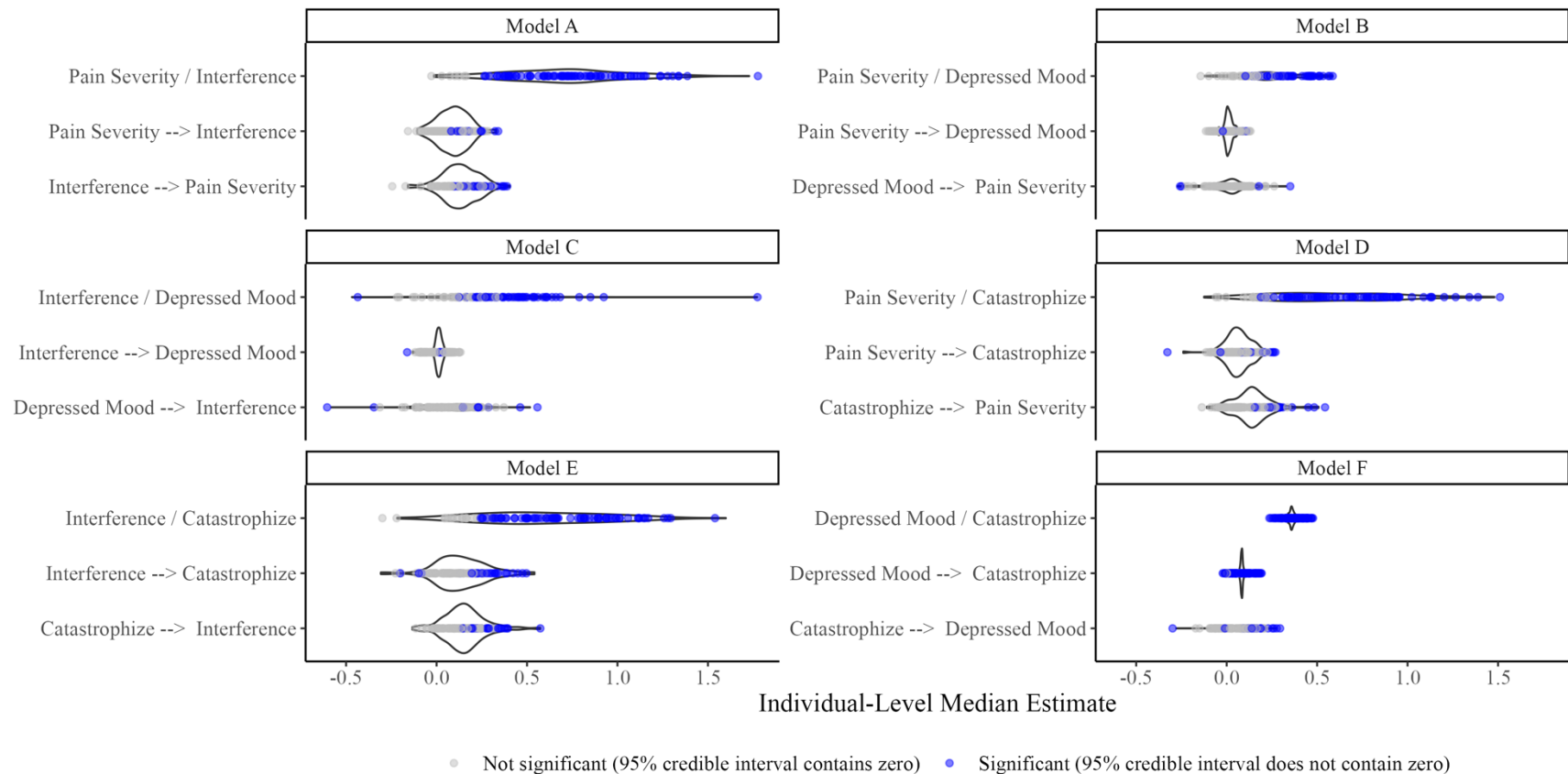


Figure 3. Variability in individual-level parameter estimates. This plot shows an estimate of within-person contemporaneous and prospective relationships for each participant. Each dot represents an individual's standardized median estimate. Blue dots have a 95% credible interval that does not contain zero, suggesting the estimate is significantly different from zero. / = contemporaneous covariance; → = lagged effect. Interference → Depressed Mood, Depressed Mood → Catastrophize, and Depressed Mood/Catastrophize paths were constrained to be fixed across participants to improve convergence.

Hypothesis 2: Individual-level paths will vary based on demographic and clinical moderators.

Associations between the strength of individual-level paths and demographic and clinical characteristics are displayed in Figure 4. In partial support of hypothesis H2a, only the covariance between depressed mood and pain interference was positively associated with depression symptom severity ($r = .27, p = .013$) and pain intensity ($r = .35, p < .001$) assessed at baseline. Contrary to hypothesis H2b, no individual-level relationships between pain and mood were associated with age or gender. Finally, in partial support of hypothesis H2c, PROMIS anxiety was positively associated with the covariance between depressed mood and pain interference ($r = .35, p = .001$). Trait-like catastrophizing reported on the PCS was also positively associated with the covariance between interference and depressed mood ($r = .25, p = .020$), as well as the covariance between depressed mood and pain severity ($r = .21, p = .045$). Finally, a greater tendency to suppress emotions as reported on the ERQ was associated with a lesser tendency for depressed mood to predict increased interference at the next time point ($r = -.22, p = .041$).

Although the pre-registered analysis plan only included paths between depressed mood and pain in this hypothesis, I did find additional associations between demographic and clinical characteristics and individual-level paths involving momentary catastrophizing. Specifically, age was negatively associated with the covariance between pain severity and catastrophizing ($r = -.20, p = .047$), the covariance between pain interference and catastrophizing ($r = -.26, p = .011$), and the degree to which increased pain predicted increased catastrophizing ($r = -.22, p = .029$). The degree to which increased pain predicted increased catastrophizing was also positively associated with trait-like catastrophizing reported on the PCS ($r = .30, p = .003$). These findings

are considered exploratory given that they were not pre-registered.

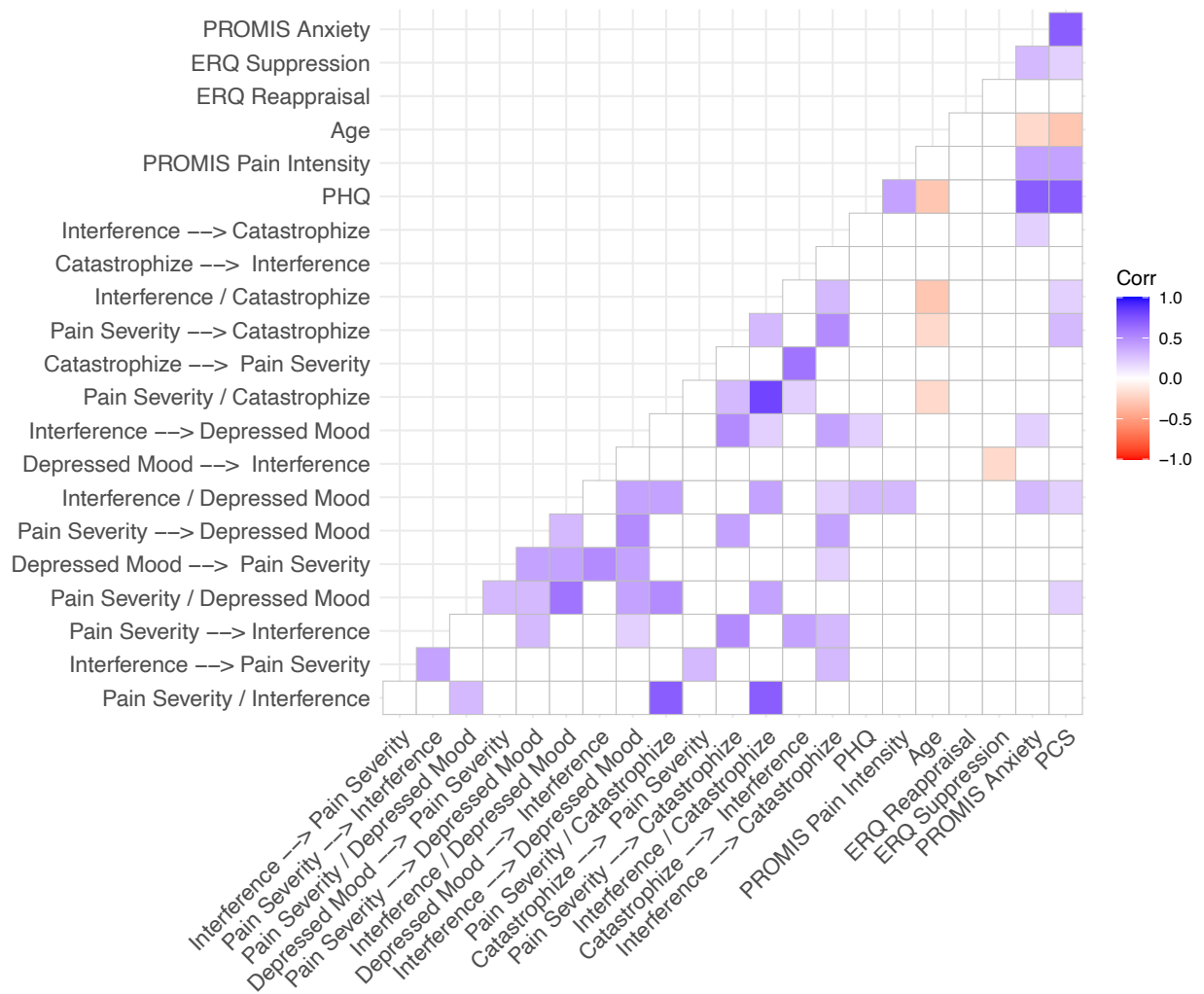


Figure 4. Pairwise correlations between individual-level estimates and demographic and clinical characteristics. Only significant relationships ($p < .05$) are displayed. PHQ = Patient Health Questionnaire-9; PROMIS = Patient-Reported Outcomes Measurement Information System; ERQ = Emotion Regulation Questionnaire; PCS = Pain Catastrophizing Scale

Finally, I examined associations between number of significant paths and demographic and clinical characteristics. As shown in Figure 5, participants with more significant paths exhibited worse preoperative physical function ($r = -.25, p = .015$) and greater preoperative pain interference, depressive symptoms, catastrophizing, and anxiety ($r_s = .22-.23, p_s < .05$). The associations between number of paths and pain intensity was not significant ($r = .15, p = .148$), nor did number of significant paths vary by age or gender.

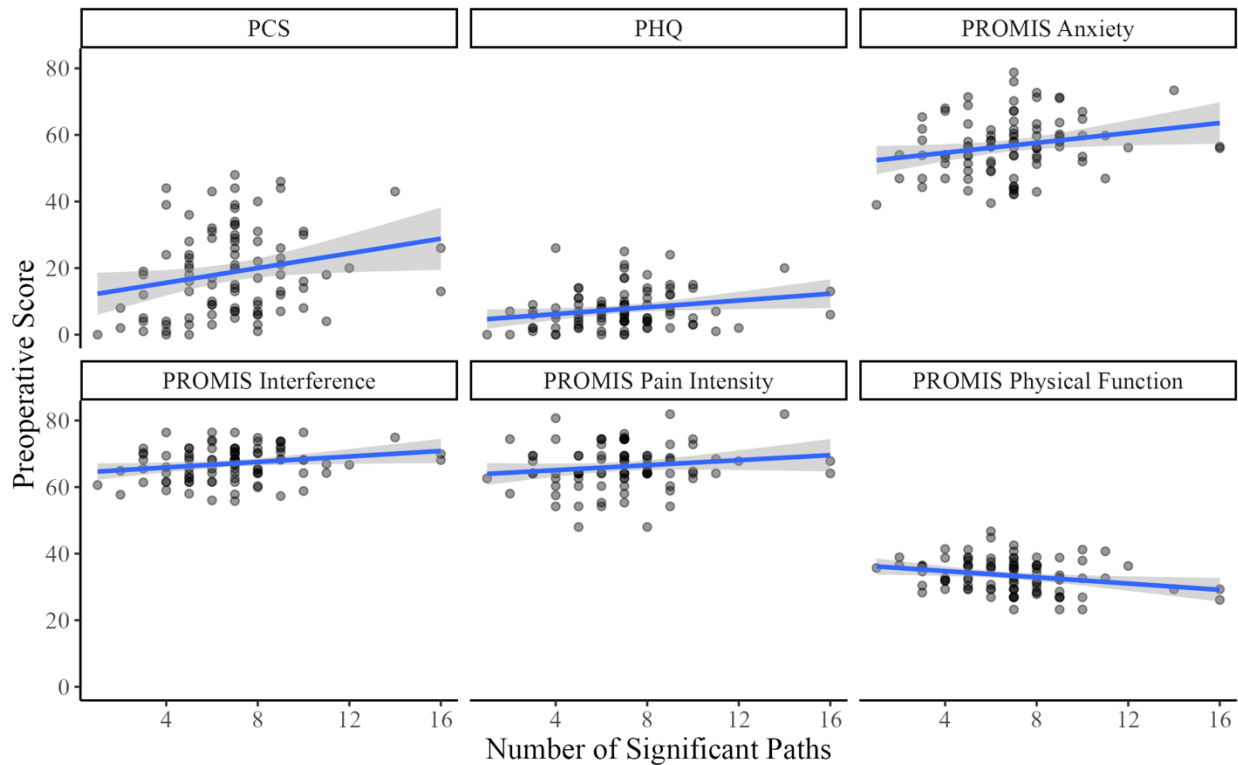


Figure 5. Association between number of significant paths from individual-level estimates and demographic and clinical characteristics. PROMIS = Patient-Reported Outcomes Measurement Information System; PHQ = Patient Health Questionnaire-9; PCS = Pain Catastrophizing Scale

Exploratory Aim 1: Relationships between Activity and EMA

As an exploratory aim, I also used two-variable ML-DSEM models to assess dynamic relationships between physical activity (i.e., step count), pain, interference, depressed mood, and catastrophizing. Similar to the models above, these analyses were conducted as two-variable models that included autoregressive and time-related effects.

For individuals on average, increased steps in the hour prior to EMA was associated with increased pain severity ($\beta = 0.107$), pain interference ($\beta = 0.063$), and catastrophizing ($\beta = 0.052$, p s < .001). Increased activity was not significantly associated with subsequent depressed mood ($\beta = -0.005$, $p = .357$). In the opposite direction, increased pain, interference, catastrophizing, and depressed mood were not associated with subsequent increases or decreases in activity for individuals on average.

Variability. Based on individual-level estimates, the parameter with the greatest between-person variability was activity predicting subsequent pain. For 28% of participants ($n = 22$), there was a significant positive relationship, such that increased steps in the hour prior to EMA predicted increased pain reports. For one individual, increased steps were associated with *decreased* subsequent pain, and the remaining participants did not have a significant relationship between step count and subsequent pain. Figure 6 provides a visualization of the time series data from the participant who showed a negative association between steps and pain (A), a participant who showed no association between steps and pain (B), and a participant who showed a positive relationship between steps and subsequent pain (C).

Associations between step count and other subjective variables were somewhat less variable across individuals. Increased steps in the hour prior to EMA significantly predicted

increased pain interference for 13% of participants ($n = 10$) and increased catastrophizing for 8% of participants ($n = 6$). For two participants, increased steps predicted decreased depressed mood.

Although EMA variables were not significant predictors of subsequent activity for individuals on average, there were still a few individuals who showed significant effects. Specifically, for 4% of participants ($n = 3$), increased pain interference was associated with *increased* subsequent steps, and for 1 individual, increased pain severity was associated with increased subsequent steps.

Moderators. Activity predicted subsequent catastrophizing more strongly among women ($M = 0.03$, $SD = 0.03$) as compared to men ($M = 0.02$, $SD = 0.03$), $t(75.95) = -2.39$, $p = .019$. Activity also predicted subsequent interference more strongly among women ($M = 0.04$, $SD = 0.03$) as compared to men ($M = 0.02$, $SD = 0.03$), $t(73.85) = -2.29$, $p = .025$. Age was positively associated with the degree to which pain severity and interference predicted subsequent steps ($r = .24-.26$, $ps < .05$). Finally, individuals for whom activity more strongly predicted subsequent catastrophizing were also likely to experience greater pain intensity ($r = .33$, $p = .004$) and pain catastrophizing at baseline ($r = .26$, $p = .024$).

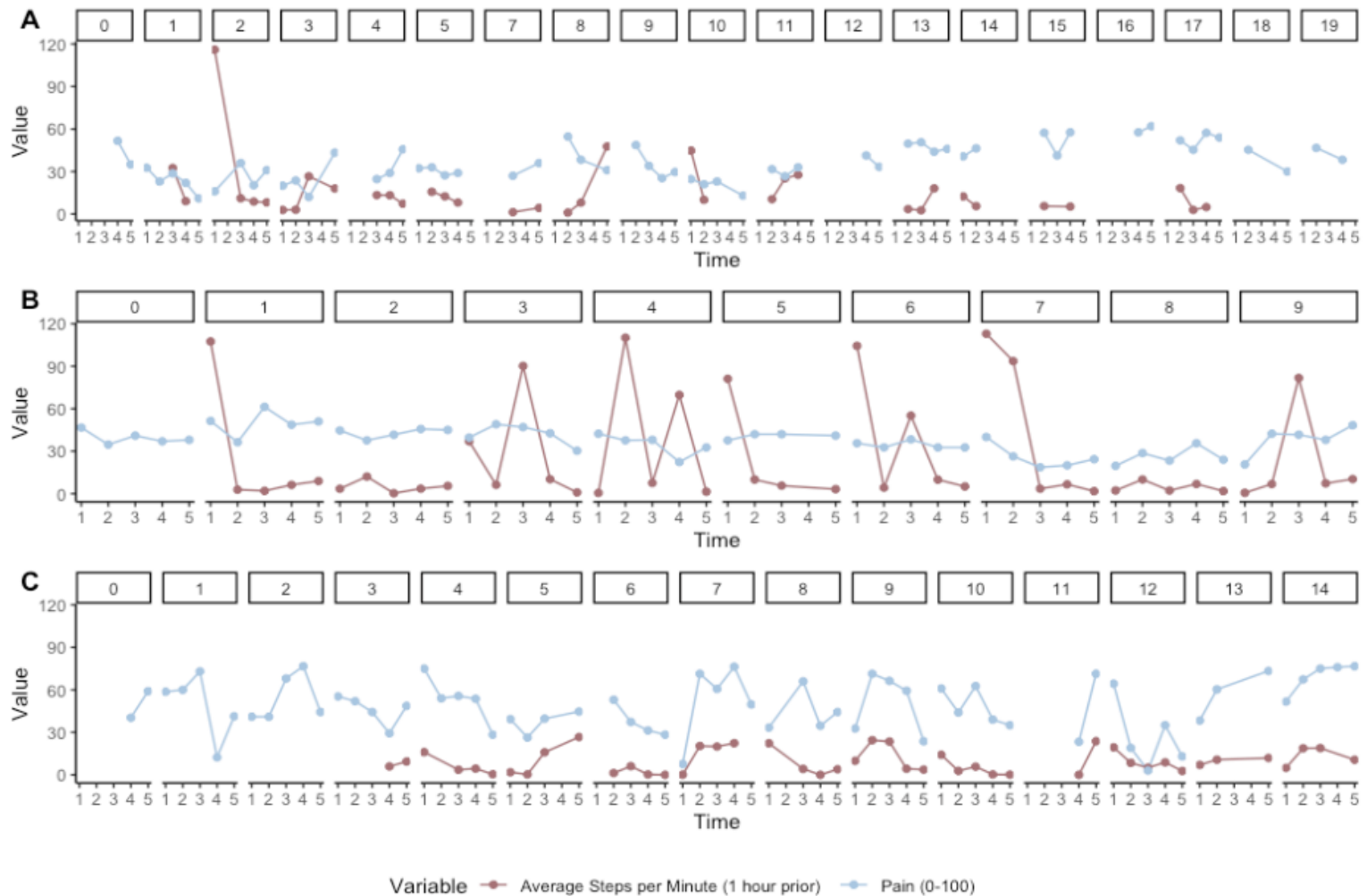


Figure 6. Time series of Fitbit step count and subsequent Ecological Momentary Assessment (EMA) pain reports for three exemplar participants: one patient with a negative relationship between steps in the hour prior to EMA and subsequent pain (A), one patient with no relationship between steps and subsequent pain (B), and one patient with a positive relationship between steps and subsequent pain (C). The boxes at the top of each panel represent day of monitoring.

Exploratory Aim 2: Associations with Surgical Outcomes

Finally, I examined whether any of the dynamic relationships described thus far were associated with surgical outcomes. Of 95 participants, 79 (83%) provided 1-month outcomes data ($n = 69$, 87% of those with Fitbit data). On average, participants reported a 6.8-point improvement in pain interference ($SD = 8.8$).

In contrast to Hypothesis 4a, only one dynamic predictor derived from preoperative EMA was associated with postoperative improvement in pain interference: Individuals with a stronger covariance between pain severity and interference were likely to report greater improvements in pain interference post-operatively ($r = -.27, p = .015$). In contrast to Hypothesis 4b, stronger dynamic relationships from subjective variables (pain, catastrophizing, depressed mood) to activity were not associated with postoperative improvement in pain interference ($r_s = -.21 - -.04, p_s > .05$). As noted above, there was limited variability in these effects across individuals.

However, as shown in Figure 7A, dynamic relationships between activity and subsequent subjective reports emerged as larger and more robust predictors of postoperative changes in pain interference. In support of Hypothesis 4c, I observed greater postoperative improvements in pain interference among individuals for whom increased activity was more strongly associated with subsequent pain interference ($r = -.40, p < .001$), pain severity ($r = -.39, p < .001$), and catastrophizing ($r = -.36, p = .003$) preoperatively. As shown in Figure 7B, individuals for whom increased steps significantly predicted subsequent increased pain had marginally greater postoperative improvement in pain interference ($M = -10.22, SD = 8.8$) as compared to individuals who did not exhibit this effect ($M = -5.46, SD = 8.38$), $t(31.16) = 2.03, p = .051$. Other paths were not examined categorically due to low numbers of participants with statistically significant paths.

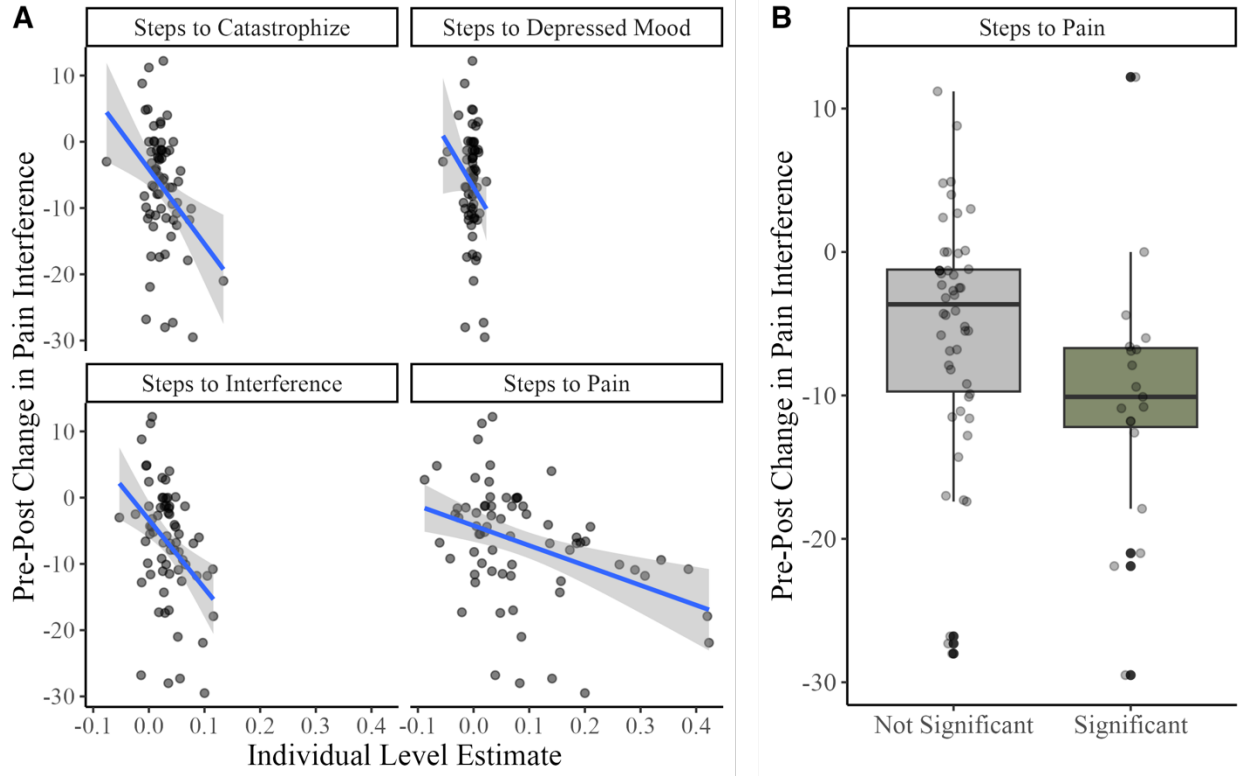


Figure 7. Associations between dynamic effects involving consequences of activity and postoperative improvement in pain interference.

Discussion

The goal of this study was to identify biopsychosocial mechanisms of CBP that might improve understanding and prediction of which treatments are most likely to maximize benefit and minimize risk for a given patient. In a sample of patients with CBP receiving spine surgery, I found substantial heterogeneity in dynamic, within-person relationships between physical, emotional, and cognitive symptoms. In contrast to my pre-registered hypotheses, these individual differences were not robustly associated with severity of pain-related or psychosocial symptoms pre-surgically. However, the strength of dynamic relationships between physical activity and subsequent subjective reports of pain, interference, and catastrophizing were predictive of postsurgical improvement in pain interference. Implications of these findings for precision pain medicine are discussed below.

First, this study is the first to my knowledge to empirically test the cognitive-behavioral model of CBP using dynamic data. Numerous biopsychosocial theories have posited that pain is bidirectionally associated with physical, emotional, and cognitive symptoms (Gatchel, 2004; Rudy et al., 1988; Turk et al., 1983). In the current study, I tested these bidirectional associations using EMA and Fitbit data. At the same occasion (e.g., contemporaneously), increased pain severity was indeed associated with increased depression mood, catastrophizing, and interference for individuals on average. Increased activity in the hour prior to EMA was also associated with increased pain, interference, and catastrophizing for individuals on average. However, activity was not clearly associated with subsequent depressed mood, nor did any subjective symptoms prospectively predict activity at the group level.

For the subjective variables, most exhibited bidirectional relationships over 3-hour lags (e.g., increased pain predicted increased catastrophizing at the next assessment, and vice versa). However, there was an important exception: Pain *interference*, but not pain *severity*, was bidirectionally associated with increased depressed mood over 3-hour lags. For several decades, cognitive-behavioral models of chronic pain have suggested that pain leads to depression through internal factors such as perceived life interference, perceived control, and maladaptive thoughts (Rudy et al., 1988; Turk et al., 1995; Wood et al., 2013). Here, we see evidence for several of these relationships at the group level. For example, pain severity predicted subsequent interference, which predicted subsequent depressed mood. Pain severity also predicted subsequent catastrophizing, which predicted subsequent depressed mood. Given that prior tests of these theories have been overwhelmingly cross-sectional, it is important to see these effects from dynamic data. Future studies should examine whether internal factors indeed dynamically *mediate* associations between pain and depressed mood (c.f., McNeish & Mackinnon, 2022).

Regarding my first primary hypothesis, I did find evidence of individual differences in dynamic relationships among physical, emotional, and cognitive symptoms. Both the strength and direction of relationships among activity, pain, interference, catastrophizing, and depressed mood varied across individuals. No lagged relationships were statistically significant for > 28% of participants. Many, including prospective relationships between pain and depressed mood, were significant for very few individuals. These findings are consistent with prior warnings that group-level or fixed effects may not be representative of many individuals in the group (Barlow & Nock, 2009; Fisher et al., 2018; Hamaker, 2012; Levine et al., 1992). Consequently, treatment decisions that are made based on what is true of the group on average may not have the intended effects on within-person processes.

It is possible, however, that such heterogeneity will be useful for tailoring treatment to the individual. In the current study, there was some evidence that individual differences in dynamic relationships among physical, emotional, and cognitive symptoms were associated with hypothesized patient characteristics or surgical outcomes. For example, the covariance between depressed mood and pain interference was positively associated with severity of depression, anxiety, catastrophizing, and pain intensity. Although these effects are consistent with biopsychosocial theories of CBP, they were not associated with surgical outcomes.

Over the past 15 years, the network theory of psychopathology has gained significant popularity as a potential means of understanding psychological disorders (Robinaugh et al., 2020). This theory suggests that disorders such as depression arise from causal relationships among symptoms (Borsboom & Cramer, 2013; Cramer et al., 2016). As such, individuals with stronger relationships among symptoms should exhibit worse symptom severity, and networks should become less dense with effective treatment. Unfortunately, the empirical evidence for this theory has been highly mixed (Pe et al., 2015; Schweren et al., 2018; Shin et al., 2022), leading to concerns over whether such methods can be used to uncover causal relationships as hoped (Huang et al., 2023; Ryan et al., 2022). The results of the current study add to these mixed findings. On the one hand, some dynamic effects, such as the covariance between pain severity and depressed mood, were associated with severity of depressive and pain symptoms. Additionally, individuals with more significant paths exhibited somewhat worse psychosocial and physical functioning pre-surgically. However, these effects were generally small and absent for many of my preregistered specific hypotheses.

Importantly, it is possible that such associations do exist, but that the current models were insufficient to uncover the underlying causal system. The models I constructed were based on

verbal theories, which describe hypothesized relationships using words. Although verbal theories are the norm in psychology, other fields such as ecology and physics rely on much more precise *formal theories* expressed using mathematical or computational models (Robinaugh et al., 2021; Smaldino, 2017). The problem with verbal theories is that they are subject to many interpretations. For example, over what time scale do we expect pain to affect depressed mood, or vice versa? Is this time scale consistent across individuals? Does it vary across contexts? What size effects do we expect? These are difficult questions that lack simple solutions. However, they are critical to study design, as data collected every 3 hours will be insufficient to uncover effects that occur over shorter time scales (Haslbeck & Ryan, 2022). It is also extremely difficult to determine whether data support or refute a theory if the theory itself is unclear.

On the other hand, the results of the current study offer optimism regarding the utility of combining subjective and passively collected objective data to improve understanding and prediction of treatment outcomes. Dynamic relationships between activity and subsequent subjective reports emerged as larger and more robust predictors of postoperative changes in pain interference. In support of my exploratory hypothesis, I observed greater postoperative improvements in pain interference among individuals for whom increased activity was more strongly associated with subsequent pain severity, interference, and catastrophizing.

Incorporating passively collected data has several advantages in this context. First, passive data is more accurate than subjective reports *if* the construct of interest can be measured objectively. That is, pain is an inherently subjective experience (Merskey & Bogduk, 1986), and should therefore continue to be measured using self-report. However, activity variables such as step count can be measured objectively using wearable devices, and this measurement is less subject to recall bias than self-report (VandeBunte et al., 2022). Objective assessment also

reduces concerns that participants may respond differently to one another, or across contexts, as data are collected in the same way across individuals and across time. Additionally, passive data allows for greater flexibility in addressing issues such as time scale, as data are continuously collected and can be examined across a range of timescales. In this way, objective data increase flexibility and confidence in our ability to uncover causal relationships, contributing to their utility for precision medicine.

The current study had several strengths. First, I used multiple modalities of assessments, including one-time self-report, EMA, and passively collected wearable data in a treatment-seeking sample of adults with CBP. EMA and passive data collection provided a very large number of assessments, which I could then use to empirically test dynamic relationships between physical, emotional, and cognitive components of CBP. Prior examinations of biopsychosocial theories of CBP have been overwhelmingly cross-sectional, which precludes our ability to draw conclusions about causality (Cole & Maxwell, 2003; Maxwell et al., 2011; Maxwell & Cole, 2007; McNeish & Mackinnon, 2022; Preacher, 2015). I also used static approaches that allowed me to examine individual differences in these dynamic relationships. Whereas traditional multi-level approaches allow for estimation of group-level effects and variability from such effects based on fewer data points per person, such estimates are subject to bias when individuals vary from one another or within themselves over time, both of which are common in symptoms relevant to CBP (Mak & Schneider, 2020; Stone et al., 2005; Wright & Woods, 2020). Dynamic structural equation modeling reduces such biases (Asparouhov et al., 2018b).

There are also limitations. All participants were recruited from a single academic hospital system in the Midwest, and the sample was overwhelmingly White and non-Hispanic. Spine surgery tends to be more common among White individuals (Marquez-Lara et al., 2014;

Sielatycki et al., 2015). Nonetheless, it is critical to replicate these results in diverse samples, especially given evidence that biopsychosocial mechanisms may differ between racial and ethnic groups (Meints et al., 2019). Participants were also only included if they owned a smartphone and agreed to complete EMA. It is possible that participants who agreed to this protocol differ from the larger population of individuals with CBP. In particular, the current sample had lower depression symptom severity than often reported in CBP samples (Currie & Wang, 2004). Relatedly, EMA of depressed mood was highly positively skewed. Normality is an assumption of DSEM, and the implications of violating that assumption are unclear. It is therefore important for future work to dynamically test antecedents and consequences of depressed mood among individuals with greater depressive symptoms. It is also important for future work to examine the predictive utility of dynamic features over longer time periods.

Finally, this study leaves several open questions for future research. In the current study, the most robust predictors of spine surgery outcomes were dynamic relationships between step count and subsequent pain, interference, and catastrophizing. It is extremely common for patients with CBP to avoid physical activity because they are afraid movement will exacerbate pain (Crombez et al., 2012; Meulders, 2019). However, the results of this study show that for over two-thirds of patients receiving spine surgery, increased steps did not dynamically predict increased pain. Furthermore, these individuals were at greater risk for poor surgical outcomes. Given difficulties in determining whether degenerative physiological findings are the cause of CBP (Brinjikji et al., 2015; Hall et al., 2021), dynamic relationships between activity and subjective experience could serve as an important tool for determining who is at risk for poor outcomes after spine surgery. These data could also provide data-driven education for patients considering spine surgery and potentially suggest adjunctive psychosocial interventions. That is,

in many cases, dynamic data could provide counterevidence to patient beliefs that being more active increases pain. Importantly, step count is only one facet of activity, and it is possible that for some individuals, other physical exertions (e.g., lifting or bending) increased pain. As wearable technology continues to evolve, it will be critical to develop new and innovative features that can dynamically capture potential triggers in patients' daily lives. Dynamic data could also be used to identify patient-specific psychosocial drivers of pain (e.g., catastrophizing) that can potentially be addressed using brief, targeted interventions.

Overall, the results of the current study add to a growing body of literature suggesting that individuals differ from one another in biopsychosocial mechanisms involved in CBP. This study also adds unique findings that some individual differences in dynamic relationships between physical, emotional, and cognitive symptoms are associated with symptom severity and surgical outcomes. There is significant potential for dynamic relationships, particularly those involving objective variables, to be harnessed to develop and test precision interventions for chronic back pain.

Appendix A. EMA Items

Please rate each item on a scale from 0 (none) to 100 (worst possible) based on how you feel right now (that is, right before you started answering these questions).

1. How intense is your back pain?
2. How intense is your leg pain?
3. How intense is your overall pain?
4. How much is pain interfering with your enjoyment of life?
5. How much is pain interfering with your activities?
6. How much is pain interfering with your ability to concentrate?
7. How depressed are you feeling?
8. How hopeless are you feeling?
9. How worthless are you feeling?
10. I keep thinking about how much I hurt.
11. My pain overwhelms me.
12. I am afraid that my pain will get worse.

[During the last survey questionnaire of the day]

1. How many of your opioid pills did you take on an “as-needed” basis today?

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