Heterogeneity in temporal dynamics of pain and affect among individuals with chronic back pain
and associations with risk for future opioid-related problems

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Abstract

Chronic low back pain is a common and heterogeneous disease defined by sensory and affective experiences. In pre-registered analyses, we examined variability in within-person relationships between negative affect (NA) and physical pain over time using multi-level dynamic structural equation modeling. We further examined how this variability relates to risk for future opioid-related problems as assessed by the Screener and Opioid Assessment for Patients with Pain-Revised. Patients with chronic low back pain ($N = 87$) completed an average of 94 Ecological Momentary Assessment surveys each over two weeks. In the group-level model, lagged pathways suggested that increased pain predicted increased NA two hours later ($\beta = 0.10, p < .001$), and increased NA predicted increased pain two hours later ($\beta = 0.16, p < .001$) for individuals on average. However, there was significant variability in these effects, such that NA significantly predicted pain two hours later for 48% of participants, and pain significantly predicted NA two hours later for 8% of participants. In a multiple regression analysis, risk for future opioid-related problems was positively associated with the prospective relationship of pain predicting increased NA two hours later ($b = 8.05, p = .007$) and the individual-level correlation between pain and NA ($b = 1.99, p = .002$). These results suggest that variability in within-person symptom dynamics may help identify chronic pain patients who are at greater risk of opioid-related problems. Future research should examine whether personalizing treatment based on relationships between pain and affect can mitigate opioid-related risks.

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Pain is a biopsychosocial phenomenon defined by sensory and affective experiences [21,54,55]. For decades, there has been an interest in disentangling complex relationships between pain and negative affect (NA), especially given that individuals who experience more NA tend to experience worse pain and poorer functioning [5,20,22,33,36]. Chronic pain patients who experience increased levels of NA are also more likely to engage in problematic substance use and more likely to die by suicide [1,38,53,59]. Thus, it is critical to better understand relationships between pain and NA among individuals with chronic pain.

Personalized or precision initiatives are becoming increasingly common given the frequent heterogeneity seen among individuals with the same condition [2,11,29]. Although personalization is often based on biological variables, many chronic pain and mental health conditions lack clear biological mechanisms or corresponding treatments [14,44]. As such, variability in within-person relationships between perceived pain and affect are promising targets for more precise treatment of chronic pain and co-occurring symptoms [34]. For example, patients who experience stronger associations between pain and affect may be more likely to benefit from psychological treatments designed to build skills for more adaptively managing NA.

Within-person relationships can be assessed using intensive longitudinal sampling approaches such as Ecological Momentary Assessment (EMA) that allow for repeated assessments within patient's daily lives via portable devices [40,49,51]. EMA studies have found that momentary increases in pain are associated with concurrent increased NA, and vice versa, across several chronic pain conditions [20]. However, fewer studies have examined prospective

relationships between pain and NA over time (hours or days), and results have been highly mixed [7,10,17,23,27,39].

Critically, none of these studies have taken a personalized approach to examining prospective relationships between pain and NA. Multiple studies have shown that within-person relationships between pain and NA range from negative or non-significant to strong and positive across different individuals [19,42,57]. However, standard practice in the field is to report average or fixed effects. A recent study found that individuals with more severe depressive and anxiety symptoms also experienced stronger concurrent relationships between pain and affect [37]. If NA *prospectively* predicts pain more strongly for some individuals than others, this may suggest a means of identifying patients for whom psychological interventions are most likely to reduce pain symptoms through the reduction of NA.

In the current study, we examined variability in within-person prospective relationships between pain and NA among patients with chronic pain using multilevel residual dynamic structural equation modeling (ML-RDSEM). First, we hypothesized that pain and NA would prospectively predict one another for the group on average. Second, we hypothesized that there would be significant variability in these effects across individuals, such that some patients would show stronger associations than others. Finally, we hypothesized that individuals who were at higher risk for future opioid-related problems would exhibit stronger relationships between affect and pain symptoms. In addition to these pre-registered confirmatory hypotheses, we explored potential moderators of any relationships between pain and NA with significant between-person variability.

Method

Participants

Participants were patients with chronic low back pain recruited from local pain management clinics and the general community in the Midwest ($N = 87$). Participants were recruited as part of a study that aimed to examine bidirectional relationships between pain, affect, and substance use. Thus, participants were recruited who used prescribed opioid medication at least every-other-day, used alcohol at least two times per week, used both alcohol and prescribed opioid medication, or did not take opioid medication or drink alcohol. Results regarding the portion of the sample who were taking prescribed opioids have been previously reported, although variability in within-person relationships was not examined in this previous study [9]. To be eligible for participation, individuals were required to be between 18 and 60 years old and be in current treatment for chronic low back pain that had persisted at least 6 months with a past-month pain severity rating of at least 3 (out of 10) on a verbal numeric pain intensity scale. Individuals were excluded who had current psychosis, intellectual disability, neurological dysfunction, or significant head trauma history; were currently in treatment or interested in treatment for substance use disorder; had significant and/or frequent prescription deviations (e.g., regularly refilling prescriptions early), evidence of drug-seeking behavior (e.g., prescriptions from multiple doctors), or significant concern from family members or physicians about their opioid use; or had past-year unsuccessful attempts to reduce drinking or physiological withdrawal symptoms (indicators of severe alcohol use problems among those drinking alcohol). Given ethical considerations regarding consuming alcohol while pregnant, people who reported being pregnant or planning to become pregnant were excluded for consistency across the sample.

Procedure

All procedures were approved by the Institutional Review Board. Interested individuals completed an initial phone screen to determine eligibility. Eligible participants completed an in-person interview regarding their medical and psychiatric history, including medication use. They next completed an orientation session, consisting of self-report questionnaires, a brief experimental acute pain task, and in-depth training on the study-provided Android smartphone and EMA application. Participants then completed EMA reports via smartphone. Over a two-week period, participants received a maximum of 6 surveys per day, which were randomly stratified across waking hours following a participant-initiated morning survey (or noon if participants did not complete a morning survey). Participants could also initiate drinking and pain medication reports when they drank alcohol or took opioid pain medication and would receive follow-ups after alcohol or opioid use. Follow-ups occurred at set intervals (30, 60, and 120 minutes) following an initial opioid use report. If participants reported additional opioid or alcohol use at 120 minutes, an additional follow-up prompt was scheduled 60 minutes later. All completed prompts were included in the current study. Additional detail on the EMA procedure is presented in the previous publication from this dataset [9].

Measures

Baseline characteristics. Several measures were administered to assess clinical characteristics prior to the initiation of the EMA procedure. The 21-item Depression Anxiety Stress Scales [35] were used to assess levels of depression and anxiety. Depression diagnoses were also determined by the clinician-administered Mini International Neuropsychiatric Interview version 7.0 [47]. Risk for future opioid-related problems was assessed by the Screener and Opioid Assessment for Patients with Pain-Revised (SOAPP-R), a 24-item measure that has

been shown to be predictive of future aberrant drug behaviors [8,45]. Patient-Reported Outcomes Measurement Information System (PROMIS) measures were used to assess pain behavior, physical function, and pain interference. These measures are considered the gold-standard in pain assessment due to the combination of low patient burden and high clinical validity [3,15]. Tendency to catastrophize about pain was assessed using the Pain Catastrophizing Scale [PCS; 52], a 13-item self-report measure assessing thoughts about pain. Pain affect was assessed using the Pain Discomfort Scale [PDS; 31], a 10-item self-report measure assessing negative affect associated with pain. Trait anger was assessed using the Spielberger Trait Anger scale [48]. Finally, tendency to suppress and cognitively reappraise emotions was assessed using the Emotion Regulation Questionnaire [25].

EMA items. At each prompt, participants rated their physical pain using a single item assessing physical pain intensity over the previous 15 minutes on a scale from 0 (no pain) to 10 (worst possible pain). Participants also rated affective components of pain, including unpleasantness, annoyance, and interference with enjoyment of life on a scale from 1 (very slightly or not at all) to 5 (extremely). Also at each prompt, a shortened version of the Profile of Mood States [12,41] was used to assess feelings of depression (sad, hopeless, discouraged), anxiety (anxious, on edge, uneasy), and anger (angry, resentful, annoyed) on a scale from 1 (very slightly or not at all) to 5 (extremely). Additional EMA items were administered but are not described here as they are not relevant to the current analyses. Construct validity was assessed as described below.

Data Analytic Plan

Data analysis was conducted in Mplus version 8.5 [43] and R version 4.0.3. A detailed data analytic plan was pre-registered on Open Science Framework prior to undertaking analyses

and prior to the first or third authors (MRF and TLR) accessing the data set (see <https://osf.io/9dmq2/>).

Construct validity. Multi-level confirmatory factor analysis (ML-CFA) was conducted to confirm the within-person factor structure of the items. Good fit was defined as comparative fit index (CFI) $\geq .90$, Tucker–Lewis index (TLI) $\geq .90$, and root mean square error of approximation (RMSEA) $< .08$ [6,28,56].

Determining empirical lags. Next, we determined the appropriate timescales on which to examine relationships between pain and affect symptoms. Given that participants completed EMA prompts at randomly stratified intervals, all observations were binned into the most common interval, which corresponded to two hours. If multiple observations occurred for an individual within a two-hour period, these observations were averaged. We then used the DTVEM package in R [30] to empirically determine optimal lag lengths (e.g., 2 hours, 4 hours, 6 hours, etc.) based on Differential Time-Varying Effect Models.

Assessing relationships between pain and affect. The primary hypotheses were evaluated using multilevel residual dynamic structural equation modeling (ML-RDSEM) in Mplus [4]. Like traditional multilevel modeling, ML-RDSEM allows for the data dependencies inherent to EMA (i.e., timepoints nested within individuals). However, ML-RDSEM also has several advantages over more traditional forms of multilevel modeling. First, as an extension of structural equation modeling, ML-RDSEM can include both observed and latent variables, thus accounting for measurement error. ML-RDSEM can also estimate reciprocal effects (e.g., the effect of X on Y and Y on X) within the same model, rather than requiring separate models for each dependent variable. Additionally, through Bayesian estimation, ML-RDSEM handles missing data and allows for more interpretable estimation of variability around group-level

pathways (e.g., through credible intervals). Uncertainty around group- and individual-level estimates can be used to examine within- and between-person variability, which was the primary focus of the current study.

All composited variables were standardized to allow for comparison across scales. To account for potential effects of time, each variable was predicted by day of the study and survey position within day. Each variable was also predicted by both itself and the other variables at the previous lags (identified through DT-VEM). All variables (except day and survey position) were allowed to correlate. This model provided the group-level estimates of within-person relationships between pain and affect variables. A 95% credible interval that did not contain zero indicated a significant relationship between two variables. To assess between-person variability in these relationships, variance of the model parameters was examined. A 95% credible interval that did not contain zero indicated significant between-person variability in within-person relationships. ML-RDSEM also provides individual-level estimates, which give a more detailed understanding of variability across the sample. In addition to individual-level estimates of all lagged pathways, an estimate of the correlation between pain and NA was extracted for each individual to examine variability in the within-person association between pain and NA.

After outputting individual-level estimates from the primary ML-RDSEM model, risk for opioid-related problems (as assessed by the SOAPP-R) was regressed on individual-level concurrent and prospective paths between pain and affect variables using the `lm()` function in R. Finally, in exploratory analyses, we examined potential moderators of individual-level relationships between pain and affect variables. Given the exploratory nature of these analyses, effects with a $p < .01$ were considered significant.

Results

Demographics and clinical characteristics are presented in Table 1. The sample was comprised of 87 individuals, most of whom were female ($n = 64$, 75%) and White ($n = 78$, 90%). Of the full sample, 27 (31%) used only alcohol, 27 (31%) used only prescribed opioids, 7 (8%) used both alcohol and opioids, and 26 (30%) used neither. The average age was 40 years old ($SD = 12$, $\min = 19$, $\max = 60$). Over the course of the EMA period, participants experienced an average pain intensity of 4 on a scale from 0 to 10 ($SD = 2$). Based on the PROMIS measures administered at baseline, participants experienced high levels of pain interference (M t -score = 61) and pain behaviors (M t -score = 60) and worse physical functioning (M t -score = 41) compared to the general population. Based on the DASS administered at baseline, participants also reported mild depressive symptoms ($M = 12.29$, $SD = 5.49$) and moderate anxiety symptoms ($M = 10.89$, $SD = 4.35$) on average, though there was substantial variation in these domains. At the time of enrollment, 14 participants (16%) met criteria for current MDD and 40 (46%) met criteria for lifetime MDD. Across all 87 participants, 7567 of 8169 possible EMA prompts were completed. Participants completed an average of 94 surveys each ($SD = 18$, $\min = 29$, $\max = 136$), for an average adherence rate of 93% ($SD = 6\%$, $\min = 77\%$, $\max = 100\%$).

Construct Validity

The initial ML-CFA model included four factors: pain (comprised of the one physical pain intensity and three physical pain affect items) and depressed, anxious, and angry mood (each comprised of three items). This model failed to converge, likely due to low variability in the individual mood items. Consequently, affect items were composited into depressed, anxious, and angry mood sum scores, which increased variability, and these sum scores were loaded onto a single NA factor that was correlated with the pain factor. The resulting model had excellent fit

(CFI = 1.00, TLI = 1.00, RMSEA = 0.00). The remaining analyses were thus conducted with NA and pain composite variables (generated by summing relevant variables, see <https://osf.io/9dmq2/> for details). Both variables had sufficient variance and normality for modeling based on visual inspection.

Determining Empirical Lags

DT-VEM did not identify any lags for pain predicting NA. For NA predicting pain, the model identified lags 1 and 2 (e.g., approximately 2 and 4 hours). Thus, relationships between pain and affect were examined over these lags.

Assessing Within-Person Relationships between Pain and Affect

Results of the ML-RDSEM analysis are presented in Table 2. Both pain and NA had a tendency to persist over two- and four-hour lags, as evidenced by significant autoregressive effects (β s = 0.080 – 0.441, $ps < .01$). There were also time trends noted, such that pain had a tendency to decrease over the course of each day ($\beta = -0.083$, $p < .001$) and over the course of the study ($\beta = -0.047$, $p = .002$). These time trends are partialled out of remaining effects.

The average individual-level correlation between pain and negative affect was moderate ($r = .37$). Lagged pathways suggested that increased pain predicted increased NA two hours later ($\beta = 0.10$, $p < .001$), and that increased NA predicted increased pain two hours later ($\beta = 0.16$, $p < .001$) for individuals on average. Neither variable significantly predicted the other over four-hour lags.

Variability in Within-person Relationships between Pain and Affect

All within-person pathways significantly varied between individuals ($ps < .001$). This variability is visualized in Figure 1. Based on individual-level estimates, all participants had a significant autoregressive effect of NA over two hours, as indicated by a 95% credible interval

that did not contain zero. The other significant effects were present for some but not all individuals. Most participants ($n = 78$, 90%) had a significant autoregressive effect of pain over two hours. Nearly half of participants ($n = 42$, 48%) had a significant effect of NA predicting pain two hours later, and fewer participants ($n = 7$, 8%) had a significant effect of pain predicting NA two hours later. The strength of these paths also varied between participants. For example, the within-person correlation between pain and NA ranged from .02 to .97 ($M = .37$, $SD = .22$). Thus, there was considerable between-person variability in within-person relationships between pain and affect.

Association with Risk for Future Opioid-Related Problems

In a multiple regression analysis, risk for opioid-related problems, as assessed by the SOAPP-R, was regressed on all individual-level pathways. The overall model was significant and explained 33% of the variance in risk for opioid-related problems, $F(9, 77) = 5.69$, $p < .001$. Results suggested that risk for opioid-related problems was significantly and positively associated with the prospective relationship of pain predicting increased NA two hours later ($b = 8.05$, $p = .007$) and the individual-level correlation between pain and NA ($b = 1.99$, $p = .002$). Thus, individuals who were at higher risk of future opioid-related problems demonstrated both a higher tendency for pain to predict NA over two hours and a stronger concurrent association between pain and NA.

Moderators of Individual-Level Relationships

Several potential moderators of individual-level relationships were examined using multiple regression, wherein each individual-level pathway (e.g., the prospective relationship from pain to NA) was regressed on all potential moderators (e.g., baseline characteristics). These results are considered exploratory due to the multiple tests and lack of specific a priori

hypotheses. For the autoregressive effects, individuals who used opioids exhibited a lesser tendency for pain to persist over two-hour lags ($b = -.13, p < .001$). Individuals who endorsed a greater tendency to suppress emotions also exhibited a lesser tendency for pain to persist over four-hour lags ($b = -.02, p = .006$).

For the remaining individual-level pathways, no potential moderators were significant predictors in multiple regression analyses examining all predictors together. As this finding may be due to high correlations among predictors (see Figure 2), we also examined pairwise correlations among individual-level pathways and potential predictors. Pain predicting NA two hours later was positively correlated with several symptom measures, including pain behavior, pain interference, pain catastrophizing, and DASS depression ($r_s = .36 - .40, p_s < .01$), and negatively correlated with physical functioning ($r = -.32, p < .01$). Similarly, the within-person correlation between pain and NA was positively correlated with pain behavior, pain interference, pain catastrophizing, anxiety, and depression ($r_s = .38 - .59, p_s < .01$) and negatively correlated with physical function ($r = -.50, p < .01$). NA predicting pain two hours later was also positively correlated with expressive suppression ($r = .28, p < .01$), and NA predicting pain four hours later was positively correlated with pain discomfort ($r = .30, p < .01$).

Discussion

Intensive sampling approaches such as EMA have shown significant promise for real-time assessment of pain experiences in participant's daily lives [40,49,51]. The vast majority of research in this area has focused on what is true for individuals on average (i.e., fixed effects). However, EMA approaches often provide sufficient data to examine what is true for a given individual, and how individuals differ from one another with regard to symptom dynamics [18,26]. In the current study, increased pain predicted increased negative affect approximately

two hours later, and increased negative affect also predicted increased pain for the group on average. However, there was significant variability in these relationships, such that pain significantly predicted affect for only a few individuals in the sample, whereas affect predicted pain for nearly half the sample.

There are multiple plausible explanations for why a group effect would be observed in the absence of many individuals showing prospective associations of pain and affect. One possibility is that more individuals within our sample would have shown these effects if we had had greater power to test the effect, which could have been achieved through collecting additional time points. However, consistent with prior research [19,37,42,57], the current study highlights the fact that individuals differ from one another in the degree to which pain and affect tended to persist, co-occur, and predict one another over time. For example, whereas some individuals demonstrated no relationship between pain and affect at the same point in time, others demonstrated a very strong correlation. Although group-level or fixed effects are often assumed to be a summary of individual-level relationships, they could reflect a minority of individuals with strong relationships. This hypothesis is supported by the results of the current study, in which individuals demonstrated significant variability from one another with regard to the dynamic relationships between pain and affect.

Importantly, variability in the relationship between pain and negative affect appeared meaningfully related to diagnostic and clinical indicators. For example, pain had less of a tendency to persist among individuals who were using prescribed opioids, plausibly because opioid medication relieved their pain. This interpretation would be consistent with the effects found by Carpenter et al. [9], in that opioid use was prospectively associated with reduced pain in the portion of this sample that took prescribed opioids. Perhaps more importantly, individuals

who exhibited a stronger correlation between pain and negative affect or had a higher tendency for pain to predict negative affect over two hours were at higher risk of future opioid-related problems as assessed by the SOAPP-R. Group-level research has identified numerous risk factors for opioid-related problems among individuals with chronic pain, including depression [24,58]. Despite these efforts, it remains difficult for clinicians to identify the subset of patients who are most likely to use opioid medication in ways other than as prescribed or develop a use disorder. Examining individual-level relationships among variables including affect, pain, and opioid use is a promising future direction for treatment personalization that maximizes benefit and minimizes harm. For example, in the subset of the current sample that used opioids, individuals at high risk for opioid-related problems took larger opioid doses following pain, but also experienced smaller subsequent reductions in pain and negative affect [9]. Further research is necessary to understand whether relationships between pain and affect as assessed by EMA can be used to identify risk prior to opioid prescription.

In addition to identifying risk for opioid-related problems, individual-level relationships may be helpful in identifying targets for precision pain medicine. Precision medicine approaches often rely on the identification of biological mechanisms that can be targeted pharmacologically [2,11]. Progress is being made toward identifying specific biomarkers involved in chronic pain conditions [16,44]. However, there is not currently a clear taxonomy of biomarkers and readily applicable treatments. Furthermore, pain is an inherently subjective experience that is often influenced by psychological experiences [21]. Therefore, precision medicine approaches may be highly useful for determining who might be most likely to respond to psychological interventions. In the current study, we found that, for nearly half the sample, negative affect predicted increased pain two hours later. Furthermore, there was preliminary evidence that this

relationship appeared stronger among individuals with a greater tendency to suppress emotions, which has been shown to be associated with negative emotional, social, and physiological consequences [13,32,46]. Thus, psychological interventions that reduce negative affect through increasing the use of healthier coping strategies may relieve both physical and emotional suffering for these individuals.

The current study had several strengths. First, we were able to examine variability in individual-level relationships because the number of observations per subject was larger than that often obtained in EMA research [40]. Whereas traditional multi-level approaches allow for estimation of group-level effects and variability from such effects based on fewer data points per subject, 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 chronic pain [37,50,60]. The analytic approach used here reduces such biases through its estimation procedures [50].

The current study also has limitations. Although chronic pain is most common in older adulthood, our sample was comprised mostly of individuals in middle-adulthood. The sample was also primarily female and White, which may limit the generalizability of the findings. Given limited existing guidance on power for this type of analysis [4], the number of time points or number of subjects may also have been insufficient to uncover smaller effects. Finally, although our results suggest that within-person relationships between pain and affect may be related to risk for opioid-related problems, further research is necessary to determine whether such relationships are indeed associated with the development of opioid use disorder.

In summary, the current study highlights the need to examine variability in individual-level relationships between pain and psychosocial variables involved in chronic pain conditions.

Although there was a bi-directional relationship between pain and negative affect for the group on average, there was significant variability in these effects that was associated with important clinical characteristics, including risk for opioid-related problems. Future research should examine whether such variability is associated with treatment outcomes, which would suggest the utility of individual-level relationships in identifying precision treatment targets.

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Table 1. Demographics and clinical characteristics

Variable	<i>M (SD)</i>
Age	39.57 (11.55)
PROMIS Pain Interference	61.13 (7.59)
PROMIS Pain Behavior	59.65 (4.64)
PROMIS Physical Function	41.2 (8.39)
Pain Catastrophizing	33.02 (13.05)
DASS Anxiety	10.89 (4.35)
DASS Depression	12.29 (5.49)
ERQ Cognitive Reappraisal	4.9 (1.34)
ERQ Expressive Suppression	3.69 (1.29)
PDS	3.11 (0.51)
SOAPP-R	21.41 (13.02)
Trait Anger	1.75 (0.53)
<i>n (%)</i>	
Gender (Male)	23 (26.44)
Race	
White	78 (89.66)
Black	5 (5.75)
Asian	4 (4.60)
MDD lifetime	40 (45.98)

Note. PROMIS = Patient-Reported Outcomes Measurement Information System; DASS = Depression Anxiety Stress Scales; ERQ = Emotion Regulation Questionnaire; PDS = Pain Discomfort Scale; SOAPP = Screener and Opioid Assessment for Patients with Pain-Revised

Table 2. Average Within-person Estimates

Path	Standardized Estimate	Posterior SD	95% Credible Interval	<i>p</i>
Day in study predicting pain	-0.047	0.016	-0.077 - -0.017	0.002
Time of day predicting pain	-0.083	0.013	-0.107 - -0.059	0.000
Day in study predicting NA	0.010	0.016	-0.022 - 0.042	0.275
Time of day predicting NA	0.000	0.012	-0.024 - 0.022	0.498
Pain autoregressive (2 hours)	0.354	0.019	0.317 - 0.391	0.000
Pain autoregressive (4 hours)	0.080	0.020	0.04 - 0.121	0.000
NA autoregressive (2 hours)	0.441	0.022	0.399 - 0.484	0.000
NA autoregressive (4 hours)	0.094	0.026	0.041 - 0.144	0.001
NA predicting pain (2 hours)	0.155	0.021	0.115 - 0.196	0.000
NA predicting pain (4 hours)	-0.005	0.020	-0.046 - 0.033	0.405
Pain predicting NA (2 hours)	0.100	0.021	0.057 - 0.141	0.000
Pain predicting NA (4 hours)	-0.014	0.021	-0.054 - 0.026	0.251

Note. SD = standard deviation; NA = negative affect; *p* values are one-tailed

Figure 1. Variability in Individual-Level Symptom Dynamics

Note. This plot shows variability in the within-person autoregressive paths (e.g., the tendency of pain and affect to predict themselves over time) and prospective paths (e.g., the tendency of pain to predict affect and vice versa over time). Each dot represents an individual's standardized median estimate, and the black line signifies the 95% credible interval around that estimate. h = hours; NA = negative affect.

Figure 2. Correlations among Individual-level Paths and Self-report Measures

Note. Variables 1-9 represent the strength of individual-level pathways generated through EMA and ML-RDSEM analyses; variables 10-21 are baseline characteristics generated through traditional self-report.