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Individual Differences in Momentary Pain-Affect Coupling and Their Associations with Mental Health in Patients with Chronic Pain

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Abstract

Objective—Pain and affect are generally associated. However, individuals may differ in the magnitude of the coupling between pain and affect, which may have important implications for their mental health. The present study uses ecological momentary assessments (EMA) to examine individual differences in momentary pain-affect coupling and their associations with depressive and anxiety symptoms.

Methods—This study is a secondary data analysis of three primary EMA studies. Participants were a total of 290 patients with chronic pain. Results were synthesized across studies using meta-analytic techniques.

Results—Individuals whose pain was more strongly concurrently coupled with affect (positively associated with negative affect or negatively associated with positive affect) reported higher levels of depressive and anxiety symptoms. Results from lagged analyses suggest that individual differences in affect reactivity to pain were not significantly associated with depressive or anxiety symptoms.

Conclusion—These findings suggest that individuals with greater concurrent coupling between pain and affect experience more mental health problems. Potential avenues for future research include intervention strategies that target the decoupling of pain and affect experiences in patients with chronic pain.

Keywords

Affect; pain; depression; anxiety; ecological momentary assessment

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Introduction

Chronic pain affects about 20% to 30% of adults in the United States [1,2]. Individuals with chronic pain not only are more likely to report poorer physical health, but also poorer mental health [3,4]. Depressive and anxiety disorders are two of the most common mental health problems co-occurring with chronic pain [5–7]. Patients with chronic pain and comorbid depression or anxiety are more likely to have poorer treatment response, prognosis, and social functioning, as well as higher levels of functional limitations and disability [8,9].

Pain and affect are closely related. There has been a long-standing interest in understanding the relationship between pain and affect [10,11]. The majority of the existing research examines the relationship between chronic pain and mental disorders globally from a between-person perspective [3,4]. Relatively less research has examined the within-person relationship between pain and affect in individuals' day-to-day life in their natural environment. The available evidence suggests that individuals report more negative affect (NA) and less positive affect (PA) on days or weeks when they experience more pain than on days or weeks when they experience less pain [12,13]. Furthermore, individuals' pain from yesterday has been found to predict more NA (but not PA) the next day [13].

Although pain and affect are often associated within persons, individuals do differ in the magnitude of association. A recent study on older adults showed that there are individual differences in the same-day coupling between pain and NA, and found that individuals with stronger same-day coupling between pain and NA (but not PA) are more likely to have poorer health conditions [13]. However, whether individual differences in affect reactivity to pain at the momentary level have implications for mental health remains unknown.

There are good reasons to believe that higher affect reactivity to pain is related to poorer mental health outcomes. First, prior research has found that individuals differ in the degree to which they react to daily stressors and that higher reactivity to daily stressors is associated with poor physical and mental health [14–16]. Second, although it is natural and adaptive for pain to induce some fear in individuals (a signal to avoid danger), some people fear pain more than others and intense fear may not be adaptive, especially in chronic pain situations. Fear may lead individuals to avoid participating in daily activities that may cause pain in the short-run, but would improve their health conditions in the long run [17,18], and limited daily and social activities in these individuals are likely to have a toll on their mental health. Lastly, some individuals are more likely to catastrophize about their pain and health condition and pain catastrophizing has been found to be associated with poor mental health outcomes in numerous studies (e.g., [19,20]). High catastrophizing individuals often ruminate, magnify, or feel helpless about their pain and health conditions [21,22]. In fact, intensified affective responses to pain have been suggested as one possible mechanism through which catastrophizing is linked to worse mental health [23]. In sum, understanding individual differences in the relationship between pain and affect is important as it may shed light on how to improve well-being in individuals experiencing chronic pain.

The Present Study

The purpose of this study was to examine whether individual differences in the magnitude of affect reactivity to pain (concurrent and/or lagged associations) are associated with depression and anxiety levels in patients with chronic pain. We focused on momentary reports of pain intensity and affect measured with ecological momentary assessment (EMA) to obtain a fine-grained picture of the pain-affect dynamics within individuals as existing evidence suggests that daily diary studies are still prone to recall bias [24]. We also examined both PA and NA reactivity to pain, given prior evidence of different processes underlying the regulation of PA and NA [12,25]. To enhance the generalizability of results, the present research analyzes three independent pre-existing EMA studies and combines results across the different studies using meta-analysis methods.

Methods

Participants

The present study is a secondary data analysis of three existing studies of patients with chronic pain. For Study 1, a total of 106 patients (85.8% female) with a chronic rheumatic disease diagnosis were recruited from two community rheumatology offices [26]. Participants' most prevalent diagnoses included osteoarthritis (48.6%), rheumatoid arthritis (28.6%), lupus (16.2%), and fibromyalgia (10.5%). For Study 2, 68 chronic pain sufferers (85.3% female) diagnosed with at least one of the four chronic pain conditions: fibromyalgia (66.2%), osteoarthritis (41.2%), rheumatoid arthritis (7.4%), and ankylosing spondylitis (7.4%) were recruited through advertisements and flyers [27]. Participants in Study 3 were 116 patients with chronic pain conditions (84.5% female) recruited from a community rheumatology practice [28]. They had a physician confirmed diagnosis of at least one of three conditions: osteoarthritis (84.5%), fibromyalgia (36.2%), and rheumatoid arthritis (20.7%). Demographic characteristics of participants in the three studies are shown in Table 1. More details regarding recruitment and procedures can be found in the original studies. The University of Southern California Institutional Review Board approved the secondary analysis project.

Procedure

In all three studies, participants completed baseline surveys at an initial visit at the research offices. Eligible participants were trained to use an electronic diary (ED) administered on a hand-held computer. In Study 1, participants were randomly prompted (within intervals) to complete EMA assessments on the ED on average seven times a day across their waking hours for about 29–31 days. For Study 2, participants were randomized into one of four groups that differed on the number of random EMA prompts that they received per day (0 – control, 3, 6, or 12 prompts). Participants who were in the 3, 6, and 12-prompts groups were asked to complete momentary assessments on the ED for two weeks. For Study 3, participants were asked to participate in a 1-week EMA assessment of pain and affect on an ED that was programmed to deliver 8 to 9 prompts a day for a week.

Measures

Momentary measures

Pain intensity: In Study 1, participants were asked to rate “Before prompt: How intense was your bodily pain?” Participants rated their pain intensity on a visual analogue scale (VAS) from 0-*not at all* to 100-*extremely*. In Study 2 and Study 3, participants were first asked “Before prompt, were you in any pain?” and if yes “how much pain did you feel?” Participants who reported that they were in pain on the first question responded to a pain intensity VAS from 0-*no pain* to 100-*extreme pain*. The two questions were combined into a pain intensity VAS rating such that pain intensity in moments with no pain was set equal to 0 on the VAS scale.

Negative affect: In Study 1, participants were asked how “frustrated” they felt before the prompt. Participants rated their frustration from 0-*not at all* to 100-*extremely*. In Study 2 and Study 3, participants rated their current NA for each of four NA items from 0-*not at all* to 100-*extremely*. For Study 2, the four NA items were “depressed/blue”, “angry/hostile”, “frustrated”, and “worried/anxious” and for Study 3, they were “depressed”, “angry”, “frustrated” and “worried”. Scores for the four items were averaged at each moment so that a higher score indicates higher NA. The within-person reliability (McDonald’s ω) was calculated using multilevel confirmatory factor analysis (see [29] for details). The reliability of NA was acceptable (ω s = .74 in Study 2 and Study 3).

Positive affect: In Study 1, participants were asked how “happy” they felt before the prompt. Participants rated their happiness from 0-*not at all* to 100-*extremely*. In Study 2 and Study 3, participants rated their current PA for each of three PA items from 0-*not at all* to 100-*extremely*. For Study 2, the PA items were “happy”, “enjoying/having fun”, and “pleased” and for Study 3, they were “happy”, “enjoying yourself”, and “pleased”. Scores for the three items were averaged at each moment so that a higher score indicates higher PA. The within-person reliability of PA was good (ω = .87 for Study 2 and ω = .86 for Study 3).

Mental Health Measures

Depression: For all three studies, depression was measured at the baseline visit using the 21-item Beck Depression Inventory-II (BDI-II; [30]). The BDI-II has demonstrated high internal consistency [31]. Participants rated each of the depressive symptoms on a 3-point scale from 0 to 3; item scores were summed so that a higher score indicates higher levels of depressive symptoms. Cronbach’s alphas were .94, .92, and .88 for Study 1 to Study 3, respectively.

Anxiety: For all three studies, trait anxiety was measured using the State-trait Anxiety Inventory (STAI; [32]) at baseline. However, due to the existing concern regarding potential conceptual overlap with depression, the 7-item STAI-A subscale focusing on anxiety was used [33]. Participants were asked how they felt generally by rating 7 items from 1-*almost never* to 4-*almost always*. Item scores were summed to a total score so that a higher score indicates higher levels of anxiety. Cronbach’s alphas were .85, .85, and .79 for Study 1 to Study 3, respectively.

Analysis Plan

The analyses were based on two different ways of operationalizing the strength of a patient's affective responses to pain. In the first set of analyses, we characterized participants based on how strongly their momentary pain and affect were interconnected at any given point in time, that is, the concurrent association between momentary pain and affect (PA or NA) levels. In the second set of analyses, we characterized patients based on the extent to which their affect changes in response to previous pain experiences, that is, a person's *lagged* affective responses to momentary pain. Whereas the first analyses capture the simultaneous coupling of pain and affect in a given person, the second analyses explicitly consider the temporal ordering of experiences to capture a person's affect reactivity to pain at a specific time lag.

Analyses were conducted using multilevel time-series modeling techniques (i.e., dynamic structural equation modeling – DSEM; [34]) as implemented in *Mplus* version 8.3 [35] software. We briefly summarize the models used to estimate individual differences in concurrent and lagged affective responses to pain below; annotated *Mplus* code is provided in the online supplementary materials (for additional details and tutorial, see also [36]). All models were estimated separately for each study, and results were then meta-analytically summarized across studies.

Individual differences in the concurrent association between momentary pain and affect—The estimated DSEM model for concurrent associations between pain and affect (e.g., NA) is shown in Figure 1a. In this model, affect (PA or NA) was regressed on pain assessed at the same time point at the within-person level. Additionally, pain and affect were regressed on study time (coded in units of days) to remove linear time trends that would violate assumptions of stationarity [37], and the residuals of pain and affect were allowed to be autocorrelated across time-points [38]. The filled circles on the within-person level in Figure 1a indicate parameters that were allowed to vary between individuals (as random effects on the between-person level): in addition to the focal concurrent regression parameter, individuals were allowed to vary in the intercepts (i.e., mean levels at Day 0), (residual) variances (i.e., the magnitude of variability), time-trends, and residual autocorrelations (i.e., the “stability” or “inertia”) of both pain and affect. To test the hypothesis that a stronger concurrent association between pain and affect levels would be associated with greater mental health problems, we estimated the correlation between the random effect of the concurrent regression of affect on pain and the observed depression/anxiety scores on the between-person level. This correlation was part of a covariance matrix in which all random effects were allowed to correlate with each other and with the observed depression/anxiety scores.

Individual differences in the lagged affect reactivity to momentary pain—In order to appropriately estimate an individual's lagged affective responses to momentary pain, our analyses needed to consider that EMA measurement occasions were randomly sampled throughout a day. As is the case in most EMA studies, this resulted in unequal time intervals between measurement occasions. Ignoring this issue may yield biased estimates and may also falsely assume that lagged effects are unchanging across different time

intervals [39]. Moreover, there have been calls for using more meaningful time lags (i.e., time lags that capture the natural change process [40]). As a preliminary step and before conducting the DSEM lagged analyses, we used time-varying effect modeling (TVEM) to explicitly examine how affect reactivity to pain naturally unfolded as a function of time elapsed between observations in order to determine a time lag that was sensitive to capturing affective response to pain for most individuals. While exploratory, this first analysis step was important because we had no prior knowledge of the timescale over which affect reactivity to pain takes place.

Briefly, TVEM is an extension of multiple regression and is used to examine how regression coefficients (intercept and slopes) vary as a function of continuous time [41]. As would be done for conventional lagged effects regression analyses, we first created variables for pain intensity and affect (PA and NA) that were lagged for one measurement occasion. Both pain intensity and affect ratings, including lagged variables, were person-centered (to avoid a conflation of within-person and between-person effects and to ensure that the models were based purely on within-person variation). The TVEM analyses were conducted in SAS using the TVEM macro [42]. The TVEM models estimated the lagged effect of pain on affect (controlling for the autoregressive effect of affect) as a continuous function of time lag (observations with lags longer than 5 hours were eliminated from these TVEM analyses). A sample equation for NA is:

$$NA_{ij} = \beta_0(t_{ij}) + \beta_1(t_{ij})NA_{ij-1} + \beta_2(t_{ij})Pain_{ij-1} + e_{ij}$$

where t is time elapsed since the previous measurement, i indicates person ($i = 1, 2, \dots, n$), and j indicates measurement occasions ($j = 1, 2, \dots, T_i$). The intercept function [$\beta_0(t_{ij})$] and regression coefficient functions for lagged NA [$\beta_1(t_{ij})$] and lagged Pain [$\beta_2(t_{ij})$] are assumed to follow a smooth curve over time. The corresponding model was also examined between pain intensity and PA. TVEM models were estimated using P-spline in which automatic knot selection was implemented to determine the complexity of the coefficient curves.

The TVEM models were used to observe the exact shape of changes in the lagged effects over time, but they do not (yet) allow for estimating individual differences in these effects. DSEM, was used for this purpose. An advantage of DSEM over conventional regression analyses is that it can estimate lagged effects for *any* time gap (observed or unobserved) specified by the researcher (see [34] for details). Thus, even though the momentary observations were unequally spaced within and across individuals and studies, we were able to estimate lagged effects for a common time gap informed by the TVEM models.

The estimated DSEM model for lagged effects of pain on affect (e.g., NA) is shown in Figure 1b. In this model, affect (PA or NA) and pain were regressed on themselves (autoregressive parameters) and on each other (cross-lagged parameters) over time at the within-person level. The residuals for pain and affect were allowed to covary (expressed as a factor capturing the common residual variance, see [36]). The remaining parameters correspond with those of the concurrent effects model. To test the hypothesis that a stronger affective response to pain would be associated with greater mental health problems, the random cross-lagged effect of pain on affect (capturing individual differences in the lagged

affective responses to pain) was correlated with the observed depression/anxiety scores at the between-person level. As for the concurrent effects model, this correlation was part of a covariance matrix in which all random effects were allowed to correlate with each other and with the observed depression/anxiety scores.

All DSEM models were estimated using Bayesian methods using *Mplus* default non-informative priors. The convergence criterion for the potential scale reduction factor ($\hat{R}$) was consistent with recent recommendation ($\hat{R} < 1.01$; see [43]) and a minimum of 10,000 iterations were requested.

Synthesizing effects across studies—Meta-analysis techniques were used to synthesize the results across all three studies using the *Metafor* R Package [44]. The effect size metric in this analysis was the correlation coefficient, r (i.e., the relationship between concurrent or lagged affective responses to pain and a mental health outcome), which was transformed to Fisher's z (this transformation was executed within *Mplus* to obtain the appropriate sample variance estimate for meta-analyses of multilevel coefficients), and back-transformed to r for presentation purposes. Statistical significance was tested using a two-tailed z -test using fixed-effects meta-analysis. Heterogeneity testing was conducted using the Q -statistic in each meta-analysis. When significant effect size heterogeneity was evident, indicated by a significant Q -statistic at $p < .05$, random-effect meta-analysis would be used.

Supplemental analyses examining incremental effects of affective responses to pain—Whereas the primary analyses addressed the question whether individual differences in the (concurrent or lagged) association between pain and affect were correlated with mental health outcomes, it was also of interest whether these individual differences uniquely predicted mental health outcomes after controlling for other within-person dynamics. Thus, in secondary DSEM analyses, rather than estimating a covariance matrix, we regressed the observed anxiety/depression scores on all random effects parameters simultaneously at the between-person level. The resulting standardized regression weights were then meta-analyzed across studies to summarize the incremental effects of the (concurrent or lagged) pain-affect association on the mental health outcomes. In the supplementary materials, we provide samples of annotated codes for both DSEM and TVEM analyses.

Results

Descriptive Statistics

Across studies, the mean EMA pain intensity levels were 42.02 ($SD = 26.65$; Study 1), 44.25 ($SD = 30.20$; Study 2), and 37.50 ($SD = 28.50$; Study 3). The mean NA in each study was 27.76 ($SD = 26.43$; Study 1), 25.30 ($SD = 22.82$; Study 2), and 18.50 ($SD = 19.32$; Study 3) and the mean PA was 58.77 ($SD = 21.23$; Study 1), 51.31 ($SD = 21.16$; Study 2), and 58.55 ($SD = 20.97$; Study 3) on a 0–100 scale.

Because (weak) stationarity is an important assumption of DSEM and other time-series models, we tested the assumption of stationarity using the Kwiatkowski-Phillips-Schmidt-Shin (KPSS) test. The KPSS test evaluates whether a time series is stationary around a level

and a trend, and it was applied separately to each respondent and variable (pain, PA, and NA) in all datasets. The test was not significant for the large majority of participants (91.4% for pain, 93.4% for NA, 91.4% for PA), suggesting that the data were sufficiently stationary.

Concurrent Pain-Affect Association and Mental Health

In the first step, concurrent associations between momentary pain and affect was derived by regressing affect on concurrent pain. The standardized mean within-person associations between pain and NA were .21 (posterior SD [SD_{post}] = .01), .24 (SD_{post} = .01), and .19 (SD_{post} = .01), for Study 1 to Study 3, respectively. The mean within-person associations between pain and PA were $-.16$ (SD_{post} = .01), $-.21$ (SD_{post} = .02), and $-.15$ (SD_{post} = .01), for Study 1 to Study 3, respectively (see supplementary materials for sample histograms of individual differences in the within-person association). The within-person regression coefficients were correlated with individuals' depression and anxiety at baseline at the between-person level. Using fixed effect meta-analyses to synthesize the effect sizes across studies, the overall correlation between the pain-NA association and mental health was $r = .31$ ($p < .001$) for depression and $r = .26$ ($p < .001$) for anxiety (see Figure 2). The overall correlation between the pain-PA association and mental health was $r = -.23$ ($p = .002$) for depression and $r = -.19$ ($p = .01$) for anxiety. The Q-test for heterogeneity was not significant in any of the four meta-analyses.

Natural Time Course of Lagged Pain-to-Affect Association

In the second step, we examined how the effect of pain intensity on subsequent affect (PA and NA) varies as a function of time lag. Results indicated that, after controlling for lagged affects, momentary pain levels significantly predicted subsequent increases in NA as well as decreases in PA between 60 minutes and 180 minutes later (see Figure 3a and 3b). Based on these results, we chose a time lag of 90 minutes for subsequent DSEM analyses as the smallest reasonable time lag for which an affective response to pain (if present) would likely be observable for most individuals.

Lagged Pain-to-Affect Association and Mental Health

In the third step, we conducted DSEM models to obtain individual differences in the lagged pain-to-affect association, controlling for lagged affect-to-pain associations as well as the autoregressive associations of pain and affect. The standardized mean within-person lagged pain-to-NA associations were .08 (SD_{post} = .01), .04 (SD_{post} = .02), and .06 (SD_{post} = .02), for Study 1 to Study 3, respectively. The mean within-person lagged pain-to-PA associations were $-.05$ (SD_{post} = .01), $-.01$ (SD_{post} = .03), and $-.05$ (SD_{post} = .02), for Study 1 to Study 3, respectively (see supplementary materials for sample histograms of individual differences in the lagged associations). Results from fixed-effects meta-analyses (see Figure 4) showed that the overall correlation between NA reactivity to pain and mental health was not significant; $r = .14$ ($p = .17$) for depression and $r = .13$ ($p = .25$) for anxiety. The overall correlation between PA reactivity to pain and mental health was also not significant; $r = -.11$ ($p = .26$) for depression and $r = -.20$ ($p = .06$) for anxiety. The Q-test for heterogeneity was not significant in any of the four meta-analyses.

Supplemental analyses examining incremental effects of pain-affect association

Finally, we examined whether individual differences in pain-affect association significantly predicted mental health outcomes after controlling for other within-person dynamics [correlations among all between-person variables (mental health symptoms, means of pain and affect, and all random effects) are shown in the [supplementary materials]]. Results from meta-analyses of the three studies showed that the concurrent pain-NA association did not significantly predict depressive symptoms ($\beta = .02$, $p = .86$) or anxiety symptoms ($\beta = -.00$, $p = .98$) after controlling for individual differences in means, variability, residual autocorrelation as well as time trends of pain and NA. Similarly, concurrent pain-PA association did not significantly predict depressive symptoms ($\beta = -.14$, $p = .13$) or anxiety symptoms ($\beta = -.15$, $p = .12$). For the lagged pain-NA association, it did not significantly predict depressive symptoms ($\beta = -.04$, $p = .99$) or anxiety symptoms ($\beta = -.29$, $p = .95$) after controlling for individual differences in means, variability and residual covariance, autocorrelation, time trends of pain and NA as well as cross-lagged effect of NA on pain. Similarly, the lagged pain-PA association did not significantly predict depressive symptoms ($\beta = .12$, $p = .99$) and anxiety symptoms ($\beta = -.75$, $p = .97$).

Discussion

Pain and affect are often closely associated. However, individuals differ in the degree to which they are related. Our findings suggest that individuals with a stronger concurrent coupling between pain and affect (both PA and NA) are more likely to experience higher levels of depressive and anxiety symptoms. We did not find significant evidence that individuals with higher affect reactivity to pain, as indicated by a greater increase in NA and a greater decrease in PA in response to pain, are more likely to experience more depressive and anxiety symptoms.

Our findings pertaining to individual differences in the concurrent associations between pain and affect may be capturing one or more possible processes. First, the concurrent association between pain and affect could reflect affect reactivity to pain that occurs over a very brief time interval. Although pain and affect were measured at the same time, individuals' affect ratings may have taken into account the pain that they were experiencing at that moment. Second, the affect individuals experience concurrently with pain may partially reflect the affective dimension of pain [45]. In other words, individuals may differ in the degree to which they experience pain as aversive. Third, a stronger concurrent coupling between pain and affect may reflect individuals' inability to differentiate among negative feelings or sensations (e.g., an inability to discriminate between bodily pain and feeling states). In fact, research suggests that individuals who are less able to differentiate among negative events or emotions tend to have poorer mental health [46]. Our results from supplemental analyses suggest that individual differences in concurrent association between pain and affect was no longer a significant predictor of mental health symptoms after controlling for individual differences in means, residual variances, residual autocorrelation, and time trends of pain and affect. A possible explanation is that individual differences in pain-affect association are also related to individual differences in these other processes. For example, individuals who are more affectively reactive to pain are likely to be more reactive to other stress in life

(represented by a higher residual variance of NA and perhaps a higher mean of NA, see correlation tables in the supplementary materials).

Our results indicate that there is insufficient evidence that individual differences in lagged effect of pain on affect at a 90-minute time lag are associated with mental health symptoms. One possible explanation is that individuals do not only differ in the degree of affect reactivity to pain, but also the timing in which such responses occur. Although the 90-minute time gap indicated a time period that affect reactivity to pain was present most strongly for the average person in this study, some individuals were likely to have peak reactivity earlier and some later. Therefore, other dimensions that may be important to examine are individual differences in when their peak reactivity occurs and how quickly their affect returns to baseline. These could be important future directions.

Findings from the current study suggest potential new avenues for pain management. For example, interventions that target the decoupling between pain and affect may be beneficial for patients with chronic pain, especially for those with a high coupling between momentary pain and affect. When pain is inevitable and chronic, consistently experiencing NA in response to pain is not likely to be adaptive. In fact, pain acceptance has emerged as a promising strategy for pain management [47]. Research has shown that for individuals with higher pain acceptance, the magnitude of the association between NA and pain severity is potentially smaller [48]. Recent evidence also shows that interventions targeting pain acceptance are effective in reducing pain, depression, anxiety, pain interference, and in increasing quality of life [49]. Even though our findings are based on associations, and do not allow for causal inference, they may reflect a potential mechanism (i.e., the decoupling of pain and affect) through which pain acceptance improves individuals' mental health. Given that affect and pain have been found to be reciprocally related, such that momentary pain can increase NA and vice versa [50]; interrupting a negative cascade of experiences that can result from elevated pain states may be an important component for pain management.

Our study is one of the few existing studies that use TVEM to examine how within-person processes, such as affect reactivity to pain, vary as a function of time lag (e.g., in minutes) [51]. This method could potentially be applied to many other within-person processes to examine the timescale in which certain processes naturally unfold over time rather than relying on an arbitrary time lag (e.g., every 5 hours) or ignoring the effect of unequal spacing on processes in EMA studies. In this study, individuals' affect reactivity to pain on average seemed to dissipate after three hours. This suggests that it is important to collect consecutive measurements within this time interval if the goal is to understand affect reactivity to pain. Nevertheless, this time lag may not generalize to other types of studies or experiences, which underscores the need to understand the timescale in which specific within-person processes unfold over time in each study.

There are several limitations in the present study and directions for future research. First, participants in our studies were mostly chronic pain patients suffering from musculoskeletal pain. Our findings may not generalize to individuals without chronic pain or individuals with other types of chronic pain (e.g., headache). Future studies should examine whether individual differences in affect reactivity to pain would have similar implications for mental

health in individuals with non-chronic pain or other chronic pain conditions. Second, participants in our studies were mostly female (as would be expected given gender differences in the prevalence of these conditions) and White. Therefore, our findings may not generalize to individuals with different demographic characteristics. Third, the measurement of pain and affect could be even more intensive throughout the day for a more careful examination of time-varying lagged effects. Future EMA studies could have more measurement occasions per day to observe how the process of affect reactivity to pain naturally unfolds over various time lags. Finally, the studies included varied in the number of affect items and in the wording of the EMA questions (study 1 asked for affect experience ‘before prompt’, whereas studies 2 and 3 asked for ‘current affect’). Additional research is needed to better understand whether such variations in time frames specified in EMA questions impact the empirical dynamics being uncovered.

Conclusion

Individuals with chronic pain differ in the magnitude of coupling between pain and affect, which may have important implications for mental health. Our findings suggest that decoupling the association between pain and affect could be an important direction for future intervention research, which could potentially benefit patients with strong coupling between pain and affect to improve their mental health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Patients with chronic pain provided momentary pain and affect reports in 3 studies
- Individuals differ in the magnitude of momentary pain-affect coupling
- Stronger pain-affect coupling is related to more depressive and anxiety symptoms

Figure 1.

a (upper panel). Conceptual model in examining individual differences in concurrent pain-NA association. The variable of main interest was COR. ε = residual. **Figure 1b** (lower panel). Conceptual model in examining individual differences in lagged pain-to-NA association. The variable of main interest was NAonP. ζ = residual; Ψ = residual covariance. Variables on the between level are random effects (in circles) or observed variables (in rectangles): COR = concurrent regression of NA on pain; Pain = intercept of pain; NA = intercept of NA; varP = residual variance of pain; varNA = residual variance of NA; ARP =

(residual) autoregressive parameter for pain; ARNA = (residual) autoregressive parameter for NA; trendP = linear time trend of pain; trendNA = linear time trend of NA; Anx = (observed) mental health variable (depression, anxiety); NAonP = cross-lagged regression of NA on pain; PonNA = cross-lagged regression of pain on NA; psi = residual covariance of pain and NA.

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Figure 2.

Meta-analysis of correlations between individual differences in concurrent pain-affect association and mental health outcomes.

Figure 3.

a (upper panel). The magnitude of NA reactivity to pain as a function of time gap between two consecutive measurements. Shaded region represents 95% confident intervals around the estimated coefficient function for the average across the three studies. **Figure 3b** (lower panel). The magnitude of PA reactivity to pain as a function of time gap between two consecutive measurements. Shaded region represents 95% confident intervals around the estimated coefficient function for the average across the three studies.

Figure 4.

Meta-analysis of correlations between individual differences in the pain-to-affect lagged association and mental health outcomes.

Table 1.

Demographic characteristics of participants in the three primary studies.

	Broderick et al. 2008 (Study 1)	Stone et al. 2003 (Study 2)	Stone et al. 2010 (Study 3)
Sample size	106	68	116
Average number of EMA days	30.7 (1.8)	15.4 (0.6)	7.5 (0.6)
EMA compliance rate, mean (SD)	87% (13%)	91% (8%)	87% (9%)
Age, mean (SD)	55.3 (10.6)	50.9 (10.5)	57.4 (13.1)
Female (%)	85.8%	85.3%	84.5%
Race (% white)	91.5%	94.1%	95.7%
Education (% with a college degree)	42.9%	33.8%	45.6%
Median Family Income	\$50,000 – \$74,999	\$50,000 – \$74,999	\$50,000 – \$74,999