HHS Public Access

Author manuscript

Ann Behav Med. Author manuscript; available in PMC 2017 February 01.

Published in final edited form as:

Ann Behav Med. 2016 February ; 50(1): 12–23. doi:10.1007/s12160-015-9723-2.

Depressive symptoms and momentary mood predict momentary pain among rheumatoid arthritis patients

Jennifer E. Graham-Engeland[1], **Mathew J. Zawadzki**[2], **Danica C. Slavish**[1], and **Joshua M. Smyth**[1]

[1]The Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA 16802

[2]Psychological Sciences, University of California, Merced, Merced, CA 95343

Abstract

Background—Although a relationship between mood and pain has been established cross-sectionally, little research has examined this relationship using momentary within-person data.

Purpose—We examined whether baseline depressive symptoms and within-person levels of negative and positive mood predicted momentary pain among 31 individuals with rheumatoid arthritis (RA).

Methods—Depressive symptomatology was measured at baseline. Mood and RA symptoms were self-reported via ecological momentary assessment five times a day for seven consecutive days. Analyses controlled for gender, age, weekend day, time of day, and experiences of stress.

Results—Greater momentary positive mood was associated with less momentary pain and fewer arthritis-related restrictions; negative mood was associated with more restrictions. Greater depressive symptomatology also predicted more pain and restrictions, an effect which was not accounted for by mood.

Conclusions—Results suggest that both depression and mood are uniquely associated with momentary pain; as such, multi-component interventions may provide optimal disease management.

Keywords

pain; negative mood; positive mood; depressed mood; ecological momentary assessment; affect

It has become relatively common to hear people in everyday life acknowledging that their mood can exacerbate their physical pain, a sentiment frequently echoed among clinicians and researchers. Most evidence for this view, however, is derived from cross-sectional comparisons or longitudinal associations over fairly lengthy periods of time (e.g., months or years) with a limited number of assessments across time. A relatively unexplored issue is the dynamic interplay of mood and pain in daily life, from moment to moment, within the same

Correspondence concerning this article should be sent to Dr. Joshua M. Smyth or to Dr. Jennifer E. Graham-Engeland, Department of Biobehavioral Health, 219 Biobehavioral Health Building, The Pennsylvania State University, University Park, PA 16802. Dr. Smyth can be contacted at jms1187@psu.edu, 814-863-8402 and Dr. Graham-Engeland at jeg32@psu.edu, 814-404-8932.

individuals. Although a link between mood and pain has been established by comparing those with chronic pain to those without chronic pain (1), particularly by examining the impact of trait-like mood tendencies and depression, less work has examined linkages between mood and pain in everyday life on a daily basis (and even less has examined correlations between naturally occurring mood and pain fluctuations from moment to moment within a day). Moreover, the relative contributions of depressive symptomatology and momentary negative affect on pain remain unclear, as does the influence of momentary positive mood. Obtaining a more detailed appreciation of how mood is connected to pain in real-time and in real-life contexts would provide better evidence of a link between mood and pain than has been heretofore available. We therefore examined these associations among individuals with rheumatoid arthritis (RA), a group for whom connections between mood and pain are common and problematic (2, 3). We had three goals: (1) to evaluate the degree to which depressive symptoms at baseline are associated with RA symptoms – within-person momentary pain and arthritis-related restrictions – as well as with momentary negative and positive mood; (2) to determine if fluctuations in within-person momentary positive and negative mood are associated with within-person momentary pain and restrictions, and (3) to determine if the associations between depressive symptoms and pain/restrictions are explained by within-person differences in mood.

For decades it has been known that there is a strong link between depressive symptoms and chronic pain (1). Those with chronic pain are more likely to be depressed not only as compared to individuals in the general population but also as compared to individuals with other chronic health conditions (1). This is certainly true of RA, symptoms of which can be resistant to treatment. A chronic and systemic disorder, RA is characterized by joint inflammation and overall malaise, typically resulting in stiffness, swelling, and pain in addition to functional limitations in daily life (4). Depression appears to be two to three times as common among individuals with RA as compared to the general population (3, 5), and a meta-analysis showed that elevated depression in RA compared to heathy controls was not attributable to demographic factors but rather was associated with amount of pain reported (5). Chronic pain can function as a stressor, and it appears to be a particularly strong contributor to depressive symptoms and negative affect when pain interferes with and erodes satisfaction with life (for reviews see 6, 7). The linkage between pain and depression, however, appears to be causal in both directions (8, 9). While it is relatively clear that pain can contribute to negative mood and depressive symptomatology, it is also important to consider the degree to which negative mood and depression may contribute to or exacerbate pain.

Although it has been known for some time that tendencies to experience negative mood (e.g., anger, anxiety) are associated with greater reports of pain among those with chronic pain conditions (10), only relatively recently has research begun to examine the impact of mood on pain across time within the same individuals and to include examination of positive mood. In a study collecting monthly reports of worry and pain symptomatology across 6 months, worrying reported in one month predicted next month's pain, self-reported disease activity, and a swollen-joint count among individuals with RA (11). Weekly reports of anxiety and depressive symptoms have been associated with weekly reports of greater pain, both using aggregated weekly scores among patients with RA (12) and via multi-level

modeling over multiple weeks among women with both RA and osteoarthritis (13). Weekly reports of greater negative and lower positive mood have also been associated with greater future weekly pain among women with osteoarthritis and fibromyalgia (14). Finally, there have been several prospective studies utilizing daily reports of pain and mood. For example, Connelly and colleagues found that regulation of both positive and negative mood across a 30 day period from one day to another predicted lower daily pain among individuals with RA (15). Daily reports (across multiple months) of negative and positive mood were also linked with same day and next day pain among African-Americans with sickle-cell disease (16). In a joint laboratory and evening diary study of individuals with knee osteoarthritis, on days when individuals had higher daily positive affect (compared to their mean across all days), daily pain severity was reduced (17). This growing literature linking recent mood with recent pain provides considerable reason to believe that daily mood can play an important role in the everyday experience of pain. Yet, despite these studies, no research to our knowledge has examined the degree to which *momentary* negative and positive mood predict pain within days (i.e., using multiple assessments each day) within individuals; this research aims to fill this gap.

Daily assessment methodologies such as those described above answer different questions than between-subject analyses. An even more nuanced examination of the dynamic interplay of mood and pain in daily life requires the use of within-person momentary analyses and is critically important for a number of reasons. Within-person analyses inherently help to control for extraneous variation between participants (including factors such as socioeconomic status and medication use) (18). Additionally, intensive data capture, such as ecological momentary assessment (EMA), provides assessments taken in multiple moments from everyday life within the same individual (typically both within and across multiple days), which enhances ecological validity and greatly reduces recall bias (18, 19). Using EMA, Sorbi and colleagues (19) showed that momentary psychological responses, including fear-avoidance responses, cognitive responses, and spousal responses (e.g., catastrophizing; reinforcing behaviors) explained 8.1% of the variance in day to day change in pain intensity (over and above variance predicted by time of day) within persons among individuals with broadly defined chronic pain disorders. To our knowledge, however, no investigation has focused on the degree to which momentary positive and negative mood predict pain in daily life.

Investigating momentary mood in the context of baseline depressed mood may also help clarify whether depressive symptomatology has an independent effect on pain, or whether the effect of depressive symptomatology is due to alterations in momentary positive and negative mood. Those who are depressed may have both greater negative and less positive mood (20), and, based on recent literature as well as theory linking affect and health, it seems likely that both momentary negative mood and positive mood help explain how depressive symptomatology relates to pain. Research supports a two-factor model by which negative and positive emotion can each differentially influence health (17, 21). Positive emotion has been viewed as part of (or a consequence of) an active form of coping with stress that may influence pain in multiple ways, such as by serving as a distraction (22) or by activating physiological changes, including the endogenous opioid system (23, 24). Negative

mood appears to influence pain in diverse ways as well, such as by increasing attention to pain, contributing to avoidance behaviors and maladaptive health behaviors, causing muscle tension, and by activating pain-related molecules, including inflammatory cytokines (3, 10, 22). Although the majority of research has been on negative affective states, trait positive affect has been associated with lower levels of pain among diverse samples of patients with chronic disease (for review see 25), and recent studies using daily assessment methodologies suggest that positive mood may be protective against the effects of negative mood and stress (7, 14, 17, 26). Importantly, Smith and Zautra (13) found that an effect of depressive symptoms on weekly pain was mediated by decreased weekly positive mood, while an effect of trait anxiety on weekly pain was partially mediated by increased weekly negative mood. It therefore seems plausible that, when assessed on a momentary basis, both decreases in positive mood and increases in negative mood help explain an association between depressive symptomatology and momentary pain.

The Present Research

We hypothesized that for individuals with RA (1) baseline depressive symptoms would be associated with greater within-person momentary pain (as well as with greater momentary negative mood and less positive mood). We further hypothesized that (2) momentary negative mood would be associated with greater within-person momentary pain, whereas momentary positive mood would be associated with less within-person momentary pain. Given that the experience of stress has also been related to pain and mood (e.g., 27, 28), we controlled for momentary stress experience in analyses to evaluate our expectation that within-person differences in negative and positive mood, rather than stress, account for the association between depressive symptoms and daily pain. We expected that both baseline depressive symptoms and mood would predict pain over and above any effects of stress experiences throughout the day. Finally, we hypothesized that (3) differences in momentary mood would mediate (account for) the relationship between depressive symptoms and momentary pain.

Method

Overview

Data were drawn from an intensive baseline measurement interval conducted as part of a larger IRB-approved intervention study that utilized ecological momentary assessment (EMA) to assess daily and momentary well-being, stress, mood, pain expression, social support, coping, and health behaviors in a sample of adult patients with physician-confirmed chronic asthma or RA. The larger study examined the relationship of an emotion-regulation intervention on the health status of asthmatic and arthritic symptoms; only the participants with RA (N = 31) were included in the present analyses.

Participants

The sample (N = 31) was 74.2% female (n = 23), and the mean age of participants was 50 ± 13.07 years. The sample identified as 87.1% (n = 27) White or Caucasian, 9.68% Black or African-American (n = 3), and 3.2% (n = 1) “other”. Fifty-three percent of the participants

were married (n = 16), 23.3% (*n* = 7) never married, and 23.3% (*n* = 6) were separated, divorced, or annulled. There was considerable variability in annual household income, although the sample was relatively disadvantaged overall: 13.33% of participants reported an annual household income of less than $20,000 a year, 33.34% an annual household income of between $20-40,000, and 53.33% an annual household income of $40,000 or greater.

Procedure

Recruitment, training, and baseline assessment—Participants were recruited for the larger study from the local community via flyers, television, and radio advertisements. Interested individuals called the research office, at which point they were screened for eligibility. Prospective participants of the larger study were excluded if they were not 18 years of age, did not have a clinically verified diagnosis of asthma or RA (with time since diagnosis at least two years), reported receiving emergency room treatment (other than minor injury) in the previous 3 months, reported current drug or alcohol abuse problems, received a diagnosis of a mental illness within the prior 3 months, had a medication or other treatment change within 3 months that might affect pain or mood, or were unable to complete the EMA portion of the study (e.g., due to poor eyesight or limitations in manual dexterity).

Those individuals who met these criteria were invited to attend a training session, which also included a baseline assessment and informed consent. At this session, participants were trained on how to use a palmtop computer (Pilot m105, Palm, Sunnyvale, California) with custom software (developed using Satellite Forms 6.0, Thacker Network Technologies Inc., Lacombe, Alberta) to collect the EMA data. Participants also filled out a series of baseline questionnaires to determine standard psychological, social, and disease-related factors to use as between-person variables.

EMA protocol—The ecological momentary assessment (EMA) phase started the day after the baseline assessment/training session. EMA reports were collected five times a day for seven consecutive days and were used to determine the within-person variables (e.g., mood, stressful occurrences, symptoms) described in detail below. Reports were collected using a stratified signal-contingent design with one signal beep randomly occurring within each of five equal intervals between 8:00am and 9:00pm (see 18). This research used the measures of momentary mood, stressful occurrences, and RA symptoms (disease-specific symptom severity, including pain and activity restrictions), which are described in detail below; additional information about other EMA (and baseline) measures obtained is available from previous reports (29, or from Dr. Smyth).

Materials

Between-person depressive symptoms—Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; 30). This scale measures depressed mood over the course of the past week and consists of 20 items answered on a scale of 0 (“rarely or none, less than 1 day”) to 3 (“most of the time, 5-7 days”), with total summed scores ranging from 0 to 60. Higher scores indicate a greater frequency of

depressed mood, and scores of 19 or greater are typically indicative of clinical levels of depression in samples with chronic pain (31). This scale has good reliability and test-retest reliability in a variety of populations, including among chronic pain patients (30-33), and with good reliability in the present sample (Cronbach's α =.80).

Mediator variables

Within-person positive and negative mood: Momentary mood was assessed with adjective-checklist measures that were adapted for EMA (see 29) to determine how much participants felt each of a number of emotions at the time of the prompt; for positive mood: happy, joyful, enjoyment, and pleased, and for negative mood: depressed, unhappy, angry, frustrated, and worried. All items were on a scale from 0 (*Not at All*) to 6 (*Extremely*) and were averaged to create a composite score (across participants $M = 2.56$, $SD = 0.66$ for positive mood and $M = 0.95$, $SD = 0.61$ for negative mood). To test for internal consistency, given the multilevel nature of the data, we follow recommended procedures in which we specified a three level model with items for the given mood scale nested within measurement occasions nested within individuals (34). We then calculated the proportion of latent to total variation for the level of interest, which in this case was the measurement occasion level. Values can be interpreted similar to Cronbach's alpha. The positive (.76) and negative (.80) mood subscales had acceptable internal consistency.

Within-person stress experience: A measure of momentary stress experience was assessed using one EMA item: Participants indicated whether or not a stressful event had occurred since the last prompt (0 for no, 1 for yes). Across all measurements, a stress experience occurred 21.3% of the time. This measure of stress experience has been successfully used in previous studies (29), and was used herein as a control variable in analyses.

Outcome variables

Within-person pain and pain-related restrictions: It is important to take a multivariate approach to assessing pain in RA by measuring self-reported pain as well as functional disability and joint assessments (35). In the present study we assessed pain and related evaluations, as well as pain-related restrictions. Three items were used to assess the severity of "stiffness", "pain", and "joint tenderness/swelling" that the participant was feeling at the time of the prompt on a scale from 0 (*Not at All*) to 6 (*Extremely*); the mean of these items was computed to create a within-person "total pain" scale (across participants $M = 2.40$, $SD = 1.37$). The total pain subscale had moderately acceptable internal consistency (.60). In addition, two items were used to assess how much the participant's arthritis had interfered with their daily routine and forced the participant to restrict their activities since the last prompt, also on a scale from 0 (*Not at All*) to 6 (*Extremely*); the mean of these items was computed to form a within-person "total restrictions" scale (across participants $M = 2.23$, $SD = 1.39$). The total restrictions subscale had moderately acceptable internal consistency (.61).

Covariates—Age and gender were based on self-report at the baseline time point. Two time-related factors were calculated from the EMA data: "Time of day" was coded into five three-hour blocks, ranging from 1 to 5, roughly coinciding with the window of time that each EMA prompt took place (i.e., higher values correspond to later times in the day when

the EMA was taken). “Weekend day” was a dichotomous variable based on the day of the week the participant responded to the EMA prompt (Saturday to Sunday coded as 1, other days as 0).

Data Analysis Techniques

Given that we had observations (Level 1) nested within participants (Level 2), data were analyzed using multi-level modeling via SAS v9.3 PROC MIXED. In general, multilevel approaches are recommended for analyzing EMA data (36). In all analyses we controlled for participant age (as a continuous variable) and gender (1 = Female; 2 = Male), time of day (ranging from 1 to 5), whether it was a weekend day or not (0 = Weekday, 1 = Weekend), and whether or not a stress experience had occurred since the last prompt (0 = No Stressor, 1 = Stressor). For hypothesis 1, we examined whether between-person depressive symptoms predicted average within-person mood and pain. As the assessment of global depressive symptoms was time invariant, the results from the multilevel model in this instance have similar interpretations as unstandardized betas from multivariate regression models (e.g., do those with greater levels of depression also have greater levels of pain across all measures). However, with pain being time-varying (i.e., having a new assessed value at each measurement occasion), the advantage of using multilevel models is that we are able to control for potential time-varying confounds, such as time of day, resulting in greater precision of the estimate between depressive symptoms and pain. Given that the timing of EMAs were not evenly spaced apart but occurred randomly within equally spaced time intervals, we *a priori* specified a spatial power covariance structure modeling time as a continuous count of elapsed minutes since the start of EMA data collection. Individuals were expected to vary on their mean levels of momentary reports (Level 1), thus, random intercepts were specified to account for individual differences in overall momentary pain levels. We used similar models throughout later analyses to test for mediation between depressive symptoms and pain via positive mood and negative mood (subsequent steps needed to test Hypotheses 2 and 3 are described in the results). In line with recommendations to improve interpretability (37), we person-mean centered positive and negative mood; thus analyses reveal the impact of experiencing more or less positive or negative mood on pain and restrictions for an individual in a particular moment relative to that person’s general levels of positive and negative affect across all measurement occasions.

To examine mediation we followed the suggested procedures outlined by Zhang, Zyphur, and Preacher (38) and Krull and MacKinnon (39). In testing for mediation, debate exists as to whether predictor variables should be grand-mean centered when level 2 variables (in this case, depressive symptoms) predict level 1 mediators (positive mood, negative mood) and level 1 outcomes (pain) (e.g., 38). Although centering may help with interpretation, centering also can over- or under-estimate mediator effects when the magnitude of within-person effect departs from the magnitude of between-person effect. Additionally, when there are many groups (in this case participants) and smaller group sizes (in this case measurements per person), both true for the present analyses, the within-person effect is less likely to bias mediation estimates because the between-person component is not under-emphasized in favor of the within-person component (38). As a result, we followed the

recommendations of Krull and MacKinnon (39) and did not grand-mean center any variables for our multilevel models.

Results

Depressive Symptoms as a Predictor of Mood and Pain

Hypothesis 1 examined if between-person depressive symptoms predicted average within-person pain using the modeling technique described above. As can be seen in Table 1, those with more depressive symptoms at baseline reported significantly higher momentary total pain and pain-related restrictions during the week-long EMA period, controlling for gender, age, time of day, weekend day, and whether a stress experience had occurred since the last prompt. We also tested the relationship between-person depressive symptoms and within-person mood using the same set of analyses as described above. For the multilevel models, as can be seen in Table 1, greater baseline depressive symptoms predicted lower within-person positive mood and higher within-person negative mood.

Within-Person Mood as Predictors of Pain

Hypothesis 2 examined whether within-person positive mood and, tested separately, within-person negative mood predicted within-person pain, controlling for gender, age, time of day, weekend day, and whether a stress experience had occurred. We used a similar set of multilevel models as described above, except that in one model positive mood was entered as a predictor (instead of depressive symptoms) and negative mood was entered in a separate model. As can be seen in Table 2, greater within-person positive mood was associated with less within-person pain and fewer within-person restrictions. For the negative mood models, greater within-person negative mood was associated with more within-person restrictions. Before controlling for stress experience, negative mood was associated with more within-person pain (b = .07, SE = .03, p = .010) but after controlling for stress experience there was only a non-significant trend for negative mood to be associated with more within-person pain.

Within-Person Mood as Mediators of the Depressive Symptoms and Pain Connection

Hypothesis 3 examined whether within-person positive mood and negative mood mediated the effect of depressive symptoms on within-person pain (the mood and pain variables tested contemporaneous associations). To test this hypothesis we ran multilevel models similar to those used to test Hypothesis 1, but added within-person positive mood and then, in a separate model, negative mood, as mediators of the association between depressive symptoms and within-person pain. We then compared whether the magnitude of the original effect of depressive symptoms on pain (as reported in Table 1) was reduced as a result of including within-person positive mood and negative mood. As can be seen in Table 3, the effect of depressive symptoms on within-person pain remained relatively unchanged with the addition of the within-person mood variables.

Exploratory and Supplemental Analyses

On a more exploratory basis, we examined whether within-person positive mood and negative mood mediated the effect of depressive symptoms on lagged within-person pain. In

particular we were interested in whether there was a lag for pain-related restrictions as the EMA questions ask whether the participant experienced any restrictions since the last prompt and thus covers the entire assessment time period between one EMA and the next (compared to the pain items that assess pain at the time of the prompt). Lagged variables within-person and within-day were created for the all pain and restriction variables (i.e., from one moment of reporting to the next, but not 'counting' reports that were non-contiguous due to missing data or those spanning days). We then ran the same set of multilevel models used to test Hypothesis 3, but with the lagged pain variables as outcomes. By testing the lagged variables, we can assess whether mood at one time point (e.g., Time 0) predicts pain at the subsequent sampling time point (e.g., Time 1). As can be seen in Table 4, the mood variables did not predict lagged pain or restrictions, controlling for baseline depressive symptoms and stressor occurrence.

We next examined whether the relationships between within-person mood and within-person pain differed as a function of those who met criteria for clinical depression compared to those who did not. Roughly one-third of participants met the clinical criteria for depression (11 out of 31) based on the CES-D cutoff of 19 (M = 19.35, SD = 1.49). Given the small sample, we were under-powered to test a "true" interaction effect of baseline depression levels by within-person mood. Thus, to explore potential differences, we re-ran the analyses used to test Hypothesis 2, but examined only those who met criteria for depression in one set of models, and then those who did not in other models (see Table 5). Across the 11 participants who met criteria for depression we had 340 observations, and across the 20 others we had 634 observations. For those who met criteria for depression, within-person positive mood predicted less within-person pain and restrictions and within-person negative mood predicted both more pain and restrictions. In contrast, for those who did not appear to be depressed, within-person positive mood only predicted within-person restrictions (and marginally predicted less within-person pain) and negative mood predicted neither pain nor restrictions. In addition, for those who did not meet criteria for depression a stress experience was related to more pain and restrictions whereas no such relationship held for those who met the criteria for depression.

Finally, we examined the correlation between positive and negative mood, allowing for observations to be nested within individuals. The resulting correlation suggested a moderate relationship at the momentary level, r = -.54, and the person-level, r = -.41. Although beyond the scope of this paper, we recognize that it may be of interest to the reader as to whether there were independent effects of positive or negative mood on pain and restrictions while controlling for the opposite mood. These analyses can be found in the electronic supplemental materials section as Supplemental Tables 1-4. Overall, the effects of depressive symptoms remained significant after partialing out effects of both positive and negative mood in one model, as did the effects of positive mood after controlling for the effect of negative mood; however, the effects of negative mood were largely mitigated after partialing out the effect of positive mood.

Discussion

The purpose of this research was to examine whether baseline depressive symptoms and within-person momentary negative and positive mood predict within-person momentary pain among individuals with RA in daily life. Although relationships between depression, mood, and pain are well-established among those with chronic pain, and are common and problematic among those with RA, we are unaware of research that has focused on assessments of mood and pain in the moment (e.g., as opposed to daily or weekly) to examine these relationships in daily life, in the same individuals across time. Using multi-level modeling, this research found evidence consistent with the common (but largely untested) contention that mood in the moment (via self-reported ratings of mood obtained five times a day across seven days) is associated with momentary pain and pain-related restrictions (via ratings of pain, swelling, stiffness, and arthritis-related restrictions to routines and activities, obtained five times a day across the same time period). As predicted, greater depressive symptoms at baseline uniquely predicted more momentary pain and restrictions, controlling for gender, age, time of day, weekend day, as well as momentary stress experience. Also as expected, and with the same covariates, greater momentary positive mood was associated with less momentary pain and fewer restrictions, whereas greater negative momentary mood was associated with more momentary restrictions. Greater negative mood was also preliminarily associated with more pain, but not statistically significantly so after controlling for momentary stress experiences.

Contrary to expectations, neither momentary negative nor positive mood accounted for the association between baseline depressive symptoms and pain or restrictions, either for contemporaneous associations (with mood and pain assessed at the same moment) or lagged associations (with mood at one moment predicting pain at the subsequent moment in a particular day). Using a different methodology with weekly ratings of pain, Smith and Zautra (13) found that an effect of depressive symptoms on pain in RA was explained by decreased positive mood. Our momentary sampling timeline may help explain why mood did not account for the relationship between depression and pain in the present research. Although mood and depressive symptomatology are associated, mood may capture a phenomenon that is more unique from depressive symptomatology in its effects when examined on a momentary basis as compared to ratings of mood measured on a weekly or even daily basis. In any given moment, situational factors (e.g., having been in a recent argument, being exposed to pretty scenery, drinking a cup of coffee) have a strong influence on momentary mood (e.g., 40); as such, the influence of situational context may outweigh, at least some of the time, the effects of depressive symptomatology on pain. In support of the idea that depressive symptoms and momentary mood are capturing different phenomena, the correlation across averaged momentary mood (across all EMA ratings for each person) and baseline depression in the present research was non-significant for positive mood ($r = -.27$, $p = .147$) and of a small to moderate size for negative mood ($r = .40$, $p = .027$). In addition, recall bias is more likely in instances where individuals are asked to report back on how they felt over longer intervals (e.g., the past week) than when asked to report current feelings (see 18). A depressed individual is particularly likely to look back over the course of the day and recall problems or negativity than someone who is not depressed (41, 42). In summary, the

impact of momentary forces and the relative lack of recall bias on momentary mood may result in depressive symptomatology and momentary mood having unique effects on momentary pain. The present research is thus in concordance with the perspective that it is critical to treat depressive symptomatology among those who have chronic pain (9, 43) and further suggests that additionally addressing momentary mood may be helpful.

Caution should be exercised when interpreting the null effects for the lagged results, which were suboptimal tests of lagged associations and which were therefore positioned as exploratory. In particular, the data were not collected at a high enough density to truly understand the decay curve of any effects (e.g., should effects last for 15 minutes, 30 minutes, two hours?). With measurements occurring an average of a few hours apart, our models assume a potentially lengthy effect of mood on pain and restrictions that may not be warranted. Further, successive measures were variable within and across participants, adding error to our models: Because participants completed each EMA at random times within five equal time intervals, successive measures could be anywhere from 30 minutes to hours apart. Future work would benefit by systematically testing potential carry-over effects of mood on pain and restrictions from one moment to the next, with enough density of assessments to test both the length and nature of any potential lagged associations.

On a more exploratory basis we examined whether the associations between mood and pain differed among those who met the cut-off for clinical depression. Among the 11 participants who appeared to be depressed based on this criterion, momentary negative mood was predictive of both greater pain as well as pain-related restrictions, and momentary positive mood was associated with less pain and pain-related restrictions. In contrast, among those who did not appear to be depressed, momentary negative mood was not predictive of either pain or restrictions, and momentary positive mood predicted only fewer restrictions and was not significantly associated with pain. Thus, it appears that mood is more likely to be linked with pain and pain-related restrictions among those who are depressed. Interestingly, exploratory analyses suggested that stress experience was also differently associated with pain based on depression status. Stress experience was strongly associated with both pain and restrictions among those who were not depressed, but was not related to either pain or restrictions among those who met the criteria for depression. Taken together, these results provide preliminary evidence that there may be different processes by which psychosocial and mood factors relate to increased pain in daily life between individuals who are depressed compared to those who are not. The pain among individuals with RA who are not depressed may be more influenced by stress experience than by negative mood, whereas the pain among depressed individuals with RA may be more likely to be influenced by negative mood than stress experience.

In combination, several of our analyses suggest that momentary positive mood is more robustly associated with momentary pain than negative mood. When examined on their own, prior to controlling for stress experience, both negative mood and positive mood predicted pain and pain-related restrictions. After controlling for stress experience, only positive mood remained a unique predictor of pain and restrictions. Further, positive mood was related to pain-related restrictions among those who were not depressed and to both pain and restrictions among those who were depressed; in contrast, negative mood was related to pain

and pain-related restrictions only among those who were depressed. This finding is in concordance with results from several studies of daily positive mood and pain (e.g., 16, 17, 44) and a perspective that positive mood is associated with resilience to pain in RA (14). Finan and colleagues (17) found what they interpreted as a more robust effect of daily positive mood than daily negative mood: On days when participants had higher daily positive affect (than their individual average across days), they had significantly lower daily pain severity, even after controlling for the effect of daily negative affect, whereas the effect of daily negative affect did not hold after controlling for daily positive affect. Further, in a daily diary study of fibromyalgia patients, pain and positive affect, but not negative affect, mediated the relationship between sleep quality and activity interference (44). Additional research will be needed to determine whether and under what circumstances positive mood has a more consistent and/or stronger effect on momentary pain.

Although our findings cannot speak to causality in either direction, this study is in concordance with the perspective that both momentary negative and positive mood may contribute to physical pain among individuals with RA in daily life. Several potential mechanisms to explain linkages between momentary mood and pain are suggested by past research. One route is via attention to painful stimuli. Negative mood has been shown to modulate pain experience by creating psychophysiological hypervigilance or arousal to stimuli (45); conversely, positive mood may serve as a distraction (22). Physiological changes in response to affective states are also a likely route by which mood relates to pain in daily life. Positive and negative mood states can activate dynamically integrated brain regions related to pain transmission (46-48). Negative mood states have also been associated with increased peripheral levels of pain-related molecules, including inflammatory cytokines (49, 50), and recent findings suggest that positive mood states may activate the endogenous opioid system (23, 24) and may also be related to oxytocin release, which has pain relieving effects (51). Other cognitive, behavioral, and physiological mechanisms are also likely and warrant additional investigation. Future work should examine these and other possible pathways. It will also be important to determine the existence of vicious cycles involving causal directionality from pain and mood in both directions. It is well-established that pain promotes negative mood (9, 10) and there is strong evidence that chronic pain contributes to depression (even more robust than evidence suggesting that depression can contribute to pain) (1). Given that both pain intensity and negative and positive mood can vary throughout the day and relate to each other in multiple ways, interventions that can be delivered “in the moment” may be particularly able to disrupt the cycle between momentary mood and pain (see 52).

There are several limitations of the present work. First, we had a relatively small sample, due in part to the intensive data collection process needed as part of this EMA study. Results may not be generalizable beyond the characteristics of our sample. Further, due to the gender differential in both RA prevalence and in our sample, we lacked a sufficient number of male participants to test gender effects. It is possible that momentary changes in mood would have accounted for the association between depressive symptoms and pain for certain groups of individuals or for a different sample. We used weekend day as a proxy for when participants were working versus not, with exploratory analyses suggesting this as an appropriate proxy (e.g., few participants reported being at their workplace on weekend

days). Yet, given the potential effect of the workday on mood and stress (53), future research should explore whether working status and other types of activities (e.g., going to clubs, religious services, exercising) moderate the relationships between depressive symptoms, mood, and pain. Finally, we did not have the density of data necessary to allow for confidence in lagged analyses well-suited to better establish causal directionality and, similarly, were not able to examine carefully the relative influence of pain on mood to differentiate directionality of effects.

In conclusion, we demonstrated that momentary levels of pain and arthritis-related restrictions among individuals with RA were predicted by momentary mood, with greater negative mood relating to greater RA-related restrictions (and to greater pain among the subset of participants who were depressed) and greater positive mood relating to less pain and fewer restrictions. Furthermore, depressive symptomatology at baseline was uniquely predictive of both momentary pain and restrictions as well, an association that was not explained by differences in momentary mood. Although questions remain about causality as well as directionality of effects, the present research suggests that interventions to target depression as well as interventions to target momentary mood (including positive mood states) warrant investigation for individuals with RA and, perhaps chronic pain in general. Multi-component interventions aimed at both mood and depression that incorporate non-traditional interventions as adjuvants to pharmaceutical therapies may be needed to optimally improve pain and pain-related quality of life in many individuals with RA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by a grant from the National Heart, Lung, and Blood Institute (R01-HL067990) to Smyth. Work on this manuscript was also supported in part by a National Science Foundation Graduate Research Fellowship Program (DGE1255832) to Slavish. Any opinions, findings, and conclusions or recommendations expressed in this material are those of the author(s) and do not necessarily reflect the views of the National Institutes of Health or the National Science Foundation.

References

1. Banks SM, Kerns RD. Explaining high rates of depression in chronic pain: A diathesis-stress framework. Psychol Bull. 1996; 119:95–110.
2. Keefe FJ, Somers TJ. Psychological approaches to understanding and treating arthritis pain. Nat Rev Rheumatol. 2010; 6:210–216. [PubMed: 20357790]
3. Huyser BA, Parker JC. Negative affect and pain in arthritis. Rheum Dis Clin North Am. 1999; 25:105–121. [PubMed: 10083961]
4. Khurana R, Berney SM. Clinical aspects of rheumatoid arthritis. Pathophysiology. 2005; 12:153–165. [PubMed: 16125918]
5. Dickens C, McGowan L, Clark-Carter D, Creed F. Depression in rheumatoid arthritis: A systematic review of the literature with meta-analysis. Psychosom Med. 2002; 64:52–60. [PubMed: 11818586]
6. Cannella DT, Lobel M, Glass P, Lokshina I, Graham JE. Factors associated with depressed mood in chronic pain patients: The role of personal coping resources. J Pain. 2007; 8:256–262. [PubMed: 17174608]

7. Zautra AJ, Smith B, Affleck G, Tennen H. Examinations of chronic pain and affect relationships: Applications of a dynamic model of affect. J Consult Clin Psychol. 2001; 69:786. [PubMed: 11680555]
8. Campbell LC, Clauw DJ, Keefe FJ. Persistent pain and depression: A biopsychosocial perspective. Biol Psychiatry. 2003; 54:399–409. [PubMed: 12893114]
9. Bair MJ, Robinson RL, Katon W, Kroenke K. Depression and pain comorbidity: A literature review. Arch Intern Med. 2003; 163:2433. [PubMed: 14609780]
10. Gaskin ME, Greene AF, Robinson ME, Geisser ME. Negative affect and the experience of chronic pain. J Psychosom Res. 1992; 36:707–713. [PubMed: 1432860]
11. Evers AW, Verhoeven EW, van Middendorp H, et al. Does stress affect the joints? Daily stressors, stress vulnerability, immune and HPA axis activity, and short-term disease and symptom fluctuations in rheumatoid arthritis. Ann Rheum Dis. 2014; 73:1683–1688. [PubMed: 23838082]
12. Strand EB, Kerns RD, Christie A, et al. Higher levels of pain readiness to change and more positive affect reduce pain reports: A weekly assessment study on arthritis patients. Pain. 2007; 127
13. Smith B, Zautra AJ. The effects of anxiety and depression on weekly pain in women with arthritis. Pain. 2008; 138:354–361. [PubMed: 18289792]
14. Zautra AJ, Johnson LM, Davis MC. Positive affect as a source of resilience for women in chronic pain. J Consult Clin Psychol. 2005; 73:212–220. [PubMed: 15796628]
15. Connelly M, Keefe FJ, Affleck G, et al. Effects of day-to-day affect regulation on the pain experience of patients with rheumatoid arthritis. Pain. 2007; 131:162–170. [PubMed: 17321049]
16. Gil KM, Carson JW, Porter LS, et al. Daily mood and stress predict pain, health care use, and work activity in African American adults with sickle-cell disease. Health Psychol. 2004; 23:267–274. [PubMed: 15099167]
17. Finan PH, Quartana PJ, Smith MT. Positive and negative affect dimensions in chronic knee osteoarthritis: Effects on clinical and laboratory pain. Psychosom Med. 2013; 75:463–470. [PubMed: 23697467]
18. Smyth, JM.; Heron, KE. Health Psychology. In: Mehl, MR.; Conner, TS., editors. Handbook of Research Methods for Studying Daily Life. New York: The Guilford Press; 2012. p. 569-584.
19. Sorbi MJ, Peters ML, Kruise DA, et al. Electronic momentary assessment in chronic pain I: psychological pain responses as predictors of pain intensity. Clin J Pain. 2006; 22:55–66. [PubMed: 16340594]
20. Clark LA, Watson D. Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. J Abnorm Psychol. 1991; 100:316–336. [PubMed: 1918611]
21. Smith B, Zautra AJ. Vulnerability and resilience in women with arthritis: Test of a two-factor model. J Consult Clin Psychol. 2008; 76:799–810. [PubMed: 18837597]
22. Hamilton NA, Zautra AJ, Reich JW. Affect and pain in rheumatoid arthritis: Do individual differences in affective regulation and affective intensity predict emotional recovery from pain? Ann Behav Med. 2005; 29:216–224. [PubMed: 15946116]
23. Benedetti F, Amanzio M. Mechanisms of the placebo response. Pulm Pharmacol Ther. 2013; 26:520–523. [PubMed: 23370213]
24. Koepp MJ, Hammers A, Lawrence AD, et al. Evidence for endogenous opioid release in the amygdala during positive emotion. NeuroImage. 2009; 44:252–256. [PubMed: 18809501]
25. Pressman SD, Cohen S. Does positive affect influence health? Psychol Bull. 2005; 131:925–971. [PubMed: 16351329]
26. Strand EB, Zautra AJ, Thoresen M, et al. Positive affect as a factor of resilience in the pain-negative affect relationship in patients with rheumatoid arthritis. J Psychosom Res. 2006; 60:477–484. [PubMed: 16650588]
27. Affleck G, Tennen H, Urrows S, Higgins P. Person and contextual features of daily stress reactivity: Individual differences in relations of undesirable daily events with mood disturbance and chronic pain intensity. J Pers Soc Psychol. 1994; 66:329–340. [PubMed: 8195989]
28. DeLongis A, Folkman S, Lazarus R. The impact of daily stress on health and mood: Psychological and social resources as mediators. J Pers Soc Psychol. 1988; 54:486–495. [PubMed: 3361420]

29. Smyth JM, Zawadzki MJ, Santuzzi AM, Filipkowski KB. Examining the effects of perceived social support on momentary mood and symptom reports in asthma and arthritis patients. Psychol Health. 2014; 29:813–831. [PubMed: 24568534]
30. Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Appl Psychol Meas. 1977; 1:385–401.
31. Turk DC, Okifuji A. Detecting depression in chronic pain patients: Adequacy of self-report. Behav Res Ther. 1994; 32:9–16. [PubMed: 8135727]
32. Geisser ME, Roth RS, Robinson ME. Assessing depression among persons with chronic pain using the Center for Epidemiological Studies Depression Scale and the Beck Depression Inventory: A comparative analysis. Clin J Pain. 1997; 13:163–170. [PubMed: 9186024]
33. Ciechanowski P, Sullivan M, Jensen M, Romano J, Summers H. The relationship of attachment style to depression, catastrophizing and health care utilization in patients with chronic pain. Pain. 2003; 104:627–637. [PubMed: 12927635]
34. Wilhelm P, Schoebi D. Assessing mood in daily life: Structural validity, sensitivity to change, and reliability of a short-scale to measure three basic dimensions of mood. Eur J Psych Assess. 2007; 23:258–267.
35. Pincus T, Strand V, Koch G, et al. An index of the three core data set patient questionnaire measures distinguishes efficacy of active treatment from that of placebo as effectively as the American College of Rheumatology 20% response criteria (ACR20) or the Disease Activity Score (DAS) in a rheumatoid arthritis clinical trial. Arthritis Rheum. 2003; 48:625–630. [PubMed: 12632413]
36. Schwartz JE, Stone AA. Strategies for analyzing ecological momentary assessment data. Health Psychol. 1998; 17:6–16. [PubMed: 9459065]
37. Singer, J.; Willett, J. Applied Longitudinal Data Analysis. New York: Oxford University Press; 2003.
38. Zhang Z, Zyphur MJ, Preacher KJ. Testing multilevel mediation using hierarchical linear models problems and solutions. Org Res Meth. 2009; 12:695–719.
39. Krull JL, MacKinnon DP. Multilevel modeling of individual and group level mediated effects. Multivar Behav Res. 2001; 36:249–277.
40. Lewis, M.; Haviland-Jones, JM.; Barrett, LF., editors. Handbook of Emotions. 3. New York, NY: Guilford Press; 2008.
41. Wenze SJ, Gunthert KC, German RE. Biases in affective forecasting and recall in individuals with depression and anxiety symptoms. Pers Soc Psychol Bull. 2012; 38:895–906. [PubMed: 22649114]
42. Bradley B, Mathews A. Negative self-schemata in clinical depression. Br J Clin Psychol. 1983; 22:173–181. [PubMed: 6626790]
43. Staiger TO, Gaster B, Sullivan MD, Deyo RA. Systematic review of antidepressants in the treatment of chronic low back pain. Spine. 2003; 28:2540–2545. [PubMed: 14624092]
44. Kothari DJ, Davis MC, Yeung EW, Tennen HA. Positive affect and pain: mediators of the within-day relation linking sleep quality to activity interference in fibromyalgia. Pain. 2015; 156:540–546. [PubMed: 25679472]
45. Janssen SA. Negative affect and sensitization to pain. Scand J Psychol. 2002; 43:131–137. [PubMed: 12004950]
46. Eisenberger NI. Meta-analytic evidence for the role of the anterior cingulate cortex in social pain. Soc Cogn Affect Neurosci. 2014
47. Shackman AJ, Salomons TV, Slagter HA, et al. The integration of negative affect, pain and cognitive control in the cingulate cortex. Nat Rev Neurosci. 2011; 12:154–167. [PubMed: 21331082]
48. Wager TD, Kang J, Johnson TD, et al. A bayesian model of category-specific emotional brain responses. PLoS Comput Biol. 2015; 11:e1004066. [PubMed: 25853490]
49. Carroll JE, Low CA, Prather AA, et al. Negative affective responses to a speech task predict changes in interleukin (IL)-6. Brain Behav Immun. 2011; 25:232–238. [PubMed: 20888901]
50. Dickerson SS, Kemeny ME, Aziz N, Kim KH, Fahey JL. Immunological effects of induced shame and guilt. Psychosom Med. 2004; 66:124–131. [PubMed: 14747646]

51. Goodin BR, Anderson AJ, Freeman EL, et al. Intranasal oxytocin administration is associated with enhanced endogenous pain inhibition and reduced negative mood states. Clin J Pain. 2014 Epub ahead of print.

52. Heron KE, Smyth JM. Ecological momentary interventions: Incorporating mobile technology into psychosocial and health behaviour treatments. Br J Health Psychol. 2010; 15:1–39. [PubMed: 19646331]

53. Damaske S, Smyth JM, Zawadzki MJ. Has work replaced home as a haven? Re-examining Arlie Hochschild's Time Bind proposition with objective stress data. Soc Sci Med. 2014; 115:130–138. [PubMed: 24869785]

Table 1

Unstandardized Estimates (Standard Errors) for Depressive Symptoms Predicting Within-Person Pain, Restrictions, and Positive and Negative Mood

	Within-Person Pain Variables		Within-Person Mood Variables	
	Total Pain	**Total Restrictions**	**Positive Mood**	**Negative Mood**
Intercept	.66 (1.65)	.55 (1.73)	4.16 (.86)***	-.43 (.72)
Age	.01 (.02)	.01 (.02)	-.02 (.01)+	.01 (.01)
Gender	-.21 (.54)	-.42 (.57)	-.004 (.28)	-.01 (.24)
Time of Day	-.09 (.02)***	-.06 (.02)**	.06 (.02)**	-.01 (.02)
Weekend Day	.08 (.05)	.21 (.06)***	.24 (.07)***	-.17 (.08)*
Stress Experience	.16 (.06)**	.29 (.07)***	-.81 (.08)***	.83 (.06)***
Depressive Symptoms	.09 (.04)*	.08 (.04)*	-.04 (.02)*	.04 (.02)**

Note.

+ $p < .10$;

* $p < .05$;

** $p < .01$;

*** $p < .001$. Age was a continuous variable. Gender (1 = Female; 2 = Male), Weekend Day (0 = Weekday; 1= Weekend), and Stress Experience (0 = No Stressor; 1 = Stress Occurrence) were dichotomous variables. Time of day was coded to approximate each EMA interval ranging from 1 to 5.

Table 2

Unstandardized Estimates (Standard Errors) for Within-Person Positive and Negative Mood Predicting Within-Person Pain and Restrictions

	Total Pain	Total Restrictions
Within-Person Positive Mood Model		
Intercept	3.78 (1.13)**	3.34 (1.15)**
Age	-.01 (.02)	-.01 (.02)
Gender	-.39 (.58)	-.58 (.59)
Time of Day	-.09 (.02)***	-.05 (.02)**
Weekend Day	.10 (.05)+	.23 (.06)***
Stress Experience	.10 (.06)	.20 (.07)**
Positive Mood	-.07 (.03)**	-.11 (.03)***
Within-Person Negative Mood Model		
Intercept	3.79 (1.12)**	3.36 (1.15)**
Age	-.01 (.02)	-.01 (.02)
Gender	-.39 (.58)	-.58 (.59)
Time of Day	-.09 (.02)***	-.06 (.02)**
Weekend Day	.09 (.05)+	.22 (.06)***
Stress Experience	.12 (.07)+	.23 (.08)**
Negative Mood	.05 (.03)	.07 (.03)*

Note.

+ $p < .10$;

* $p < .05$;

** $p < .01$;

*** $p < .001$. Age was a continuous variable. Gender (1 = Female; 2 = Male), Weekend Day (0 = Weekday; 1= Weekend), and Stress Experience (0 = No Stressor; 1 = Stress Occurrence) were dichotomous variables. Time of day was coded to approximate each EMA interval ranging from 1 to 5. Positive and negative mood were person-mean centered.

Table 3

Unstandardized Estimates (Standard Errors) for Within-Person Positive Mood and Negative Mood Mediating the Effect of Depressive Symptoms on Within-Person Pain and Restrictions

	Total Pain	Total Restrictions
Within-Person Positive Mood Model		
Intercept	.67 (1.65)	.56 (1.73)
Age	.01 (.02)	.01 (.02)
Gender	-.21 (.54)	-.42 (.57)
Time of Day	-.09 (.02)***	-.05 (.02)**
Weekend Day	.10 (.05)+	.23 (.06)***
Stress Experience	.10 (.06)	.20 (.07)**
Positive Mood	-.07 (.03)**	-.11 (.03)***
Depressive Symptoms	.09 (.04)*	.08 (.04)*
Within-Person Negative Mood Model		
Intercept	.68 (1.65)	.57 (1.73)
Age	.01 (.02)	.01 (.02)
Gender	-.21 (.54)	-.42 (.57)
Time of Day	-.09 (.02)*	-.06 (.02)**
Weekend Day	.09 (.05)+	.22 (.06)***
Stress Experience	.12 (.07)+	.23 (.08)**
Negative Mood	.05 (.03)	.07 (.03)*
Depressive Symptoms	.09 (.04)*	.08 (.04)*

Note.

+ $p < .10$;

* $p < .05$;

** $p < .01$;

*** $p < .001$. Age was a continuous variable. Gender (1 = Female; 2 = Male), Weekend Day (0 = Weekday; 1= Weekend), and Stress Experience (0 = No Stressor; 1 = Stress Occurrence) were dichotomous variables. Time of day was coded to approximate each EMA interval ranging from 1 to 5. Positive and negative mood were person-mean centered.

Table 4

Unstandardized Estimates (Standard Errors) for Within-Person Positive and Negative Mood Mediating the Effect of Depressive Symptoms on Lagged Within-Person Pain and Restrictions

	Lagged Total Pain			Lagged Total Restrictions		
	Depression Only	**Mood Only**	**Depression & Mood**	**Depression Only**	**Mood**	**Depression & Mood**
			Within-Person Positive Mood Model			
Intercept	.50 (1.15)	2.84 (.81)**	.49 (1.15)	.45 (1.06)	2.41 (.74)**	.45 (1.07)
Age	.01 (.01)	-.01 (.01)	.01 (.01)	.01 (.01)	-.01 (.01)	.01 (.01)
Gender	-.12 (.38)	-.25 (.41)	-.12 (.38)	-.24 (.35)	-.36 (.38)	-.24 (.35)
Time of Day	-.10 (.02)***	-.10 (.02)***	-.10 (.02)***	-.08 (.02)**	-.08 (.02)**	-.08 (.02)**
Weekend Day	.11 (.06)+	.11 (.06)+	.11 (.06)+	.14 (.07)*	.15 (.07)*	.14 (.07)*
Pain/Restrictions#	.31 (.04)***	.31 (.04)***	.31 (.04)***	.37 (.03)***	.37 (.04)***	.37 (.04)***
Stress Experience	-.03 (.07)	-.02 (.07)	-.02 (.07)	-.13 (.08)+	-.15 (.08)+	-.15 (.08)+
Positive Mood	--	.02 (.03)	.02 (.03)	--	-.02 (.03)	-.02 (.03)
Depressed Symptoms	.07 (.03)**	--	.07 (.03)**	.06 (.03)*	--	.06 (.03)*
			Within-Person Negative Mood Model			
Intercept	.50 (1.15)	2.52 (.86)**	.48 (1.15)	.45 (1.06)	2.38 (.73)**	.43 (1.06)
Age	.01 (.01)	-.01 (.01)	.01 (.01)	.01 (.01)	-.01 (.01)	.01 (.01)
Gender	-.12 (.38)	-.19 (.44)	-.12 (.38)	-.24 (.35)	-.35 (.37)	-.24 (.35)
Time of Day	-.10 (.02)***	-.10 (.03)***	-.10 (.02)***	-.08 (.02)**	-.08 (.02)**	-.08 (.02)**
Weekend Day	.11 (.06)+	.20 (.07)**	.11 (.06)+	.14 (.07)*	.13 (.07)*	.13 (.07)*
Pain/Restrictions#	.31 (.04)***	.39 (.04)***	.31 (.04)***	.37 (.03)***	.38 (.04)***	.38 (.04)***
Stress Experience	-.03 (.07)	-.05 (.09)	.004 (.08)	-.13 (.08)+	-.10 (.09)	-.10 (.09)
Negative Mood	--	-.06 (.04)	-.04 (.04)	--	-.03 (.04)	-.03 (.04)
Depressed Symptoms	.07 (.03)***	--	.07 (.03)**	.06 (.03)*	--	.06 (.03)*

Note.

+ $p < .10$;

* $p < .05$;

** $p < .01$;

*** $p < .001$.

#When Lagged Total Pain ("Time 1") was examined, Total Pain at the previous assessment ("Time 0") was controlled; likewise when Lagged Total Restrictions was examined, Total Restrictions at the previous assessment was controlled. Age was a continuous variable. Gender (1 = Female; 2 = Male), Weekend Day (0 = Weekday; 1= Weekend), and Stress Experience (0 = No Stressor; 1 = Stress Occurrence) were dichotomous variables. Time of day was coded to indicate the EMA interval ranging from 1 to 5. Positive and negative mood were person-mean centered.

Table 5

Unstandardized Estimates (Standard Errors) for Within-Person Positive and Negative Mood Predicting Within-Person Pain and Restrictions by Clinical Depression

	Among Those Not Clinically Depressed		Among Those Clinically Depressed	
	Total Pain	**Total Restrictions**	**Total Pain**	**Total Restrictions**
	Within-Person Positive Mood Model			
Intercept	2.17 (1.78)	2.30 (1.86)	4.06 (1.16)**	3.49 (1.33)*
Age	.004 (.03)	.01 (.03)	.02 (.03)	.01 (.04)
Gender	-.17 (.78)	-.67 (.82)	-1.44 (.83)	-.72 (.96)
Time of Day	-.06 (.02)**	-.02 (.02)	-.11 (.03)***	-.08 (.03)*
Weekend Day	.08 (.06)	.17 (.07)*	.14 (.14)	.34 (.15)*
Stress Experience	.15 (.07)*	.29 (.09)**	.002 (.11)	.07 (.12)
Positive Mood	-.05 (.03)+	-.09 (.03)**	-.17 (.05)***	-.16 (.05)**
	Within-Person Negative Mood Model			
Intercept	2.17 (1.78)	2.31 (1.86)	4.10 (1.15)	3.51 (1.32)*
Age	.004 (.03)	.01 (.03)	.02 (.03)	.01 (.04)
Gender	-.17 (.78)	-.68 (.82)	-1.45 (.83)+	-.73 (.95)
Time of Day	-.06 (.02)**	-.02 (.02)	-.12 (.03)***	-.09 (.03)*
Weekend Day	.06 (.06)	.14 (.07)*	.16 (.14)	.36 (.15)*
Stress Experience	.19 (.08)*	.32 (.09)***	.02 (.11)	.09 (.12)
Negative Mood	.01 (.04)	.04 (.04)	.14 (.06)*	.13 (.06)*

Note.

+ $p < .10$;

* $p < .05$;

** $p < .01$;

*** p < .001. Age was a continuous variable. Gender (1 = Female; 2 = Male), Weekend Day (0 = Weekday; 1= Weekend), and Stress Experience (0 = No Stressor; 1 = Stress Occurrence) were dichotomous variables. Time of day was coded to indicate the EMA interval ranging from 1 to 5. Positive and negative mood were person-mean centered.