Effects of Day-to-Day Affect Regulation on the Pain Experience of Patients with Rheumatoid Arthritis

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Abstract

Background—Individual differences in the regulation of affect are known to impact pain and other symptoms in rheumatoid arthritis. However, no studies have yet used a rigorous daily diary methodology to address the question of whether current pain is reduced when positive or negative affects are effectively regulated.

Purpose—We used a prospective, repeated daily sampling design to infer the regulation of affect from day-to-day changes in affect intensity and examined how these changes in affect were prospectively related to pain from rheumatoid arthritis.

Method—Ninety-four adult patients diagnosed with rheumatoid arthritis completed daily measures of pain and positive and negative affect over a period of 30 days. Information on demographic and disease status variables was collected during a medical evaluation.

Results—Results of hierarchical linear model analyses indicated that the regulation of both positive and negative affect from the prior day to the current day predicted significantly greater decreases in pain that day, resulting in up to a 28% reduction in pain intensity. These findings were partly influenced by disease status and demographic variables.

Conclusion—This study suggests that the day-to-day regulation of negative and positive affect is a key variable for understanding the pain experience of individuals with rheumatoid arthritis and is a potentially important target for intervention.

Keywords

pain; arthritis; affect regulation; emotion; daily diary
1. Introduction

Recent studies suggest that the regulation of affect (viz., how one controls the experience and/or expression of emotion) appears to be important in understanding symptom expression in rheumatoid arthritis (Kelley et al. 1997; Smyth et al. 1999; van Middendorp et al. 2005a, 2005b). However, data addressing this topic has been primarily generated from cross-sectional studies that inherently define regulation of affect as relatively constant over time (i.e., a trait). This is inconsistent with broader literature showing that affect regulation is a dynamic process (Gross 1998) and other studies demonstrating that an individual’s affects themselves can wax and wane sometimes dramatically within a given day and across days (Stone and Shiffman 1994; Zautra et al. 2005). A broader understanding of the influence of affect regulation on pain from rheumatoid arthritis thus requires assessing an individual’s changes in affect across multiple time points.

To date, only one published study has evaluated the role of affect regulation in pain from rheumatoid arthritis using a prospective design. Hamilton and colleagues (2005) sampled pain and affect reports by phone weekly for 12 weeks from 81 women diagnosed with rheumatoid arthritis. Although affect was sampled prospectively, the researchers treated affect regulation as a trait by using a single set of scores taken from the Mood repair subscale of the Trait Meta-Mood Scale. Results showed that pain and negative affect were related at a lower level for women reporting strong affect regulation skills compared to women reporting poorer affect regulation skills.

Although the results of the Hamilton and colleagues study suggest that affect regulation may have special significance for understanding rheumatoid arthritis pain, the study leaves unaddressed the following question: are individuals with rheumatoid arthritis less likely to have pain when affect is regulated from one time point to the next? Answering this question requires defining affect regulation as a dynamic variable that changes over time. The only study to date that has evaluated affect regulation in such a way was conducted by Paquet and colleagues (Paquet et al. 2005) in a general sample of geriatric patients. Negative affect regulation was inferred from maintaining negative affects at or below one’s usual (mean) level between any two moments of data collection, or recovering back to one’s usual level of negative affect (or below) following a time of higher than average negative affect. Positive affect regulation was defined in a similar manner but using maintenance of or recovery to one’s usual level of positive affect (or above) as the criterion. The investigators found that increased negative and positive affect regulation were prospectively related to lower levels of pain intensity.

The present study sought to extend our understanding of how affect regulation impacts the pain experience of patients with rheumatoid arthritis by inferring affect regulation from day to day changes in affect intensities. Our primary hypothesis was that the regulation of positive and negative affect from one day to the next would be prospectively related to lower levels of pain intensity in rheumatoid arthritis patients.

2. Method

2.1 Participants

Participants for this study initially comprised 98 patients with rheumatoid arthritis who were studied at baseline before becoming involved in a larger study evaluating the efficacy of an emotional disclosure intervention. All patients were recruited from clinics affiliated with the Ohio University College of Osteopathic Medicine or Duke University Medical School. Patients were given a history and physical examination by one of the study rheumatologists, and diagnosis of rheumatoid arthritis was based on criteria established by the American College of...
Rheumatology (Arnett et al. 1988). Patients were excluded from the larger study if they had other known organic disease that would significantly affect pain or functioning (e.g., chronic obstructive pulmonary disease) or if they had other rheumatic diseases besides rheumatoid arthritis. In addition, patients were excluded if they had severe personality disorders or substance abuse problems or were involved in current psychiatric treatment. Finally, patients were excluded from the current analyses if they did not provide sufficient diary data (at least 20 days out of a possible 30); this criterion resulted in a loss of 4 participants, for a final sample of 94. Table 1 displays summary characteristics for the final sample.

### 2.2 Defining Affect Regulation

Affect regulation was assessed empirically to address the question for each patient of whether (and to what extent) negative and positive affect intensities changed between any two consecutive days of data collection, and if so, what impact this had on pain for the given day. Other researchers who have prospectively evaluated affect intensity in the context of arthritis have typically assessed the effects of affect at a given time by looking at both what happens to the given outcome variable at an individual's estimated usual (average) level of affect intensity (the intercept estimate in multilevel models) and at what happens when affect intensities deviate from that usual level (the slope estimate) (e.g., Schanberg et al. 2003; Zautra et al. 2001). The primary reference point for examining the effects of affect is thus the estimate of one's level of affect intensity. What these methods do not also consider as a reference point that is important when trying to prospectively evaluate affect regulation is the level of affect intensity at the prior assessment point. Evaluating affect with the additional consideration of changes in intensity relative to the reports of the individual on the prior day can yield additional important information more in line with current conceptions of affect regulation as a dynamic process.

In keeping with our intent of evaluating affect prospectively in a way that may have important implications for our understanding of the effects on pain of affect regulation, we coded affect regulation variables based on day to day changes in affect using an adaptation of the approach developed by Paquet and colleagues (Paquet et al. 2005). The affect regulation variables included two measures of positive affect regulation: (a) maintenance of positive affect, defined as keeping positive affect at or above one's usual (mean) level between any two days (potentially indicative of “antecedent-focused affect regulation” described in the broader literature on affect regulation, Gross 2002), and (b) recovery of positive affect, defined as increasing positive affect to a person's usual level or above of positive affect in response to a drop below the person's usual level the previous day (potentially indicative of “response-focused affect regulation,” Gross 2002). The affect regulation variables similarly included two measures of negative affect regulation: (a) containment of negative affect, defined as keeping negative affect at or below one's usual (mean) level between any two days (antecedent-focused affect regulation); and (b) recovery from negative affect, defined as bringing negative affect back down to at least a person's usual level or lower following an unusually high negative affect day (response-focused). By taking into account an individual's affect level the prior day and coding changes between the prior day and current day, this method permits an evaluation of our primary research question: what happens to a current day's pain intensity in adults with rheumatoid arthritis when yesterday's affect level is regulated?

### 2.3 Procedure

Following an initial screening appointment, a research assistant gave detailed instructions on completing diaries to participants. Each participant was given a set of 30 booklets (three pages each) that included measures of daily pain and positive and negative affect. Participants were told to complete a booklet once each night just before retiring for the next 30 days. To ensure compliance in completing diaries, participants were instructed to mail each night's completed
diary the following morning; postmarks on the envelopes were tracked for compliance monitoring. Participants were paid $0.25 for each booklet that was completed and returned according to these instructions, and earned an additional $1.25 for providing complete data for each half of the 30-day assessment period. Of the original 2940 total diaries to be completed (98 patients × 30 days), 2703 were completed and returned according to instructions, yielding a 92% overall diary completion rate. The diary completion rate was 96% for the 94 patients who composed the final study sample.

2.3 Measures

2.3.1 Daily Affect—Daily levels of positive and negative affective states were assessed using the Positive and Negative Affect Schedule (PANAS) (Watson et al. 1988). The PANAS is a 20-item scale that includes descriptors of both positive and negative affects. Participants indicate the degree to which they experienced a given affect that day using a response scale of 1 (very slightly or not at all) to 5 (extremely). Raw scores for negatively valenced descriptors are averaged to comprise an index of negative affect, and scores for positively valenced descriptors are averaged to comprise an index of positive affect. Many studies, including daily diary studies (Zautra et al. 2005), have used this instrument and have demonstrated its internal consistency. For the current study, between-person internal consistency estimates (Cronbach's alphas averaged across patients and days) were .92 for positive affect and .91 for negative affect. Within-person internal consistency estimates (Cronbach's alphas computed on within-person item $z$-scores) were .85 for positive affect and .82 for negative affect. The average day-to-day total score correlation was .80 for positive affect and .79 for negative affect.

2.3.2 Daily Pain—Pain intensity was assessed daily using a 100mm visual analog scale containing anchors of “no pain” and “pain as bad as it can be.” Patients were asked to mark the scale to indicate the average level of pain they experienced that day. This method of assessing pain is consistent with several other studies that have provided support for the reliability, validity, and sensitivity of visual analog pain scales and has been validated in other daily diary studies of rheumatoid arthritis patients (Lefebvre et al. 1999). In the present study, the average day-to-day correlation of pain scores was .80.

2.3.3 Demographic and Disease Variables—Demographic variables (age, gender, and education level) were assessed using an evaluation form developed by the investigators. Disease variables (disease duration in years and baseline count of swollen, tender, abnormal, and repaired joints) were assessed in a medical exam by a study rheumatologist. In addition, erythrocyte sedimentation rate assays were performed and values were used as an estimate of inflammation.

3. Data Analysis

3.1 Overview

Multilevel random coefficient model (MRCM) analyses were used to account for the nesting of the 30 measurement occasions (“level 1 units”) within the 94 participants (“level 2 units”). In MRCM analysis, coefficients describing the within-person relationship of an outcome taken at a given time (e.g., pain) to one or more predictors taken at a given time (e.g., maintenance of or recovery from a given level of affective intensity) are modeled at the between-person level (level 2) as a function of both fixed effects (e.g., estimates of population average values and/or potential between-person predictor variables) and random effects (variability of unexplained individual differences) (Nezlek 2001). Findings can thus be generalized to the population of rheumatoid arthritis patients from which our sample is drawn and to the population of days from which each assessment is sampled. HLM software (Raudenbush et al. 2004) was used to furnish full maximum likelihood estimates of the fixed and random model.
parameters. In all models, continuous level 1 variables were centered on each individual’s mean and continuous level 2 variables were centered on the grand mean. The affect regulation variables (maintenance of positive affect, containment of negative affect, and recovery of positive and negative affect) were dummy coded and left uncentered (with 0 representing unregulated affect for each dummy-coded variable).

### 3.2 Initial Analyses

Descriptive analyses were conducted on each of the daily diary measures and summarized using daily means, variances, and frequency distributions to explore normality assumptions. Potential reactivity associated with the multiple assessments was statistically evaluated by testing for temporal trends in the reports of pain and affect over the course of the study (i.e., by specifying “day” as a predictor of pain and affect in hierarchical linear models). Various forms for the error variance-covariance matrices associated with models were statistically compared by testing log-likelihood value differences against a chi-square sampling distribution; the form that most parsimoniously accounted for the pattern of auto-correlated residuals was selected for analyses. Fully unconditional models (i.e., intercept only models at level 1 and level 2) were fit prior to proceeding with our primary analyses to estimate the intraclass correlation coefficient (the proportion of variance in pain accounted for by individual differences relative to that accounted for by the occasion of measurement).

### 3.3 Affect Regulation Analyses

All multilevel conditional analyses proceeded by first testing a model of the effect of the affect regulation variables on pain, controlling for time (day) and coefficients of variation for negative and positive affect. Time was included as a level 1 covariate to control for potential time-related trends in responses, and coefficients of variation (within person standard deviation divided by the within person mean) for negative and positive affect were added as level 2 covariates to control for individual differences in variability of affect reporting. For example, the initial corresponding level 1 model for positive affect regulation was as follows: $Pain_{ij} = \pi_0 + \pi_1 \text{Day}_j + \pi_2 \text{PAmain}_{ij} + \pi_3 \text{PAreco}_{ij} + r_{ij}$, where “PAmain” is positive affect maintenance for the given individual (between the prior day and the current day) and “PAreco” is positive affect recovery for the given individual (between the prior day and the current day). The relevant corresponding level 2 models were specified as $\pi_0 = \beta_00 + \beta_01 \text{CVforPA} + r_{ij}$, $\pi_2 = \beta_20 + \beta_01 \text{CVforPA} + r_{ij}$, and $\pi_3 = \beta_30 + \beta_01 \text{CVforPA} + r_{ij}$. If statistical tests of the coefficients associated with these variables were not significant in the models (i.e., $p < .05$), they were dropped from the analysis.

Negative affect regulation was coded for each patient as containment of negative affect at or below one’s usual (mean) level between any two consecutive days (i.e., between $i-1$ and $i$) or recovery to one’s usual (mean) level or below following a day of higher than usual negative affect intensity. Positive affect regulation was coded for each patient in a similar way: maintenance of positive affect intensity at or above the patient’s usual (mean) level or a recovery from lower than usual positive affect intensity to at least the patient’s usual level (or greater) between any two consecutive days. Conditional multilevel models were then specified to evaluate the effect on current day’s pain of dummy-coded unregulated vs. maintained positive affect or contained negative affect, and of unregulated vs. recovered positive or negative affect. For example, the basic level 1 equation testing the effect of positive affect regulation on pain was as follows: $Pain_{ij} = \beta_0 + \beta_1 \text{PAmain}_{ij} + \beta_2 \text{PAreco}_{ij} + r_{ij}$, where pain on current day $i$ for individual $j$ is specified as a function of the individual’s intercept (his/her mean level of pain across days when positive affect is unregulated, viz., lower than usual for that individual), the change in a given day’s pain level when positive affect has been maintained from the previous day to the current day, and the change in a given day’s pain level when positive affect has been recovered to at least the patient’s usual (mean) level from the previous day to the current day.
The level 2 model was then specified to predict the level 1 coefficients based on the population mean (intercept) for the relationship (slope) between affect regulation and pain and a residual term corresponding to unaccounted for between-patient variance in the relationship between affect regulation and pain.

In order to test whether the affect regulation variables were related to current pain independent of the prior day's pain level, we also added prior day's pain as a control variable into the above models. For example, the level 1 model for positive affect regulation, controlling for previous day's pain level, was specified as:

\[ \text{Pain}_{ij} = \beta_0 + (\beta_1 \text{Pain}_{i-1}) + \beta_2 \text{PAmain}_{ij} + \beta_3 \text{PAreco}_{ij} + \epsilon \]

The corresponding level 2 models specified the intercept and slope coefficients as a function of the population average (plus unaccounted for between-patients error).

### 3.4 Exploratory Analyses

Based on prior research suggesting potential demographic and disease status differences in affect variables in rheumatoid arthritis patients (e.g., van Middendorp et al., 2005a, 2005c), we evaluated between-patient differences in affect regulation (by itself) and in the relationship between affect regulation and pain. Specifically, demographic (age, gender, and education level) and disease status variables (erythrocyte sedimentation rate, joint count, and disease duration) were specified as predictors of the (log) odds of regulating (versus not regulating) positive and negative affect using a logit link function in hierarchical linear models. Additionally, these variables were added as between-patient (level 2) variables and were specified as predicting the within-patient relationship (slope) between pain and affect regulation. If a coefficient associated with any of the demographic or disease status variables was statistically significant \( (p < .05) \) in these models, this would provide evidence of a moderation effect.

### 4. Results

#### 4.1 Descriptive Statistics

Table 2 presents descriptive statistics on the diary measures (and indices derived from the daily diaries), collapsed across day and patients. Maintenance of positive affect above the patient's mean was more common than recovering from days of lower than usual positive affect. Similarly, containment of negative affect at or below the patient's mean was more common than recovery from days of high negative affect. According to our empirical definitions, negative affect was more regulated on average than positive affect. Specifically, positive affect remained unregulated (i.e., lower than a patient's usual level) on 52% of days, whereas negative affect remained unregulated (i.e., higher than a patient's usual level) on 32% of days. One potential explanation for this is that higher than usual levels of negative affect were experienced as more aversive by patients and thus were more likely to prompt regulation efforts than lower than average positive affect.

We computed the correlation (using the phi coefficient for dichotomous data) between positive and negative affect regulation to test for independence. Although the resulting coefficient was statistically significant \( (\Phi = .15, p < .01) \) and thereby suggested an association between negative and positive affect regulation, enough of the variance between these two variables was unshared to justify conducting separate analyses for positive and negative affect. Collapsed across days, the distributions of continuous variables were relatively normal.

Figure 1a and 1b present daily pain as a function of regulated versus unregulated negative and positive affect, respectively. The effect of negative affect regulation (including both containment and recovery) on current day's pain appears to be more consistent than that of
positive affect regulation: On 93% of all occasions, pain is lower when negative affect is regulated to at or below a patient's usual level. On 76% of all occasions, pain is lower when positive affect is regulated to at or above a patient's usual level. Collapsed across days, the average level of pain when negative affect was unregulated (i.e., higher than a patient's usual level) was 36.61 (SD = 25.47). When negative affect was regulated (i.e., contained at or recovered to a patient's usual level or below), the average level of pain was 26% lower than when it was unregulated (M = 27.20, SD = 21.64). For positive affect, the average level of pain when positive affect was unregulated (i.e., lower than a patient's usual level) was 32.31 (SD = 24.29). The average level of pain when positive affect was regulated (i.e., maintained at or recovered to at least a patient's usual level) was 14% lower than when it was unregulated (M = 27.80, SD = 21.90).

Descriptive analyses were subsequently conducted to examine the effects of the two variants of affect regulation separately: 1) maintaining positive affect/containing negative affect, and 2) recovering positive and negative affect. These analyses showed that pain levels on average dropped by 28% to 26.20 (SD = 21.42) when individuals were able to contain negative affect at or below their usual level from the prior day to the current day. Pain levels dropped by 17% to 30.27 (SD = 22.03) when individuals were able to recover from a prior day's elevated level of negative affect by the current day. For positive affect, pain levels on average dropped by 17% to 26.69 (SD = 21.68) when positive affect was maintained at or above a patient's usual level. Pain levels dropped by 8% to 29.62 (SD = 22.15) when individuals were able to recover back to their usual level from a prior day's drop in positive affect.

4.2 Statistical Tests of the Effect of Affect Regulation on Pain

The unconditional means model was fit prior to adding affect regulation variables. The intraclass correlation coefficient for pain was estimated based on this model and was found to be .65 (thus, approximately 65% of the variance in daily pain reports in this sample is attributable to differences among patients). Conditional models were then specified with day to day positive and negative affect regulation variables as level 1 predictors of pain. Results are given in Table 3. As can be seen from Table 3, both negative affect containment (containing negative affect at or below an individual's usual level from a prior day to the current day) and recovery (decreasing negative affect to an individual's usual level or below following a day of higher than usual negative affect) were found to significantly predict lower levels of the current day's pain. Similarly, both positive affect maintenance (containing negative affect at or above an individual's usual level) and recovery (increasing positive affect to an individual's usual level or above following a day of lower than usual positive affect) were found to significantly predict lower levels of current day's pain.1 Time (day) was initially included in these models at level 1, and coefficients of variation for positive and negative affect were initially included at level 2. However, these variables were not significant and did not alter results; they were thus dropped from the models. In addition, prior day's pain was initially included as a control variable in these models. Findings for both positive and negative affect regulation remained significant and largely unchanged after accounting for prior day's pain.

4.4 Exploratory Analyses

Differences in positive and negative affect regulation as a function of demographic variables (age, gender, education level) and disease status variables (disease duration, joint count, and erythrocyte sedimentation rate) were evaluated using a logit link function in hierarchical linear models. Of the demographic variables, age predicted greater log odds of regulating positive affect (t(90) = 2.03, p = .04), and there was a trend suggesting greater log odds of regulating negative affect with increasing education (t(90) = 1.70, p = .09). No disease status variables significantly predicted positive or negative affect regulation, although there was a trend.
suggested lower log odds of regulating negative affect with increased erythrocyte sedimentation rate ($t(90)$ = -.01, $p$ = .10).

The disease and demographic variables were subsequently added to models to determine if the relationships between affect regulation and pain were different for different patients. Gender was not a significant moderator in any of the models. The relationship (slope) between recovery from higher than usual negative affect and current pain was greater for individuals with less education (or lower for individuals with more education), $b$ = 1.88, $t(90)$ = 2.38, $p$ = .02. Recovering positive affect to at or above a patient’s average level following a day of lower than usual positive affect had a more pronounced effect on reducing daily pain levels for individuals who were relatively younger, $b$ = .20, $t(90)$ = 2.10, $p$ = .04. In addition, active joint count significantly impacted the relationship (slope) between negative affect recovery and pain, $b$ = −.09, $t(90)$ = 2.21, $p$ = .03. There also was a trend for the effect of erythrocyte sedimentation rate (ESR) on the relationship between negative affect recovery and pain, $b$ = −.09, $t(90)$ = 1.78, $p$ = .08. The direction of both of these findings indicated that recovering negative affect to at or below a patient’s usual level following a day of higher than usual negative affect had a more pronounced effect on reducing daily pain levels for individuals with greater baseline disease activity.

5. Discussion

The purpose of the present study was to examine how affect regulation, inferred empirically from daily reports of positive and negative affect, relates to the pain experience of individuals with rheumatoid arthritis. Using multilevel modeling techniques, we found support for our hypothesis that the regulation of positive and negative affect from one day to the next is important in understanding the daily pain experience of individuals with rheumatoid arthritis. Individuals who contained negative affect levels at or below their typical level between any two days or decreased negative affect to usual levels or below following a day of elevated negative affect were found to experience significantly lower levels of arthritis pain on the current day. Also, individuals who maintained positive affect levels at or above their typical level between any two days or recovered more quickly from a prior day’s decrease in positive affect intensity were significantly related to pain on the next day, $t(1.29)$, $p$ = .20, and $t(.75)$, $p$ = .46. When looking at same day models, results from the main models presented in the paper (see Table 3) suggest that pain intensity on a given day for the average individual increases (or decreases) in affect intensity at a given day are predictive of pain that same day but not of pain the next day. By comparison, results from the models presented in the paper (see Table 3) suggest that pain intensity on a given day for the average individual would be expected to be reduced by up to 4.78 points when also considering how negative affect changed (i.e., was “contained” or “recovered”) between the prior day and given day and by up to 3.23 points when also considering how positive affect changed (i.e., was “maintained” or “recovered”) between the prior day and given day. In particular, the unique information about how the current pain experience is likely to be affected by changing affect levels back to a typical level or “better” for a given individual (i.e., a reduction in pain of 3.89 points for negative affect “recov­ery” and 2.08 points for positive affect recovery) has important clinical and theoretical implications about affect regulation that cannot be derived from the comparison models described in this footnote.

Although there is no way to explicitly statistically compare the “traditional” method of prospectively evaluating affect to the prospective affect regulation models described in this paper, the two approaches may be crudely compared based on level 1 variance components (i.e., variance left unaccounted for in pain after the level 1 affect variables are entered). When comparing the negative affect models, specifying affect based on day to day changes produced a lower level 1 residual variance in predicting pain ($t(172)$, $p$ = .27) relative to the residual variance when specifying affect as deviations from the individual’s mean ($t(180)$, $p$ = .39). Similar results were observed for comparing the different ways of specifying positive affect ($t(171)$ vs. $t(177)$). Thus, examining affect using the “affect regulation” definitions presented in this paper (adapted from Paquet et al. 2005) appears to produce somewhat superior prediction of the pain experience of patients with RA. Further, this method furnishes more information about how pain changes with important day to day within-person affect shifts than is available with defining affect based only with reference to the individual’s estimate of usual affect intensity.
affect were found to experience significantly lower levels of pain intensity on the current day. These findings remained after accounting for the prior day’s pain level but were partly influenced by disease status and demographic variables.

The finding that negative and positive affect regulation from day to day were prospectively related to the pain experience of individuals with rheumatoid arthritis lends further support to current theories of pain and fits well with results from other studies. In particular, emotional processing is a central component of both the “neuromatrix” theory of pain (Melzack 1999), in which regulation of emotional inputs is thought to affect what is ultimately the “output pattern” for pain, and the “homeostatic” model of pain (Craig 2003), in which emotions are thought to impact the experience of pain through how they influence the perception of the body’s condition at any given time. Other studies have found that affect regulation, albeit defined cross-sectionally at a single time point, impacts perceptions of health (van Middendorp et al. 2005b) and recovery from rheumatoid arthritis pain (Hamilton et al. 2005). Further, regulation of specific affects has been found to prospectively influence pain in a general sample of residents at a geriatric facility (Paquet et al. 2005).

Results of analyses in which the potential moderating effect of demographic and disease status variables were taken into account suggested that the effect of affect regulation on pain varied somewhat based on one’s demographic and disease status variables. Specifically, recovery of positive affect intensity to one’s normal (or above) level had a more pronounced effect on reducing current pain levels for patients who were relatively younger. Recovery of negative affect intensity to one’s normal (or below) level had a more pronounced effect on reducing current pain levels for patients with relatively greater baseline disease activity and less education. Analyses also suggested that age was associated with greater odds of regulating positive affect, and education was somewhat associated with greater odds of regulating negative affect. In general, research has suggested that competency and stability in affect regulation increases over the life span and with education level, and there is a positive association between age and maintenance of positive affect and between education and controlling negative affect (Pasini et al. 1992; Gross et al. 1997; Mroczek and Kolarz 1998). Thus, age may have impacted the relationship between positive affect recovery and pain by virtue of relatively older individuals having less day-to-day variability in recovering positive affect. Similarly, education may have impacted the relationship between negative affect recovery and pain by virtue of relatively more educated individuals having less day-to-day variability in recovering negative affect. Individuals with greater disease activity also would be expected to have more day-to-day variability in negative affect. To test these possibilities, we conducted post-hoc analyses and indeed found a positive association between within-patient negative affect variances and both erythrocyte sedimentation rate ($r = .30, p < .01$) and joint count ($r = .20, p = .05$). We also found a significant inverse association between age and the coefficient of variation for positive affect ($r = -.26, p = .01$), suggesting that age is associated with less variability in positive affect relative to a patient’s mean. The finding for education level, however, could not be explained based on differences in affect regulation variability and remains a question for future studies.

Although the results of this study largely concur with findings in related literature, there are several important advances. This study to our knowledge is the first to examine affect regulation prospectively in a specific disease-related pain population. Clearly patients with rheumatoid arthritis must contend with a multitude of disease-related and associated factors that may differentially impact their emotional state on any given day. However, the extant literature on pain and emotion in rheumatoid arthritis has fallen short of capturing how differences in how a given patient contends with emotional ups and downs over time impact his or her experience of pain. Data from the present study suggest that pain from rheumatoid arthritis can be lessened based on modulating affect levels from one day to the next. Further, defining affect regulation
prospectively has direct clinical implications for intervening in disease-related pain: Although all patients start with a trait-like “usual” level of positive and negative affect, our data suggest that each patient's pain experience may be significantly impacted by teaching response-focused affect regulation skills (i.e., bringing one's negative and positive affect back to desirable levels following inevitable “bad days”) and antecedent-focused affect regulation skills (i.e., preventing unusually high negative affect or low positive affect days). Additionally, results suggested that the regulation of both negative and positive affect is important in understanding pain related to rheumatoid arthritis. As noted by Zautra et al. (2001), prior studies have disproportionately emphasized the influence of negative affect, but our study suggests that the regulation of both positive and negative affect have unique significance in understanding disease-related pain.

Although our data demonstrate a prospective link between affect regulation and pain in rheumatoid arthritis, we did not evaluate the mechanisms of this relationship. There are a number of possible pathways through which affect regulation may operate. For example, affect regulation may impact symptoms of rheumatoid arthritis through its influence on neuroendocrine and/or immune system variables (van Middendorp et al. 2005c). There also are data to suggest that unregulated negative affect can reduce cognitive resources that might otherwise be available for cognitive reappraisal of pain (Gross 2002). Finally, individuals who are poor at regulating affect may find it difficult to develop and sustain social relationships that could serve to buffer the impact of pain. Exploration of these biological, cognitive, and social pathways in future daily process studies could significantly advance our understanding of the pain experience of patients with rheumatoid arthritis and may suggest novel avenues for clinical intervention.

Our results need to be interpreted within the limitations of the study. Our sample was relatively well educated and primarily Caucasian, which may not be representative of the general population of rheumatoid arthritis patients. Subsequent studies should include multiple rheumatology sites having varying demographic patient profiles to determine if findings are generalizable. In addition, there remain limitations associated with inferring affect regulation from day to day changes in affect intensities. Inferring that these changes result from active attempts by patients to reset or maintain affect levels (consistent with the conception of affect “regulation”) may not be accurate as there may be additional reasons for these changes. We accounted for this in part by controlling for individual differences in the variability of how individuals report affect relative to mean levels (coefficients of variation), but it remains possible that day to day changes in affect levels were produced by variables other than patient volition. Nonetheless, the data suggest that changing (or maintaining) affect levels from a prior to a current day can systematically influence a patient's arthritis pain. As another limitation to our definition of affect regulation, we based this on day to day changes in affect intensities rather than within-day changes. It is possible that findings would have been different had we sampled pain and affect reports using an ecological momentary (real-time) assessment design. A subsequent study of arthritis patients could sample pain and affect reports randomly throughout a given day to determine if regulating affect between moments in a given day has an impact on pain at a subsequent time that day. Subsequent studies also could explore multiple levels of observing the relationship between affect regulation and disease-related pain by combining both moment to moment and trait affect regulation measures. Such studies could further broaden our understanding of the links between affect regulation and pain.

In conclusion, data from the current study suggest that regulation of affect defined empirically based on changes in affect intensities from one day to the next is an important variable for understanding the pain experience of individuals with rheumatoid arthritis. Future research could expand on these findings by examining potential mediating pathways, further exploring demographic and disease status variables that moderate the affect regulation and pain
relationship, and devising interventions capable of modifying how individuals with rheumatoid arthritis regulate affect on a day to day basis.

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Figure 1a.
Daily pain as a function of unregulated versus regulated negative affect
Figure 1b.
Daily pain as a function of unregulated versus regulated positive affect
Table 1

Sample characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender [n %]</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>26 (28%)</td>
</tr>
<tr>
<td>Female</td>
<td>68 (72%)</td>
</tr>
<tr>
<td>Age [mean, SD]</td>
<td>56 years (10.60 years)</td>
</tr>
<tr>
<td>Race [n %]</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>85 (91%)</td>
</tr>
<tr>
<td>African-American</td>
<td>5 (5%)</td>
</tr>
<tr>
<td>Native-American</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Education Level [n %]</td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Some high school</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>Completed high school</td>
<td>16 (17%)</td>
</tr>
<tr>
<td>Some college</td>
<td>22 (23%)</td>
</tr>
<tr>
<td>College degree</td>
<td>20 (21%)</td>
</tr>
<tr>
<td>Graduate training or degree</td>
<td>18 (19%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>11 (12%)</td>
</tr>
<tr>
<td>Disease duration [mean, SD]</td>
<td>14 years (8 years)</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate</td>
<td>23.96 mm/hr (20.54 mm/hr)</td>
</tr>
</tbody>
</table>
Table 2
Means and standard deviations for daily diary variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain VAS [mean, SD]</td>
<td>30.00 (23.35)</td>
</tr>
<tr>
<td>PANAS Positive Affect [mean, SD]</td>
<td>4.00 (1.73)</td>
</tr>
<tr>
<td>PANAS Negative Affect [mean, SD]</td>
<td>3.60 (.30)</td>
</tr>
<tr>
<td>Derived Variables</td>
<td></td>
</tr>
<tr>
<td>Positive Affect Regulation</td>
<td></td>
</tr>
<tr>
<td>% Unregulated Days</td>
<td>52.1%</td>
</tr>
<tr>
<td>% Maintained Days</td>
<td>29.6%</td>
</tr>
<tr>
<td>% Recovered Days</td>
<td>18.3%</td>
</tr>
<tr>
<td>Negative Affect Regulation</td>
<td></td>
</tr>
<tr>
<td>% Unregulated Days</td>
<td>31.7%</td>
</tr>
<tr>
<td>% Maintained Days</td>
<td>51.5%</td>
</tr>
<tr>
<td>% Recovered Days</td>
<td>16.8%</td>
</tr>
</tbody>
</table>
## Table 3
Results of hierarchical linear analyses predicting pain from affect regulation

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>B</th>
<th>t(df)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative affect model intercept *</td>
<td>33.18</td>
<td>15.90 (93)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Negative affect maintenance</td>
<td>-4.78</td>
<td>4.80 (93)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Negative affect recovery</td>
<td>-3.89</td>
<td>3.66 (93)</td>
<td>.001</td>
</tr>
<tr>
<td>Positive affect model intercept *</td>
<td>31.40</td>
<td>14.84 (93)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Positive affect maintenance</td>
<td>-3.23</td>
<td>2.67 (93)</td>
<td>.009</td>
</tr>
<tr>
<td>Positive affect recovery</td>
<td>-2.08</td>
<td>2.30 (93)</td>
<td>.024</td>
</tr>
</tbody>
</table>

* The coefficient estimate for the intercept in these models refers to the average level of pain when affect is unregulated.