

Global Versus Momentary Osteoarthritis Pain and Emotional Distress: Emotional Intelligence as Moderator

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Abstract

Background Pain and emotional well-being are complexly associated both globally and in the moment. Emotional regulation strategies may contribute to that complexity by shaping the pain–well-being association.

Purpose Using emotional intelligence (EI) as an integrative conceptual framework, this study probed the role of emotional regulation in the associations of osteoarthritis pain with emotional well-being in varying time frames. Perceived attention to, clarity, and regulation of emotions were examined as predictors of well-being, and as moderators of the well-being–pain association, at global and momentary (within-day) levels.

Methods In a microlongitudinal study, 218 older adults with physician-diagnosed knee osteoarthritis self-reported global pain, depressive symptoms, and EI (mood attention, clarity, and repair). Momentary pain and positive and negative affect were then assessed four times daily for 7 days. EI subscales were examined as moderators of the pain–well-being association at global and

momentary levels, controlling demographics and general health.

Results Global and momentary pain were positively associated with mood clarity and negatively with attention, but not with repair. Clarity and repair negatively predicted depression, and buffered effects of pain on depression. Momentary negative affect was negatively predicted by mood clarity and repair; again, clarity and mood repair buffered effects of momentary pain on negative affect. Only mood repair predicted positive affect, with no interactions emerging.

Conclusions Attention to mood states exacerbates the experience of pain in both short and long terms. In contrast, both mood clarity and ability to repair moods appear important to both momentary and longer-term emotional well-being.

Keywords Osteoarthritis • Pain • Depression • Affect • Emotional intelligence

Introduction

The experience of pain, particularly chronic pain, clearly has a strong emotional component. At the broadest level, a large and still growing literature documents the association of persistent pain with diagnosable psychopathology [1]. Historically, depression has received the greatest research attention, with the consistent finding that chronic pain is a strong risk factor for both diagnosable depressive disorders and lower level symptoms [2]. The linkage of pain with depression has been demonstrated both cross-sectionally and longitudinally, and appears to be bidirectional [3, 4].

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Research on the link between pain and emotional distress has, over the past two decades, moved beyond global symptomatology to examine short-term dynamics among pain and various indices of subjective well-being. Experimental work has shown that induced negative mood increases the unpleasantness of pain [5] (but see [6]), as well as its rated intensity and pain tolerance [7]. Conversely, positive mood induction reduces pain in laboratory settings [7, 8]. Several in vivo microlongitudinal studies, using daily or weekly diary approaches, have similarly demonstrated that variation in persistent pain is associated in predictable ways with contemporaneous and lagged fluctuation in mood states [9–12]. Similar patterns have been documented on even shorter, within-day time frames using experience sampling methods [13–15].

Microlongitudinal studies also illustrate how short-term affect may interact with more stable personal characteristics to influence the experience of pain. For example, Graham-Engeland and colleagues [15], using experience sampling with a sample of persons with rheumatoid arthritis, found that globally assessed depressive symptoms and momentary positive affect contributed independently to momentary pain. Interestingly, the association of negative affect with pain was attenuated by statistical control of stress, age, gender, and time of measurement. History of depression, net of current distress, has similarly been shown to intensify the day-to-day relationship of pain with mood [16, 17]. Broader personality and cognitive characteristics are also associated with momentary pain, often in complex ways. For example, Affleck and colleagues [9] found that neuroticism moderated the daily pain–mood linkage among rheumatoid arthritis patients, such that persons higher in neuroticism displayed weaker within-day associations of pain with affect. A similar interaction of trait and state anger on momentary pain ratings has been reported among persons with lower back pain [18].

Given this close and somewhat complicated linkage of pain with short- and long-term emotional function, it is not surprising to observe growing interest in emotion regulation as a modulator of chronic and acute pain. Several lines of inquiry indicate that awareness of and ability to regulate one's emotions may be a crucial aspect of psychological response to chronic pain. A prime example is mindfulness, which has been related to reduced pain intensity and interference both in treatment studies [19] and in correlational research on trait mindfulness [20, 21]. Observed short-term regulation of mood states—recovery from negative affective states or maintenance of positive mood—has similarly been related to reduced pain intensity in such diverse groups as hospitalized older adults [22], rheumatoid arthritis patients [23], and children with juvenile idiopathic arthritis [24]. Conversely, instability of negative affect is associated

with increased daily pain severity [12], again underscoring the importance of mood regulation as a component of pain self-management. The high prevalence of alexithymia among chronic pain patients [25, 26] further suggests that inability to identify or articulate one's own emotions may exacerbate (or, perhaps, be exacerbated by) pain.

Although the studies cited above provide some compelling glimpses of the dynamics of emotional response to pain, they fail to capture in integrative fashion the full complement of emotional functions that may come into play. Mindfulness, in either state or trait form, is essentially pure *awareness*: attention to emotions and the context in which they occur, without attempt to evaluate or modify them. In contrast, work on alexithymia highlights *clarity* of moods, in terms of ability to understand, articulate, and differentiate one's feelings. Studies of mood variability capture direct or unconscious efforts to *regulate* emotional states, presumably in order to control their effects on physical and emotional well-being. Each of these functions appears to be an important element of emotional response to chronic pain. However, these various components have typically been examined separately and independently, without reference to how they may interact to affect pain and its emotional impact.

A potential consolidating framework is the construct of *emotional intelligence* (EI) as set forth by Salovey, Mayer and colleagues [27, 28]. Working from an abilities-focused perspective, these investigators conceptualized EI as the capacity correctly to perceive emotions in oneself and others; to use emotions to facilitate adaptive action; to understand the causes, consequences, and flow of emotions, and to manage emotional experience. In the context of persistent pain, this implies that EI may facilitate monitoring and managing emotional responses to pain—and, hence, avoiding its long-term harmful effect on psychological and functional well-being.

There have been only a few attempts explicitly to apply the EI framework to the experience and effects of chronic pain. In two experimental studies, Ruiz-Aranda, Salguero, and Fernández-Berrocal demonstrated that general EI [29], as well as more specific perceived ability to repair or regulate one's mood [30], predicted reduced sensory and affective response to a cold pressor task. This overall negative relationship of global EI with pain has been replicated in self-report studies of persons with chronic pain [31]. Particularly interesting are findings regarding the moderating effects of specific mood regulation abilities on the association of pain with both emotional well-being and transient mood states. Using a 2-year longitudinal design, Kennedy and colleagues [32] found that perceived mood clarity—the extent to which persons feel certain of their emotions and the implications thereof—buffered the negative relationship of osteoarthritis pain with depressive symptoms. In the shorter term, Zautra

and colleagues [10] assessed pain and mood weekly for 12–20 weeks in a sample of women with diverse painful conditions. Paralleling Kennedy et al. [32], they reported a buffering effect of mood clarity on the pain–positive affect association among older women with arthritis (both osteoarthritis and rheumatoid arthritis), but did not observe that effect in women with fibromyalgia. The same team, analyzing data only for women with rheumatoid arthritis, identified a similar short-term effect for mood repair [33]. Here, high perceived ability to regulate one’s own moods attenuated the negative impact of current pain on next-week positive affect.

In sum, it appears that the self-regulatory abilities that comprise EI may be important contributors to individuals’ maintaining positive emotional well-being in the face of persistent pain. However, as noted earlier, the literatures on emotional awareness, clarity, and regulation have developed largely independently. There has been little attempt to examine EI in integrative fashion, identifying which aspects of the trait may be most strongly predictive of adaptive response to pain. Further, the majority of research in this area has focused *either* on global emotional well-being, measured in terms of depression, anxiety, or other mood-related syndromes, *or* on short-term affective response to pain and its accompanying limitations. Aside from a handful of studies linking, for example, current or past depressive disorders with daily or momentary responses to pain [15, 16, 34], we know little about how momentary emotional responses translate to more generalized, global well-being among individuals with persistent pain.

To address these gaps, the current research examined the association of multiple aspects of EI with emotional well-being among a sample of older adults with osteoarthritis of the knee. Using the Trait Meta-Mood Scale [35], we examined unique contributions of perceived attention/awareness, clarity, and regulation of emotions to indices of emotional well-being at both global and momentary levels. For global analyses, we followed the bulk of extant literature, operationalizing emotional well-being negatively, in terms of depressive symptoms, and using a general self-report measure of pain intensity. For momentary analyses, we used experience sampling method to capture pain and positive and negative affect at multiple points over a 7-day period. At both levels of analysis, we explored both main effects of EI components on emotional well-being as well as their moderating effects on the relationship between pain and emotional well-being.

Method

Sample and Recruitment

The sample comprised 218 individuals with physician-diagnosed osteoarthritis of the knee, recruited

from west-central Alabama ($N = 133$) and Long Island, New York ($N = 85$) as part of an ongoing longitudinal study. Recruitment methods were diverse and differed somewhat at the two sites. In Alabama, respondents were recruited from a university-based general medical clinic, an urban rheumatology clinic, and a network of federally qualified health centers; from senior centers and other community service networks for older adults, and through public service announcements and word of mouth. The New York sample was recruited from university-based general medical clinics, advertisements in campus publications, and commercially prepared mailing lists. Respondents were required to be at least 45 years of age, able to converse over the telephone in English, and to provide contact information for a physician willing to confirm the knee osteoarthritis diagnosis. Exclusion criteria were presence of other painful or disabling disorders (e.g. rheumatoid arthritis, lung disease requiring oxygen use, current malignancy) and cognitive impairment sufficient to preclude completion of interviews (Short Portable Mental Status Questionnaire score ≥ 6 ; [36]). A final selection criterion was completion of at least 14 of 28 scheduled within-day phone calls (see “Measures and Procedures,” below).

Recruitment process varied by referral source, as approved by institutional review boards at participating institutions. Clinic patients and those on nonclinical mailing lists received a letter introducing the study and alerting them to expect a telephone inquiry; an opt-out card was included for those who preferred not to be contacted. At community service centers, project staff gave brief informational presentations and described the research; respondents to flyers, public service announcements, and word of mouth phoned the project offices directly. Regardless of referral route, interested individuals were first screened for eligibility and, if interested, gave verbal assent to participate. They then received a mail-out package including an informed consent form, authorization for physician contact to confirm the osteoarthritis diagnosis, and a packet of self-report questionnaires. After physician confirmation was obtained, an in-person interview was scheduled to complete the consent process, retrieve completed questionnaires, collect additional data, and train respondents on the experience sampling protocol.

Table 1 presents demographic characteristics and all study variables for the final sample of 218.

Measures and Procedures

Background characteristics

Background characteristics, included as potential covariates, were *race* (African American vs. non-Hispanic white), *age*, *gender*, *marital status* (married/cohabiting

Table 1 Sample Characteristics and Primary Study Variables

Variable (range in this sample)	Mean or <i>N</i>	<i>SD</i> or %
Age, years (48, 97)	64.6	9.3
Male	53	24.3%
Female	165	75.7%
African American	85	39.0%
Non-Hispanic White	133	61.0%
Unmarried ^a	109	50.5%
Married/cohabiting	107	49.5%
Grade school or less	27	12.4%
High school graduate	47	21.6%
Some post-high school training	50	22.9%
College graduate	40	18.3%
Graduate/professional degree	54	24.8%
Osteoarthritis in both knees ^a	133	61.6%
Duration of osteoarthritis symptoms, months (0, 624)	133.4	117.4
Health problems (0, 13)	3.28	2.67
Mood attention (1.92, 4.67)	3.41	0.44
Mood clarity (2.33, 4.92)	3.63	0.45
Mood repair (2.33, 5.00)	3.77	0.50
Depressive symptoms (0, 54)	11.0	10.4
Significant depression (CESD \geq 16)	50	22.9%
Global pain (0, 4.17)	2.05	0.88
Momentary positive affect (1.32, 4.99)	3.29	0.70
Momentary negative affect (1.00, 3.53)	1.29	0.40
Momentary pain (1.00, 4.90)	2.04	0.80

^a*N* = 216.

vs. not), and *education* (5-point scale, “less than high school” through “graduate degree”). *Physical health* was represented by a count of chronic and acute conditions experienced over the past year on a 28-item checklist (e.g. heart trouble, diabetes, broken hip).

Global measures

Global measures included: (i) *pain*, assessed with the Philadelphia Geriatric Center Pain Scale [37], a 6-item measure of pain intensity currently and over the past few weeks. Cronbach’s alpha for the current sample was .857. (ii) *Depressive symptoms* were captured with the Center for Epidemiologic Studies Depression Scale (CESD [38]; 20 items, $\alpha = .907$). (iii) EI was tapped by the three subscales of the 30-item Trait Meta-Mood Scale [35]. (a) *Attention to mood* (12 items; $\alpha = .724$) addresses the extent to which persons focus on their emotions and believe that emotions should guide action, for example, “I often

think about my feelings,” “I believe in acting from the heart.” (b) *Mood clarity* (12 items, $\alpha = .800$) represents insight into and understanding of one’s own emotions, for example, “Sometimes I can’t tell what my feelings are” (reverse coded), “I feel at ease about my emotions.” (c) *Mood repair* comprises six items that address maintaining positive affect balance, for example, “When I become upset, I remind myself of all the pleasures in life.” Although alpha for this subscale attained only .580, we retained the composite because of its conceptual importance for current analyses.

Experience sampling measures

Experience sampling measures were obtained during a 7-day protocol during which respondents, using personal phones or cell phones provided by the project, were telephoned four times daily by trained research assistants. The calls, each lasting roughly 5 min, were randomized within 3-hr blocks over a 12-hr time period daily. Measures relevant to the current analyses included (i) *momentary pain* at the time of the call, rated on a 5-point scale (“not at all” to “extremely”). (ii) *Momentary positive* and (iii) *negative affect* were assessed with the Philadelphia Geriatric Center Positive and Negative Affect Scales [39]. The five positive (*content, energetic, happy, interested, warm toward others*) and five negative items (*annoyed, depressed, irritated, sad, worried*), each rated from 1 (“not at all”) to 5 (“extremely”), were averaged to produce composite positive and negative affect scores for each call. Based on data from the first call of the first day’s experience sampling protocol, Cronbach’s alpha for the positive affect composite was .761 and for negative affect, .756.

In such an intensive procedure, some data loss is to be expected. For current analyses, we excluded 6 of 224 individuals (0.27%) for whom fewer than 14 of the 28 calls yielded complete data. Number of completed calls for the 218 active participants ranged from 14 to 29 (one respondent accidentally received 8 days’ calls). The modal number of calls yielding valid data was 23 ($M = 22.8$, $SD = 3.3$); only 17% of the sample completed fewer than 20 calls. Overall, 81.4% of calls were completed and yielded useable data.

Analysis Plan

Covariates for primary analyses were selected via a series of ordinary least squares multiple regression analyses for relationships of background characteristics with global pain, depressive symptoms, and EI composites; parallel multilevel models were used for momentary pain and positive and negative affect. Primary analyses examined the associations among EI, pain, and emotional well-being at global and momentary levels. For global measures,

regression analyses first delineated the association of the three EI variables (attention, clarity, repair) with pain, controlling for covariates. We then examined independent and moderating effects of EI variables and global pain upon depressive symptoms, using centered variables to compute multiplicative interaction terms (We recognize the advantages of bootstrapping techniques, such as those advanced by Hayes [44] for examining moderator effects in ordinary least squares regression. However, to our knowledge, no currently available program permits testing of more than one interaction at a time. Thus, for parsimony, we examined interactions in omnibus fashion using traditional multiplicative interaction terms. We replicated all moderation analyses in univariate fashion using Hayes's PROCESS macro [44]. Patterns of significant results exactly replicated those reported here.)

Relationships among momentary pain, positive and negative affect, and EI were explored using random intercepts multilevel models that paralleled those for global variables. This approach permits examination of patterns of within-person covariation of momentary pain and affect (Level 1, within-person variables) as a function of between-person (Level 2) individual differences [40]. Initial analyses examined Level 2 (between person) linkages of mood attention, clarity, and repair with momentary pain across all experience sampling data points; improvement in prediction over covariates alone was tested using a likelihood ratio test distributed as χ^2 . Next, associations of pain with momentary positive and negative affect were tested separately in nested equations, beginning with covariates and momentary pain at Step 1. Here, two measures of pain were used. *Average momentary pain*, the within-person mean of pain ratings across all phone calls, functioned as a Level 2 variable to represent individual differences in overall levels of pain assessed in the moment. *Momentary pain* ratings at each call were person-centered by subtracting average momentary pain from the momentary rating, thus yielding a measure of momentary deviation from one's own average at each experience sampling data point. At Step 2, group-centered mood attention, clarity, and repair were entered as Level 2 predictors, followed at Step 3 by multiplicative interaction terms testing moderating effects of each of the three EI variables on the within-person association of person-centered momentary pain with mood. Likelihood ratio tests again estimated improvement in model fit at each step.

Results

Covariate Analysis

Regression equations using background characteristics to predict global pain, depressive symptoms, and the EI variables (attention, clarity, repair) were all significant;

test statistics are available on request. Age was significantly related to depressive symptoms ($\beta = -.237, p < .001$) and marginally to pain ($-.114, p < .07$). Gender predicted attention to mood, with women scoring higher on that variable, $\beta = .193, p < .007$. Education predicted lower levels of pain ($\beta = -.297, p < .001$) and greater mood clarity ($\beta = .212, p < .004$). Marital status (1 = single, 2 = coupled) was associated with mood clarity (.163, $p < .03$), and marginally with mood repair (.139, $p < .07$). Physical health was a significant predictor of pain ($\beta = .173, p < .02$), depression (.262, $p < .001$), and mood clarity ($-.149, p < .05$). The only association that race showed with any outcome was a marginal coefficient for pain, $\beta = .124, p < .06$.

Parallel associations of potential covariates with momentary pain and affect were examined using multilevel modeling. Age was significantly associated with pain ($b = -.014, SE = .006, p < .02$), positive affect (.017, .005, $p < .001$), and negative affect ($-.006, .003, p < .05$). Number of health conditions also predicted pain ($b = .072, SE = .022, p < .001$), positive affect ($-.044, .018, p < .02$), and negative affect (.031, .011, $p < .007$). Momentary positive affect was positively related to education ($b = .072, SE = .027, p < .01$) and marital/partnered status ($-.193, .097, p < .05$; married/partnered as index group). Neither gender nor race was associated with momentary pain or affect.

Because race showed only a single, marginal association with any of the primary variables of interest, it was excluded from further analysis. Age, gender, education, marital status, and physical health were controlled in all further analyses.

EI, Pain, and Depressive Symptoms

A first analysis delineated the association of EI with global pain. An initial regression equation, including the five covariates, yielded a significant overall effect, $F(5, 210) = 13.31, p < .001$, attributable to effects of education ($\beta = .332, p < .001$), health conditions (.173, $p < .02$), age ($\beta = -.122, p < .05$) and, marginally, marital status ($-.114, p < .092$). Addition of EI variables significantly increased explained variance, $\Delta F(3, 207) = 6.94, p < .001$. Mood clarity was the strongest contributor, $\beta = -.305, p < .001$, reflecting lower pain among persons with higher perceived mood clarity. Attention was positively associated with global pain, $\beta = .202, p < .002$, but no effect emerged for mood repair.

To examine linkages of EI with global emotional well-being, we regressed CESD depression onto covariates and pain (Step 1), and then added EI variables as a block (Step 2). At Step 3, interaction terms were entered to test moderating effects of mood attention, clarity, and repair upon the pain–depression relationship. Table 2 presents results for each step.

Table 2 Depressive Symptoms as a Function of Background Characteristics, Mood Regulation Strategies, and Global Pain

	Step 1			Step 2			Step 3		
	<i>R</i>	Adj. <i>R</i> ²	β	<i>R</i>	Adj. <i>R</i> ²	β	<i>R</i>	Adj. <i>R</i> ²	β
	.579	.316		.725	.505		.756	.546	
Sex			.050			.088*			.077
Age			-.177****			-.173*****			-.141****
Marital status			-.055			.017			.010
Education			.070			.117**			.115**
Health conditions			.181***			.123**			.134**
Global pain			.471*****			.382*****			.399*****
Mood attention						.022			.024
Mood clarity						-.293*****			-.278*****
Mood repair						-.242*****			-.228*****
Attention \times pain									.072
Clarity \times pain									-.197*****
Repair \times pain									-.044

Step 1: $F(6, 209) = 17.58, p < .001$; Step 2: $F(9, 206) = 25.37, p < .001$; Step 3: $F(12, 203) = 22.54, p < .001$.

*.05 < p < .10; ** p < .05; *** p < .01; **** p < .005; ***** p < .001.

At Step 1, an overall significant effect reflected the association of depressive symptoms with age, physical health, and pain. Entry of EI variables at Step 2 significantly increased explained variance, $\Delta F(3, 206) = 27.58, p < .001$; mood clarity and repair, but not attention, were significant predictors. The association of education with depressive symptoms was artifactually increased to traditional significance levels; no other changes in patterns of significance were observed.

At Step 3, addition of the interaction terms again improved prediction of depression, $\Delta F(3, 203) = 7.18, p < .001$, due entirely to the interaction of pain with mood clarity (rightmost columns of Table 2). To address concern about overfitting of the Step 3 model, we reran the analysis entering only one interaction term at a time. When entered alone, the mood clarity \times pain interaction again significantly improved prediction, $\beta = -.203, \Delta F(1, 205) = 18.80, p < .001$. Also as before, there was no pain \times attention interaction, $\beta = .013, \Delta F < 1$. In contrast to the block entry model, when entered alone, mood repair significantly modified the association of pain with depressive symptoms, $\beta = -.140, \Delta F(1, 205) = 8.32, p < .004$. Regression slopes plotted at low ($-1 SD$), medium (mean), and high ($+1 SD$) levels of mood clarity (Fig. 1A) indicate that the relationship between pain and depression is attenuated among individuals with greater mood clarity. A similar buffering effect was observed for mood repair (Fig. 1B).

EI, Pain and Momentary Affect

Initial, null multilevel models were run to estimate the proportion of variance in momentary pain, positive

affect, and negative affect attributable to between-person versus within-person variability. Resulting intraclass correlations were .472 for pain, .506 for negative affect, and .650 for positive affect, indicating sufficient within-person variability to proceed with primary analyses.

A first analysis examined EI variables as predictors of momentary pain. An initial model containing only covariates, $-2LL = 12272.45$, was significantly improved with addition of the EI variables, $-2LL = 12263.81, \chi^2 = 8.64, df = 3, p < .04$. Significant effects emerged for age, $b = -.013, SE = .006, p < .03$, and health conditions, $b = .065, SE = .022, p < .004$, reflecting greater pain among younger individuals and those with more health problems. Momentary pain was predicted by attention to mood, $b = .271, SE = .125, p < .04$, and marginally by mood clarity, $b = -.304, SE = .158, p < .06$.

The associations of EI and momentary pain with momentary affect were examined first for positive affect. Table 3 presents the final step of the nested analysis. The effect of covariates alone, $-2LL = 7980.61$, was significantly improved by entry of average momentary pain and momentary pain, $-2LL = 7932.59, \chi^2 = 48.02, df = 2, p < .001$. The improvement was driven by momentary pain, $b = -.059, SE = .009, t = -6.36, p < .001$; average mean pain showed no association with momentary positive affect, $p > .19$. Addition of EI variables further enhanced prediction of positive affect, $-2LL = 7882.25, \chi^2 = 50.34, df = 3, p < .001$. The effect was driven by mood repair, $b = .490, SE = .097, t = 5.07, p < .001$; coefficients for attention and clarity were not significant. Pain did not interact with any of the EI variables when entered as a group, Step 4 $\chi^2 = 5.50, df = 3, n.s.$ When entered singly, marginal interactions with momentary pain emerged for

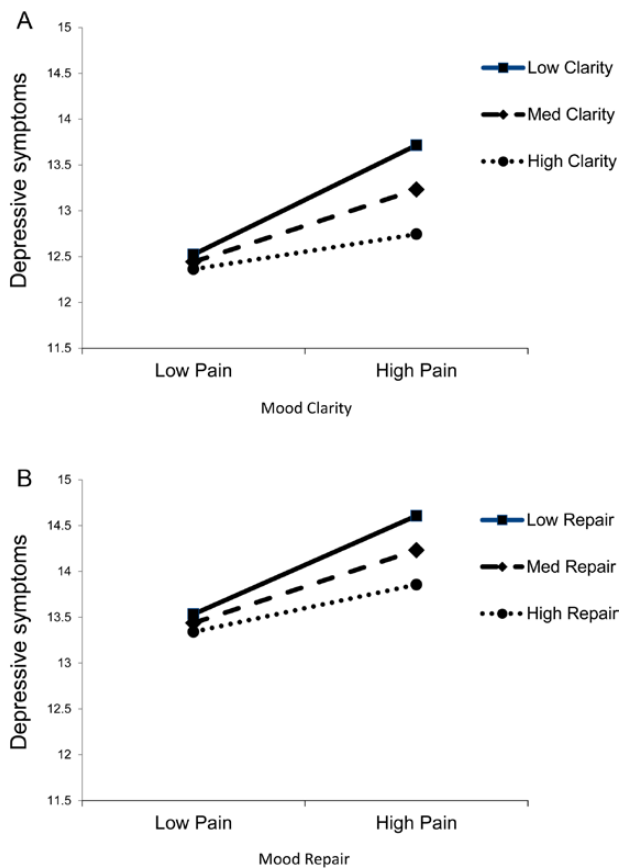


Fig. 1. Moderating effect of mood clarity (A) and mood repair (B) on the relationship of global pain to global depressive symptoms.

both mood attention, $p < .06$, and mood clarity, $p < .07$. Greater attention to mood exacerbated the association of pain with positive affect; greater mood clarity attenuated that association.

For momentary negative affect, model fit using covariates alone, $-2LL = 5238.78$, was significantly improved when contemporaneous pain and average momentary pain were added, $-2LL = 5088.86$, $\chi^2 = 149.87$, $df = 2$, $p < .001$. Both momentary pain, $b = .068$, $SE = .007$, $t = 9.52$, $p < .001$, and average momentary pain, $b = .239$, $SE = .030$, $t = 7.87$, $p < .001$, contributed to the effect. Entry of EI variables at Step 3 further improved fit, $-2LL = 5064.51$, $\chi^2 = 24.35$, $df = 3$, $p < .001$, due to a significant effect of mood repair, $b = -.159$, $SE = .056$, $t = -2.88$, $p < .004$, and a marginal contribution of mood clarity, $b = -.126$, $SE = .069$, $t = -1.84$, $p < .07$.

Interactions of momentary pain with EI variables further increased explained variance, $-2LL = 5040.26$, $\chi^2 = 24.25$, $df = 3$, $p < .001$. As the right portion of Table 3 indicates, the only significant effect was for the pain-by-mood repair interaction. Reanalysis, entering interaction terms individually to address concerns about overfitting of the model, confirmed the significant interaction of pain with mood repair, $b = -.066$, $SE = .014$, $t = -4.61$, $p < .001$, and the lack of effect for pain \times attention, $p = .692$. However, when entered alone, the pain \times mood clarity interaction emerged as statistically significant, $b = -.060$, $SE = .017$, $t = -3.51$, $p < .001$. Figure 2 portrays the weakened linkages of momentary pain with negative affect among persons higher in mood repair (Fig. 2A) and mood clarity (Fig. 2B).

Discussion

These analyses confirm that perceived EI is associated with pain at both global and momentary levels. Building on earlier work that used the same EI measure as a

Table 3 Momentary Affect as a Function of Background Characteristics, Mood Regulation Strategies, and Momentary Pain

	Positive affect					Negative affect				
	<i>b</i>	<i>SE</i>	<i>df</i>	<i>t</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>df</i>	<i>t</i>	<i>p</i>
Sex	.165	.096	215.9	1.72	.087	-.046	.055	215.6	-0.84	.403
Age	.015	.004	216.0	3.40	.001	-.002	.002	215.6	-0.82	.415
Marital status	-.088	.087	215.8	-1.02	.311	-.004	.050	215.2	0.72	.942
Education	.048	.024	216.2	1.97	.050	.014	.014	216.1	1.04	.298
Health conditions	-.026	.017	215.8	-1.52	.130	.009	.010	215.3	0.89	.375
Average momentary pain	-.033	.051	216.3	-0.64	.521	.219	.029	216.2	7.46	.001
Momentary pain	-.062	.009	4708.8	-6.57	.001	.065	.007	4709.0	9.05	.001
Mood attention	.051	.095	215.5	0.54	.588	.023	.054	214.7	0.43	.667
Mood clarity	.180	.120	215.7	1.50	.136	-.126	.069	215.1	-1.84	.067
Mood repair	.490	.097	215.8	5.07	.001	-.159	.055	215.3	-2.87	.004
Attention \times pain	-.030	.022	4708.8	-1.38	.169	.024	.017	4709.3	1.44	.151
Clarity \times pain	-.038	.030	4708.8	-1.32	.189	-.030	.022	4709.3	-1.35	.176
Repair \times pain	.010	.023	4708.8	0.44	.660	-.055	.018	4709.3	-3.08	.002

Coefficients represent last step of hierarchical multilevel modeling analysis; see text for details.

unitary construct [29, 31], we found that the most consistent EI predictor of pain was attention, reflecting higher pain both globally and in the moment among those who reported greater awareness of their moods. Mood clarity was a negative predictor of global pain and, marginally, of momentary pain. Interestingly, mood repair—the ability to maintain or regain a positive mood—was not directly related to pain in this sample of older adults with knee osteoarthritis.

Paralleling previous research [35], EI was a significant predictor of emotional well-being, in terms of both global depressive symptoms and momentary positive and negative affect. However, attention to mood was not related to well-being at either level in this group of osteoarthritis sufferers. Rather, both depression and negative affect were inversely associated with mood clarity and mood repair; for positive affect, only mood repair was significant. Thus, attention to one's moods, operationalized here as awareness and valuation of the role of mood in everyday life, is associated with heightened experience of pain; however, it does not appear to be a strong factor in emotional distress. Rather, knowing how one feels (mood clarity) and ability to maintain a positive outlook (mood repair) were the better predictors of both global and momentary affective well-being.

Emergence of a significant interaction of mood clarity with global pain on depressive symptoms corroborates the longitudinal work of Kennedy et al. [32] among persons with osteoarthritis. The current data extend this finding to within-day processes, yielding a similar buffering effect; here, however, it was mood repair, rather than clarity, that buffered the momentary linkage of pain with negative affect. This pattern suggests that whereas having a clear understanding of one's moods may help buffer against pain-fueled depression in the long term, it may be more important in day-to-day life to “accentuate the positive” in managing the emotional impact of chronic pain.

This conclusion is, of course, tempered by the fact that these patterns changed somewhat when interaction terms were examined singly rather than together. Taken in isolation, mood repair and mood clarity each displayed a significant buffering effect on the association of pain with negative mood states both globally and in the moment. Together, however, there emerged a difference in strength of association, such that mood clarity was the stronger independent predictor of depressive symptoms, versus mood repair for momentary negative affect. Although further work is obviously needed, the potentially differential roles of mood clarity and repair in long-term versus short-term pain-related distress are intriguing.

The general lack of association of EI variables with momentary positive affect in our sample is also notable. Specifically, positive affect was related only to mood

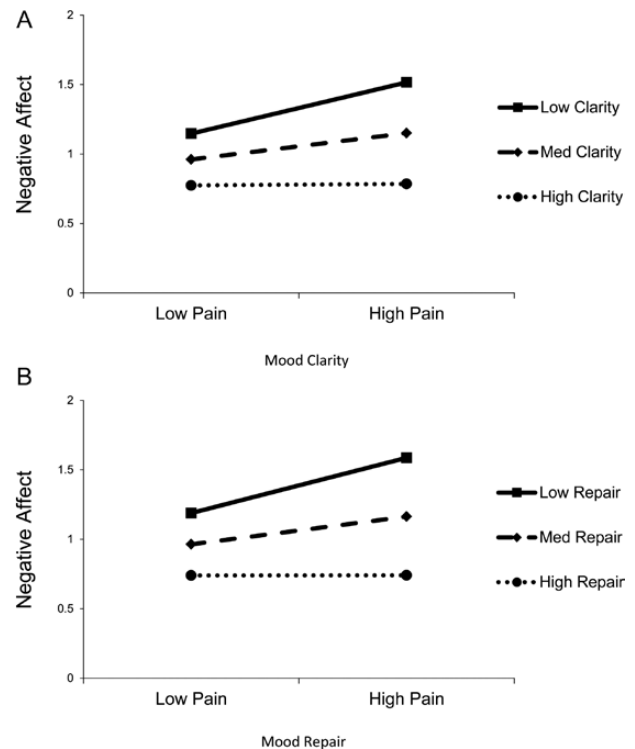


Fig. 2. Moderating effect of mood clarity (A) and mood repair (B) on the relationship of momentary pain to momentary negative affect.

repair, versus both clarity and repair for depression and negative affect. It is also perplexing that we failed to replicate the findings of Zautra's team regarding moderating effects of mood clarity [10] and repair [41] on the association of pain with positive affect. There are several plausible explanations for these differences. First, as noted by previous investigators [10, 32], it is logical that the role of mood regulation varies as a function of the particular type of pain experienced or, more generally, the type of stressor involved. Although Zautra et al.'s [10] sample included some women with osteoarthritis, the majority suffered non-age related rheumatologic disorders (rheumatoid arthritis, fibromyalgia) that may evoke differential affective and coping responses. A second consideration is time frame. Although our analysis was strictly cross-sectional, its replication of Kennedy et al.'s [32] pain-by-clarity interaction on subsequent depression confirms the role of clearly understanding one's moods for long-term emotional well-being in the presence of persistent pain. In contrast, Zautra's team worked on a different time frame with respect not only to within-person measures (weekly, vs. 4 times daily in the current study), but also to microlongitudinal time frame. Specifically, whereas we examined contemporaneous covariation of affect with pain, Zautra's team consistently looked at lagged (predictive) associations, with positive affect measured the week after pain. This raises the thought-provoking possibility that mood regulation

strategies may play differential roles in the flow of physical and emotional responses even over relatively brief time frames. This question—and, more generally, the short- versus longer-term interplay of pain, affect, and generalized well-being—is strongly deserving of further study.

A few limitations of this study bear mention. Sample and sampling frames are always a concern. We purposely used diverse sampling techniques to ensure a broad range of participant characteristics both clinically and demographically. Although all our respondents had physician-confirmed knee osteoarthritis, they varied widely in duration and severity of symptoms. This doubtlessly introduced a great deal of noise into analyses, basically trading off internal validity for generalizability. Second, although we controlled self-reported medical comorbidities, it is not unlikely that the complement of health problems, medications or other treatments used, and perhaps even clinical characteristics of the disorder, may have affected patterns of findings. Similarly, one important covariate not examined in this or others' work is activity limitation: the extent to which pain interferes with valued activities [42, 43].

An additional, broad concern is the measure of EI itself. Although both subscales and overall composite have previously been used in studies of chronic pain, few published studies have examined all three subscales simultaneously. In the current data, attention to moods was relatively independent of both clarity ($r = .331$) and repair (.169), but clarity and repair were highly correlated ($r = .597$). We found no evidence of confounding due to collinearity in current analyses, but some caution should be exercised when interpreting findings. In particular, the differential patterns of significance for interaction terms examined singly versus as a group underscores the sizeable overlap between these two constructs, and the potential importance of weighing EI constructs as “general factor” versus specific skills or perceptions. A related concern is the low reliability for the mood repair scale in this sample. Although widely used, the Trait Meta-Mood Scale was developed and validated largely on samples of college students. Further investigation of the factor structure and performance of this measure across the lifespan, particularly with chronic pain samples, is needed to inform future work.

It is also important to take into account that EI, as conceptualized and measured here, is just one of a number of cognitive and affective processes that may affect the experience of chronic pain. As noted earlier, the constructs of emotional awareness, clarity and repair overlap substantially with other constructs, for example, mindfulness, resilience, and perhaps even catastrophic thinking. It would be helpful, in future research, to examine a range of these related constructs in tandem,

in a single study, to begin more clearly to depict areas of overlap versus unique contributions to well-being of individuals coping with chronic illness.

These caveats aside, the current data provide intriguing insights into the role mood regulation, represented here as perceived EI, plays in the experience and effects of chronic pain. Our data underscore the complexity of dynamics encompassed by the construct of EI, and raise questions about its conventional construal as an inherently positive, adaptive orientation. Clearly, simple attention to mood exacerbates the experience of pain, suggesting that therapeutic techniques emphasizing emotional awareness (e.g. mindfulness) should be applied with careful emphasis on nonevaluative, accepting cognitive stances. At the same time, mood clarity—the ability to understand and differentiate emotions and their effects—may be a major aspect of long-term coping with chronic pain. In contrast, positive attitude and the ability to repair or bounce back from negative moods may be an important short-term coping mechanism. Although further research is needed, the current findings clearly demonstrate a key role of perceived mood regulation strategies in minimizing the effects of persistent pain on emotional well-being both in the moment and for the long term.

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Compliance with Ethical Standards

Ethical Approval *Research involving human participants and/or animals:* All activities performed in this research were preapproved by the Institutional Review Boards at the University of Alabama and Stony Brook University, and were performed in accordance with the ethical standards laid out by the 1964 Declaration of Helsinki and its later amendments.

Informed Consent Informed consent was obtained from all individuals participating in the study before study procedures were initiated.

References

1. Dersh J, Polatin PB, Gatchel RJ. Chronic pain and psychopathology: research findings and theoretical considerations. *Psychosom Med.* 2002;64(5):773–786.

2. Allaz AF, Cedraschi C. Emotional aspects of chronic pain. In: Pickering G, Gibson S, eds. *Pain, Emotion and Cognition: A Complex Nexus*. Geneva, Switzerland: Springer; 2015:21–34.
3. Bair MJ, Robinson RL, Katon W, Kroenke K. Depression and pain comorbidity: a literature review. *Arch Intern Med*. 2003;163(20):2433–2445.
4. Kroenke K, Wu J, Bair MJ, Krebs EE, Damush TM, Tu W. Reciprocal relationship between pain and depression: a 12-month longitudinal analysis in primary care. *J Pain*. 2011;12(9):964–973.
5. Berna C, Leknes S, Holmes EA, Edwards RR, Goodwin GM, Tracey I. Induction of depressed mood disrupts emotion regulation neurocircuitry and enhances pain unpleasantness. *Biol Psychiatry*. 2010;67(11):1083–1090.
6. Loggia ML, Mogil JS, Bushnell MC. Experimentally induced mood changes preferentially affect pain unpleasantness. *J Pain*. 2008;9(9):784–791.
7. Tang NK, Salkovskis PM, Hodges A, Wright KJ, Hanna M, Hester J. Effects of mood on pain responses and pain tolerance: an experimental study in chronic back pain patients. *Pain*. 2008;138(2):392–401.
8. Davis MC, Thummala K, Zautra AJ. Stress-related clinical pain and mood in women with chronic pain: moderating effects of depression and positive mood induction. *Ann Behav Med*. 2014;48(1):61–70.
9. Affleck G, Tennen H, Urrows S, Higgins P. Neuroticism and the pain-mood relation in rheumatoid arthritis: insights from a prospective daily study. *J Consult Clin Psychol*. 1992;60(1):119–126.
10. Zautra A, 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(5):786–795.
11. Connelly M, Keefe FJ, Affleck G, Lumley MA, Anderson T, Waters S. Effects of day-to-day affect regulation on the pain experience of patients with rheumatoid arthritis. *Pain*. 2007;131(1–2):162–170.
12. Rost S, Van Ryckeghem DM, Koval P, Sütterlin S, Vögele C, Crombez G. Affective instability in patients with chronic pain: a diary approach. *Pain*. 2016;157(8):1783–1790.
13. Smith DM, Parmelee PA. Within-day variability of fatigue and pain among African Americans and non-Hispanic Whites With Osteoarthritis of the Knee. *Arthritis Care Res (Hoboken)*. 2016;68(1):115–122.
14. Vendrig AA, Lousberg R. Within-person relationships among pain intensity, mood and physical activity in chronic pain: a naturalistic approach. *Pain*. 1997;73(1):71–76.
15. Graham-Engeland JE, Zawadzki MJ, Slavish DC, Smyth JM. Depressive symptoms and momentary mood predict momentary pain among rheumatoid arthritis patients. *Ann Behav Med*. 2016;50(1):12–23.
16. Conner TS, Tennen H, Zautra AJ, Affleck G, Armeli S, Fifield J. Coping with rheumatoid arthritis pain in daily life: within-person analyses reveal hidden vulnerability for the formerly depressed. *Pain*. 2006;126(1–3):198–209.
17. Tennen H, Affleck G, Zautra A. Depression history and coping with chronic pain: a daily process analysis. *Health Psychol*. 2006;25(3):370–379.
18. Bruehl S, Liu X, Burns JW, Chont M, Jamison RN. Associations between daily chronic pain intensity, daily anger expression, and trait anger expressiveness: an ecological momentary assessment study. *Pain*. 2012;153(12):2352–2358.
19. Veehof MM, Oskam MJ, Schreurs KM, Bohlmeijer ET. Acceptance-based interventions for the treatment of chronic pain: a systematic review and meta-analysis. *Pain*. 2011;152(3):533–542.
20. McCracken LM, Gauntlett-Gilbert J, Vowles KE. The role of mindfulness in a contextual cognitive-behavioral analysis of chronic pain-related suffering and disability. *Pain*. 2007;131(1–2):63–69.
21. Schütze R, Rees C, Preece M, Schütze M. Low mindfulness predicts pain catastrophizing in a fear-avoidance model of chronic pain. *Pain*. 2010;148(1):120–127.
22. Paquet C, Kergoat MJ, Dubé L. The role of everyday emotion regulation on pain in hospitalized elderly: insights from a prospective within-day assessment. *Pain*. 2005;115(3):355–363.
23. Connelly M, Keefe FJ, Affleck G, Lumley MA, Anderson T, Waters S. Effects of day-to-day affect regulation on the pain experience of patients with rheumatoid arthritis. *Pain*. 2007;131(1–2):162–170.
24. Connelly M, Bromberg MH, Anthony KK, Gil KM, Franks L, Schanberg LE. Emotion regulation predicts pain and functioning in children with juvenile idiopathic arthritis: an electronic diary study. *J Pediatr Psychol*. 2012;37(1):43–52.
25. Keefe FJ, Lumley M, Anderson T, Lynch T, Studts JL, Carson KL. Pain and emotion: new research directions. *J Clin Psychol*. 2001;57(4):587–607.
26. Hosoi M, Molton IR, Jensen MP, et al. Relationships among alexithymia and pain intensity, pain interference, and vitality in persons with neuromuscular disease: Considering the effect of negative affectivity. *Pain*. 2010;149(2):273–277.
27. Salovey P, Mayer JD. Emotional intelligence and its relationship to other intelligences. *Imagin Cogn Pers*. 1990;9(3):185–211.
28. Salovey P, Mayer JD, Caruso D, Yoo SH. The positive psychology of emotional intelligence. In: Cassady JC, Eissa MA, eds. *Emotional Intelligence: Perspectives on Educational and Positive Psychology*. New York: Peter Lang; 2008:185–208.
29. Ruiz-Aranda D, Salguero JM, Fernández-Berrocal P. Emotional intelligence and acute pain: the mediating effect of negative affect. *J Pain*. 2011;12(11):1190–1196.
30. Ruiz-Aranda D, Salguero JM, Fernández-Berrocal P. Emotional regulation and acute pain perception in women. *J Pain*. 2010;11(6):564–569.
31. Wright CJ, Schutte NS. The relationship between greater mindfulness and less subjective experience of chronic pain: mediating functions of pain management self-efficacy and emotional intelligence. *Aust J Psychol*. 2014;66(3):181–186.
32. Kennedy LA, Cohen TR, Panter AT, et al. Buffering against the emotional impact of pain: mood clarity reduces depressive symptoms in older adults. *J Soc Clin Psychol*. 2010;29(9):975–987.
33. 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(3):216–224.
34. Tennen H, Affleck G, Zautra A. Depression history and coping with chronic pain: a daily process analysis. *Health Psychol*. 2006;25(3):370–379.
35. Salovey P, Mayer JD, Goldman SL, Turvey C, Palfai TP. Emotional attention, clarity, and repair: exploring emotional intelligence using the trait meta-mood scale. In: Pennebaker JW, ed. *Emotion, Disclosure, and Health*. Washington, DC: American Psychological Association; 1995:125–154.
36. Pfeiffer E. A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *J Am Geriatr Soc*. 1975;23(10):433–441.
37. Parmelee PA, Katz IR, Lawton MP. The relation of pain to depression among institutionalized aged. *J Gerontol*. 1991;46(1): P15–P21.

38. Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977;1(3):385–401.
39. Lawton MP, Kleban MH, Dean J, Rajagopal D, Parmelee PA. The factorial generality of brief positive and negative affect measures. *J Gerontol.* 1992;47(4):228–237.
40. Raudenbush SW, Bryk AS. *Hierarchical Linear Models.* 2nd ed. Thousand Oaks, CA: Sage; 2002.
41. Hamilton NA, Affleck G, Tennen H, Karlson C, Luxton D, Preacher KJ, et al. Fibromyalgia: the role of sleep in affect and in negative event reactivity and recovery. *Health Psychol.* 2008;27(4):490–497.
42. Katz PP, Yelin EH. Activity loss and the onset of depressive symptoms: do some activities matter more than others? *Arthritis Rheum.* 2001;44(5):1194–1202.
43. Parmelee PA, Harralson TL, Smith LA, Schumacher HR. Necessary and discretionary activities in knee osteoarthritis: do they mediate the pain-depression relationship? *Pain Med.* 2007;8(5):449–461.
44. Hayes AF. PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper]; 2012. Available at <http://www.afhayes.com/public/process2012.pdf>. Accessibility verified April 28, 2016.