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The Effects of Anxiety and Depression on Weekly Pain in Women with Arthritis

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Abstract

This study examined the effects of anxiety and depression on pain in women with rheumatoid arthritis (RA; $n = 82$) or osteoarthritis (OA; $n = 88$). Anxiety and depression symptoms were assessed at the beginning of the study. Arthritis pain, interpersonal stress, negative affect, and positive affect were assessed weekly for 11 consecutive weeks. Multilevel analyses were conducted to investigate direct, indirect, and interactive effects of anxiety and depression on weekly changes in pain. When entered separately into the prediction equations, anxiety and depression were both related to elevations in current and next week pain, although the effects were nearly twice as large for anxiety. In addition, both anxiety and depression were indirectly related to current pain through negative and positive affect and depression interacted with stress to predict current pain in the RA group. When entered together into the prediction equations, anxiety alone was still related to elevations in current and next week pain. In addition, anxiety alone was indirectly related to current pain through negative affect and depression alone was indirectly related to current pain through positive affect. These results highlight the need for careful study of the differential effects of anxiety and depression and treatments that target their unique mechanisms.

Keywords

pain; arthritis; anxiety; depression; arthritis; women

While anxiety and depression have both been associated with the experience of chronic pain [23,24], there have been few attempts to compare their effects. This is important because anxiety and depression may affect pain in different ways and may require kinds of different interventions. For example, if anxiety plays a primary role in exacerbating pain, then treatments might focus on teaching relaxation techniques and the use of anxiolytics. If depression is primary, then treatments might focus on increasing pleasurable activities and the use of antidepressants.

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While both anxiety and depression have been examined, most studies have focused on the role of depression in chronic pain [4,7]. Depression symptoms have been frequently associated with reports of increased pain [8,12,21]. Anxiety symptoms have also been related to increased pain [9,36].

One way to understand the effects of anxiety and depression is by studying their effects on the stress process. Bolger and Zuckerman [6] have proposed a framework for understanding the effects of individual differences in relation to stress. They suggest that vulnerability factors may affect negative outcomes by increasing exposure or reactivity to stress. Using this framework, anxiety or depression may increase exposure or reactivity to stress and thereby increase reports of pain.

Another way to understand the effects of anxiety and depression is by studying their effects through emotion. Clark and Watson [10] developed a model whereby physiological hyperarousal is specific to anxiety and anhedonia is specific to depression. Since positive affect may decrease and negative affect may increase pain [25], depression may affect pain by reducing positive affect while the effects of anxiety may be more through increasing negative affect.

The purpose of this study was to examine the effects of anxiety and depression on weekly pain reports in women with arthritis. Anxiety and depression symptoms were assessed initially and pain assessed weekly for 11 weeks. In a previous paper using data from the same participants, we examined the effects of depression on stress and pain [43].¹ This current paper adds to this earlier work by comparing the effects of depression to anxiety, focusing on a measure of stress that may be less confounded with affect, and testing two distinct affective disturbances as mediators of the effects of anxiety and depression.

We predicted that both anxiety and depression, when entered separately into the prediction equations, would be related to weekly elevations in pain. In addition, we predicted that anxiety and depression would increase exposure and reactivity to interpersonal stress [6]. Finally, we expected the effects of anxiety and depression to be mediated by affect with larger effects for anxiety through negative affect and larger effects for depression through positive affect [10].

Methods

Participants

The sample consisted of 170 older women who had rheumatoid arthritis (RA; $n = 82$) or osteoarthritis (OA; $n = 88$). The study was conducted in compliance with the Institutional Review Board and informed consent was obtained by trained research assistants. Only post-menopausal women were studied to reduce heterogeneity in the hormonal profiles that underlie stress-related inflammatory processes. The participants were recruited in a variety of ways including newspaper ads, mailings to Arthritis Foundation members, and through rheumatology clinics. All participants had their diagnosis (RA or OA) confirmed by a physician and were paid a total of \$100 (\$50 after the initial interview and \$50 after the final interview).

Participants were between the ages of 42 and 76 ($M = 63.8$, $SD = 7.3$). The majority were married (57%) while fewer were divorced (24%), widowed (16%), or never married (3%). The majority were Caucasian (94%) while small proportions were Hispanic (2%), African-

¹The other papers that have been published from the weekly data collected with this sample examined the effects of personality characteristics on disease activity and negative affect [32] and compared the relationship between pain and negative affect in this sample with that found in women with fibromyalgia [44]. There is also a paper on additional immune system data that was collected from participants from this sample who had a week of high stress [45].

American (1%), or Native-American (1%). The mean income range was between \$21,000 and \$25,000. Ninety-four percent completed high school and 29% completed four years of college. Twenty-six percent were employed. There was a small but significant difference between the RA and OA respondents in age. The mean age was 62.1 years ($SD = 7.3$) for people with RA and 65.4 ($SD = 7.3$) for those with OA ($t(168) = 2.940, p = .004$). There were no significant differences between the groups on marital status, ethnic background, income, or employment.

Procedure

The participants had a telephone screening interview, were mailed an initial questionnaire, and were interviewed weekly by telephone for 12–20 consecutive weeks. Participants were in the study for 12 weeks unless they did not meet the criteria for a stressful week or arthritis flare. If they did not meet the criteria, they were continued in the study for up to 20 weeks until they did meet the criteria. Only the data from weeks 2–12 were used in the analyses for this study. The initial week was not included because positive and negative affect were not assessed. Weeks 13–20 were not included so that all participants would contribute approximately the same number of observations. This was important because the week number in the study was related to less pain, interpersonal stress, and negative affect and we did not want participants lower on these variables to be overrepresented in the analyses.

Measures

Anxiety and depression were assessed in an initial questionnaire. Functional disability was assessed during an initial telephone screening interview and arthritis pain, negative affect, positive affect, and interpersonal stress were assessed weekly during phone interviews. The following measures were used for the study.

1. Baseline Measures

Anxiety and Depression Symptoms—Anxiety and depression symptoms were assessed using 19 items from the Mental Health Inventory [37]. The second author of the paper has conducted extensive factor analytic work on the Mental Health Inventory with community samples of older adults [41]. That work supported the distinction made here between anxiety and depression as separate yet correlated factors contributing to mental health and provided guidance in the selection of items from the parent scale that best represented the constructs. There were ten anxiety items such as “how much of the time have you been a nervous person?” and nine depression items such as “how much of the time have you felt downhearted and blue?” Participants were asked to respond to each of these items with regard to the previous week. The responses were scored on a six-point scale for all items except for the last item on each subscale which was scored on a five-point scale. Cronbach's alpha was .93 for anxiety and .91 for depression.

Functional Disability—The Health Assessment Questionnaire was used to measure the degree to which participants experienced arthritis-related activity limitation [15]. Participants were asked to respond to each of the items in relation to the previous week. There were 20 items assessing the amount of difficulty the participant experiences when performing daily tasks. Items were rated on a four-point scale.

2. Prospective Measures

Arthritis Pain—Arthritis pain was assessed each week during the telephone interviews. There were three items including: (1) “please choose a number between 0 and 100 that best describes the average level of pain you have experienced over the past week due to your arthritis,” (2) “please choose a number between 0 and 100 that best describes your current level of arthritis pain,” and (3) “please choose a number between 0 and 100 that best describes the worse level

of pain over the past week.” For each of these items, 0 = “no pain” and 100 = “pain as bad as it could be.” Cronbach’s alpha for the three items was .95.

Interpersonal Stress—Interpersonal stress was assessed using the negative interpersonal events scale of the Inventory of Small Life Events [40]. All of the items on this list were written to have a discrete beginning and end. They refer to observable changes in behavior in everyday activities rather than internal states. Participants were asked to report the number of times each event occurred during the past week. Examples of the negative events sampled include arguments, disagreements, and being criticized by others. We obtained the overall score by taking the mean score of the total number of negative events during the previous week for each category in which the participant had relationships. The four categories were “significant other,” “family members,” “friends,” and “coworkers.” If participants did not have relationships in all four categories, then the mean score for the items in the relevant categories was used. For example, if the participant was not employed but had a significant other, family members, and friends, then only the mean for the significant other, family member, and friend items were averaged to compute the total mean score. Finally, the negative events scores were square root transformed to reduce the skewness and kurtosis that is common with events measures.

Positive and Negative Affect—Positive and negative affect were measured weekly during the telephone interviews using the Positive and Negative Affect Schedule (PANAS) [39]. There are 10 items assessing positive affective states (e.g., “enthusiastic,” “proud,” “inspired”) and 10 items assessing negative affective states (e.g., “upset,” “hostile,” “afraid”). Participants indicated the extent to which they had experienced each affective state during the previous week. Responses were rated on a five-point scale where 1 = “very slightly or not at all” and 5 = “extremely.” Cronbach’s alpha was .90 for positive affect and .88 for negative affect.

Data Analysis Methods

Multilevel modeling was used to analyze the weekly data and test our predictions. This method is useful for the analysis of data that have a nested hierarchical structure. The weekly data take a hierarchical form, with up to 11 weekly observations nested within each of the 170 participants. There were 1758 total observations (1870 possible – 112 missing). The SPSS 14.0 Mixed program was used for the multilevel analyses and the specifications of the models were based on the guidelines provided by Singer [31] and Peugh and Enders [28].

The weekly measures of stress, affect, and pain were the criterion variables to be predicted in the analyses. There were two basic types of prediction equations in the multilevel analyses: a Level 1 equation, which examined the influence of within-person variations on pain, and a Level 2 equation, which tested the effects of anxiety and depression on stress, affect, and pain. In essence, the Level 2 equations address questions regarding between-person differences and take the following form: Do people who have different scores on a between-persons predictor (e.g., anxiety) have different levels on the criterion (e.g., arthritis pain)?

Level 1 questions address the issue of “when” rather than “who”: for example, “When a person has more interpersonal stress, do they also report more arthritis pain?” The Level 1 question examined within-person variation in interpersonal stress, negative affect, positive affect, and pain. To prepare for these analyses, weekly deviation scores on the events and affect measures were computed by subtracting each participant’s average score on those variables across the 11 weeks from her weekly report on each variable [20]. The equation was initially specified at Level 1 as follows:

$$\text{weekly pain} = \beta_0 + \beta_1 \text{interpersonal stress} + \beta_2 \text{negative affect} + \beta_3 \text{positive affect} + r$$

Level 1

β_0 yields an estimate of the average weekly pain, and β_1 , β_2 , β_3 provide slope estimates for interpersonal stress, negative affect, and positive affect, respectively. In models predicting pain during the next week, current pain was included as an additional Level 1 control variable.

Individual differences in the average level of the weekly variables were also probed through analyses at Level 2. For these analyses, we focused on individual differences in anxiety and depression. These variables were used as predictors of variance in the Level 1 pain, interpersonal stress, negative affect, and positive affect (the Level 1 intercept: β_0). The initial Level 2 equation for this model was as follows:

$$\beta_0 = \gamma_{00} + \gamma_{01} \text{anxiety} + \gamma_{02} \text{depression} + \mu_0 \quad \text{Level 2}$$

Cross level interactions were also probed by examining the effect of anxiety and depression on the relationship between the interpersonal stress, negative affect, and positive affect predicting each dependent variable. The Level 2 equation for modeling these interactions was as follows:

$$\beta_1 \text{interpersonal stress} = \gamma_{10} + \gamma_{11} \text{anxiety} + \gamma_{12} \text{depression} + \mu_1 \quad \text{Level 2}$$

In this equation, the slope (β_1) designates the estimated relationship between interpersonal stress and the dependent variable for each participant. This slope is predicted by individual differences anxiety (γ_{11}) and depression (γ_{12}). This provides a test of the interaction between anxiety and depression and Level 1 variables (e.g., interpersonal stress, negative affect, positive affect) on the dependent variables.

The other specifications for the multilevel model were selected following Singer [31] to identify the best fitting model of the variables under study. The intercept and weekly variables that initially showed significant random effects were kept in the model and modeled using an unstructured covariance matrix.

Finally, effects sizes were computed for all of the predictors in our hypotheses. According to guidelines outlined in Singer [31], effect sizes in multilevel modeling were derived with the variance-covariance parameter estimates. By using these estimates for the intercept, we determined the proportion of explainable between-subjects variance accounted for when anxiety or depression was added to the model.

Results

Table 1 displays means, standard deviations, and t tests of the differences for key variables for the two groups: OA and RA participants. The OA group had less functional disability and more interpersonal stress than the RA group. The rest of the results are presented for the combined groups and differences in the effects between the groups are noted. The week number in the study, functional disability, and income were controlled in the multilevel analyses because they were related to weekly pain.

The between-subjects correlations were calculated for anxiety, depression, and functional disability. The correlation between anxiety and depression was $r = .696$ ($p < .01$) and there was no significant correlation between functional disability and anxiety or depression. Because the correlation between anxiety and depression was strong, we combined the groups and examined collinearity in multiple regression analyses predicting pain from anxiety and depression. The variance inflation factor (VIF) for anxiety and depression was 1.948, suggesting that collinearity was not a problem.

We also examined the within-subjects correlations between the variables measured each week. Pain was positively related to negative affect ($r = .244, p < .01$) and negatively related to positive affect ($r = -.201, p < .01$) in both groups. Also, negative affect was positively related to interpersonal stress ($r = .324, p < .01$) and negatively related to positive affect ($r = -.209, p < .01$). The only difference between the groups was that pain was related to interpersonal stress in the OA group ($r = .112, p < .01$) but not in the RA group. Interpersonal stress was not related to positive affect.

Direct Effects on Pain

We used multilevel level analyses to determine whether anxiety, depression, interpersonal stress, and negative and positive affect predicted weekly changes in pain. Table 2 displays the results of these analyses when each variable was entered separately into the prediction equation. Anxiety, depression, negative affect, and interpersonal stress were all related to increases in pain. Positive affect was related to decreases in pain. The largest effect was for anxiety with a 10.13 point increase in pain for every standard deviation increase in anxiety accounting for 14.51% of the explainable between subject variance in pain. The effect for depression was about half the size with a 5.41 point increase in pain accounting 4.15% of the variance.

Next, we entered all of the predictors simultaneously in the multilevel equation to determine their relative importance in predicting pain. The results of these analyses are displayed in Table 3. Anxiety and negative affect were still significant predictors of elevations in weekly pain and positive affect was still a significant predictor of decreases in pain. Anxiety still had the largest effect on pain and the effects for depression and interpersonal stress were no longer significant. Anxiety accounted for an additional 10.86% of the variance in pain when added to the model with all of the other variables.

Finally, we wanted to determine whether anxiety, depression, and the other key variables predicted pain during the next week when controlling for current pain. When entered separately, anxiety and depression were the only variables that were related to next week's pain. The effect for anxiety was again nearly twice the size of that for depression ($B = 9.9584, t = 5.146, p = .000$ and $B = 5.2233, t = 2.650, p = .009$, respectively). Anxiety accounted for 13.73% and depression accounted for 3.60% of the variance in pain. When anxiety and depression were entered into the equation together, only anxiety was still related to elevations in next week pain ($B = 11.7247, t = 4.429, p = .000$) accounting for 10.49% of the variance when added to the model.

Pathways through Stress

Next, we wanted to determine whether the effects of anxiety and depression increased exposure or reactivity to interpersonal stress. When entered separately, anxiety alone was related to weekly elevations in interpersonal stress ($B = .2465, t = 3.140, p = .002$ and $B = .0697, t = 2.246, p = .026$, respectively) accounting for 5.44% of the variance in interpersonal stress. However, although anxiety did increase exposure to stress, there was no indirect effect of anxiety on pain through stress because stress was not related to pain in the full model (see Table 3).

To examine reactivity to stress, we tested whether anxiety and depression interacted with interpersonal stress to predict weekly changes in pain. When entered separately, neither anxiety nor depression each interacted with interpersonal stress to predict more pain. However, depression did interact with interpersonal stress to predict more pain in the RA group ($B = 2.3044, t = 2.364, p = .018$). Participants high in depression had more pain when interpersonal stress was high vs. low. Those low in depression had less pain when interpersonal stress was high vs. low.

Pathways through Emotion

Next, we wanted to determine whether there were indirect effects of anxiety and depression on pain through negative affect and positive affect. When entered separately, both anxiety and depression were related to weekly elevations in negative affect ($B = .2300$, $t = 6.624$, $p = .000$ and $B = .1691$, $t = 4.842$, $p = .000$, respectively). Anxiety accounted for 22.77% and depression accounted for 12.32% of the variance in negative affect. When entered together, only anxiety significantly predicted negative affect ($B = .2008$, $t = 4.344$, $p = .000$) accounting for 11.65% of the variance when added to the model with depression.

Because negative affect was related to more pain in the full model, we calculated the indirect effects of anxiety and depression on pain through negative affect. When entered separately, there was an indirect effect of anxiety on pain through negative affect ($B = 1.6266$, $Z = 4.571$, $p = .000$) [34] and for depression on pain through negative affect ($B = 1.1959$, $Z = 3.844$, $p = .000$). When entered together, there was still a significant indirect effect of anxiety on pain through negative affect ($B = 1.4200$, $Z = 3.580$, $p = .000$) but no indirect effect for depression.

Next, we examined the pathways to pain through positive affect. When entered separately, both anxiety and depression were related to weekly decreases in positive affect ($B = -.2492$, $t = -4.234$, $p = .000$ and $B = -.3098$, $t = -5.755$, $p = .000$, respectively). Anxiety accounted for 9.23% and depression accounted for 16.47% of the variance in positive affect. When entered together, only depression was a significant predictor of positive affect ($B = -.2809$, $t = -3.740$, $p = .000$) accounting for 7.54% of the variance when added to the model with anxiety.

Because positive affect was related to less pain in the full model, we calculated the indirect effects of anxiety and depression on pain through positive affect. When entered separately, there was an indirect effect of anxiety on pain through positive affect ($B = 1.0048$, $Z = 3.030$, $p = .002$) and for depression on pain through positive affect ($B = 1.2491$, $Z = 3.467$, $p = .001$). When entered together, there was a significant indirect effect of depression on pain through positive affect ($B = 1.1326$, $Z = 2.834$, $p = .005$) but no indirect effect for anxiety.

Finally, to determine whether anxiety or depression influenced reactivity to negative or positive affect, we examined the anxiety x negative affect, anxiety x positive affect, depression x negative affect, and depression x positive affect interactions predicting pain. None of these interactions were significant predictors of pain.

To summarize, when entered separately, both anxiety and depression had direct effects on current and next week pain and indirect effects on current pain through both positive and negative affect. In addition, depression interacted with stress predicting current pain in the RA group. When entered together, anxiety alone was directly related to current and next week pain and indirectly related to current pain through negative affect while depression alone was indirectly related to current pain through positive affect. Anxiety alone was also related to more interpersonal stress.

Discussion

This study examined the effects of anxiety and depression on weekly changes in pain in women with arthritis. We considered the effects of anxiety and depression both separately and together and examined them in light of established models of stress [6] and emotion [10]. When entered separately, anxiety and depression appeared to have similar effects. As predicted, both were directly related to elevations in weekly pain and indirectly related to pain through negative and positive affect. In addition, depression interacted with interpersonal stress to predict more pain in the RA group. Although anxiety increased exposure to stress, there was no indirect effect on pain through stress because stress did not predict pain in the full model.

However, important differences between anxiety and depression became evident when comparing the sizes of their effects and when entering them together into the prediction equations. Most striking, the direct effect of anxiety was nearly twice as large as depression and the effect of depression was reduced to non-significance when anxiety was controlled. In addition, the effect for anxiety was just as strong in predicting next week pain when controlling for same week pain. Moreover, the fact that interpersonal stress did not predict next week pain suggests that anxiety may be a more important prospective predictor of pain than interpersonal stress. Also, as predicted, the indirect effect of anxiety on pain was largest through negative affect while the indirect effect of depression on pain was largest through positive affect.

When anxiety and depression were entered together, only anxiety had a direct effect on pain and an indirect effect on pain through negative affect and only depression had an indirect effect through positive affect. Even though most previous studies have focused on depression rather than anxiety, this study showed how important it is to consider them together. While we examined the effects of depression in the same sample [43], this paper demonstrates that the effects of anxiety were greater than those of depression and that the effects of each were differentially mediated through negative and positive affect, respectively. It also shows that while anxiety increased exposure to interpersonal stress, it did not increase reactivity.

To distinguish the unique contributions of anxiety and depression to the experience of pain, the tripartite model of Clark and Watson [10] may be particularly useful. As noted above, their model proposes that physiological arousal may be specific to anxiety and that anhedonia may be specific to depression. This suggests that the presence of arousal (rather than the absence of pleasure) may have played a role in the direct effects that we found for anxiety on negative affect, stress, and pain. Arousal involves a sympathetic nervous system response that sensitizes the organism to danger, heightens attention to pain, and increases the likelihood of misinterpreting innocuous bodily sensations as painful. Attention to pain has been identified as a possible link between anxiety and pain [18]. Interpretive biases have been shown to link anxiety sensitivity and pain [19]. An anxious person may be more likely to pay attention to painful bodily sensations and interpret them as harmful.

The tripartite model also suggests that the absence of pleasure (rather than the presence of arousal) may have played a role in the greater vulnerability experienced by depressed persons in times of stress. The ability to experience pleasure in stressful times may be a buffer that reduces the perception of pain. Alternatively, the accumulation of pleasurable experiences over time may facilitate the building of resources for coping with stress [14]. Indeed, positive affect has been found to be a source of resilience for women with chronic pain [42].

Interestingly, we found in the RA group that those low in depression had less pain in times of stress. Although group difference needs to be replicated, the RA group may have been more vulnerable to stress because of a number of factors including greater disability, the involvement of more joints, or greater uncertainty about the course of their illness. For the RA group, it may be that those who were not depressed were able to cope with stressful situations in a way that distracted them from their pain. For example, the ability to experience pleasure may have enhanced the ability to use humor to cope resulting in reduced perceptions of pain.

While the tripartite model has provided important clues to the differences between anxiety and depression, the optimal way to clarify their unique effects may be a careful delineation of the mechanisms. The effects of anxiety on pain have been linked to anxiety sensitivity [30], pain-relevant anxiety [2], and fear-avoidance which can result in deconditioning [3]. In addition, neuroimaging studies have suggested that self-focused attention or priming for the worst outcome may help explain the link between anxiety and pain [26,29].

Depression has been associated with a variety of processes that may increase pain including catastrophizing [16], less positive reinforcement [22], and helplessness [33]. More recently, serotonergic and noradrenergic pathways have been identified which may increase vulnerability to painful episodes in those who are depressed [11,35]. Despite these initial findings, there is much work to be done in refining and integrating these models of the influence of anxiety and depression on pain.

While highlighting the differential effects of anxiety and depression is important, the strongest implications of this study may be for the treatment of those who suffer with chronic pain. First, our findings have implications for what to target in interventions. Interventions designed to directly target anxiety or depression may both be helpful in reducing pain [5,17]. In addition, simple cognitive and behavioral interventions, such as relaxation techniques and pleasant events scheduling, may also be useful in heightening positive or reducing negative affect, which appear to mediate the effects of anxiety and depression [13]. Finally, teaching new coping skills and the ability to better match them to specific stressors may reduce the chance that stress will increase the suffering of those who are anxious or depressed [27].

Most important, if the direct effects of anxiety on pain are consistently found to be larger than that of depression, then interventions that target anxiety may be the most effective for those who suffer from both anxiety and depression. These interventions may have the added benefit of reducing the stress and negative affect that add to the malaise of chronic pain conditions. In addition, individual patients could be assessed on the three dimensions that constitute the tripartite model and treatments could target the most elevated dimension. Watson and Clark [38] have developed the Mood and Anxiety Questionnaire (MASQ) that makes it possible to reliably assess them. If the arousal dimension is highest, then the focus could be on biofeedback and simple relaxation techniques. If the anhedonia dimension is highest, then the focus could be more increasing the appreciation of and number of pleasurable events and interactions.

Finally, it is important to note the limitations of this study. First, the sample was all women, heavily Caucasian, fairly well educated, and may not represent people of other demographic characteristics. Thus, care must be exercised in generalizing the findings to men, those who have types of arthritis with a different etiology or pathogenesis, and those with other kinds of chronic pain conditions. Second, we assessed anxiety and depression symptoms one time at the beginning of the study. However, while symptoms may fluctuate over time, we only included data collected within three months of the initial assessment which should have minimized any changes.

Third, we assessed anxiety and depression symptoms rather than the diagnostic categories of anxiety disorders and major depression (DSM-IV-TR) [1]. However, anxiety disorders include disparate diagnoses like panic disorder, obsessive-compulsive disorder, and posttraumatic stress disorder. Using a measure of anxiety symptoms may better capture the construct of anxiety than combining a heterogeneous group of diagnoses. In addition, using continuous measures of anxiety and depression symptoms makes it possible to assess the full range of the experience of anxiety and depression, including the milder forms that may afflict many people with chronic pain.

Conclusions

In sum, this study identified important differences between the effects of anxiety and depression on weekly fluctuations of pain in women with arthritis. Anxiety had stronger direct effects on pain while indirect effects were mediated primarily through negative affect. In contrast, depression had no direct effect on pain, indirect effects through lower positive affect, and increased pain reports during stressful weeks in the RA group. These results point to the

importance of careful study of both anxiety and depression and treatments that more specifically target their unique mechanisms.

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Table 1
Scores on Key Variables for Participants with Osteoarthritis and Rheumatoid Arthritis

Variable	Osteoarthritis		Rheumatoid Arthritis		t	p
	M	SD	M	SD		
Baseline Variables						
Anxiety	2.49	0.84	2.36	0.79	1.109	.269
Depression	2.20	0.86	2.07	0.85	0.942	.348
Functional Disability	2.23	0.53	2.49	0.55	-3.166	.002
Prospective Variables ^a						
Arthritis Pain	45.68	23.09	45.32	22.10	0.101	.920
Interpersonal Stress	1.24	1.24	1.03	1.01	4.030	.000
Negative Affect	1.59	0.38	1.55	0.45	0.756	.451
Positive Affect	3.15	0.64	3.06	0.67	0.906	.366

Note. df = 168.

^aThe overall mean for each of the within subjects variables was computed.

Table 2
Multilevel Analyses with Univariate Predictors in Weekly Changes in Arthritis Pain

	Estimate	SE	df	t	p
Anxiety	10.1322	1.9047	166.32	5.320	.000
Depression	5.4093	1.9440	166.27	2.783	.006
Interpersonal Stress	1.1818	.4015	1586.33	2.944	.003
Negative Affect	8.1614	1.0867	141.73	7.511	.000
Positive Affect	-5.5872	.9737	134.49	-5.738	.000

Note. Negative affect, positive affect, and interpersonal stress were included as random effects in their respective equations. Week number in the study, functional disability, and income level were controlled in these analyses.

Table 3
Multilevel Analyses of the Full Model of Predictors in Weekly Changes in Arthritis Pain

	Estimate	SE	df	t	p
Anxiety	11.6024	2.6023	165.32	4.458	.000
Depression	-2.2091	2.5117	165.28	-0.880	.380
Interpersonal Stress	.1700	.3893	1535.18	0.437	.662
Negative Affect	7.0788	1.0978	143.67	6.448	.000
Positive Affect	-4.1062	.9225	133.01	-4.451	.000

Note. Negative affect and positive affect were included as random effects variables in the analysis. Week number in the study, functional disability, and income level were controlled.