Pain, Negative Mood, and Perceived Support in Chronic Pain Patients: A Daily Diary Study of People With Reflex Sympathetic Dystrophy Syndrome

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Chronic pain patients show substantial psychological distress, including depressed mood, anxiety, and anger. Nevertheless, the causal role of negative mood in the course of chronic pain conditions remains unclear. This study prospectively investigated the relationship between daily pain, negative mood, and social support in 109 people with reflex sympathetic dystrophy syndrome. Participants completed 28 daily diaries that included questions about pain, mood, and perceived support. Time-lagged within-subject analyses indicated that pain led to increases in depressed, anxious, and angry mood. Depressed mood, but not anxiety or anger, contributed to increases in pain. Perceived support had both main and buffering (interaction) effects on negative mood and a main effect on pain.

Chronic pain patients show higher than average depression (for reviews, see Banks & Kerns, 1996; Gupta, 1986; Kerns, Rosenberg, & Jacob, 1994; Linton & Gotestam, 1985; Magni, 1987; Romano & Turner, 1985; Roy, Thomas, & Matas, 1984; Smith, 1992; Sullivan, Reesor, Mikail, & Fisher, 1992). This observation holds whether the focus is on clinical diagnosis, depressive symptomatology, or depressed mood. Furthermore, those chronic pain patients in greatest pain report the highest levels of depression. However, among pain patients, those with the highest level of perceived social support are least distressed (Jamison & Virts, 1990; Mann & Zautra, 1989).

These observations raise questions about the causal nature and specificity of the relationships among pain, depression, and social support. What role does depression play in the development and maintenance of chronic pain conditions? Conversely, what role does pain play in generating and maintaining depression? Does the pain–depression relationship reflect a general relationship between pain and emotional distress, or do the processes linking pain and depression differ from those linking pain and other common forms of distress, such as anxiety and anger? Does social support play a causal role in reducing the negative impact of chronic pain conditions? The present investigation was undertaken to address these questions.

Prior efforts to answer these questions have been limited by the cross-sectional nature of studies of the associations among pain, the three dimensions of distress (depression, anger, and anxiety), and social support. Although there have been a number of recent prospective studies of the pain–depression relationship, these studies have not yielded unambiguous answers. Whereas some of these studies have found that pain predicted subsequent increases in depression, others have found that depression or distress predicted subsequent increases in pain. For example, the pain experienced by rheumatoid arthritis patients predicted increased depressive symptomatology over 6- (Brown, 1990) and 24-month periods (Nicassio & Wallston, 1992), but their depressive symptomatology did not predict increased pain (Brown, 1990). In myofacial pain patients, distress (a composite of anxiety, somatization, and depressive symptoms) contributed to increased pain over a 1-month period, but pain did not predict increased distress (Zautra et al., 1995). Similarly, depressive symptoms led to increased musculoskeletal pain over a 5-year period (Leino & Magni, 1993) and predicted the development of chronic pain in patients with acute herpes zoster (Dworkin, Hartstein, & Rosner, 1992).

Taken together, these prospective studies indicate some role for pain in the development of depressive symptomatology and for depressive symptoms in the course of chronic pain. Zautra et al.
(1995) suggested that an analysis of the transaction of pain and depressive symptoms over shorter periods of time could further refine our understanding of their relationship. Daily diary studies appear to be well suited to this task (see Larson & Almeida, 1999). This approach is particularly appropriate for investigating the processes linking perceived pain with depressed mood. An understanding of the pain–mood link potentially can help explain the co-occurrence of pain syndromes and global depressive symptomatology or clinical depression. By obtaining reports of mood, pain, and other relevant variables over consecutive days, the diary methodology makes it possible to examine the covariation of pain and depressed mood, as well as other dimensions of negative mood, such as anger and anxiety, in the same person over time. In addition, this type of within-subject design controls for between-subjects factors, such as neuroticism, that may account for observed between-subjects associations between pain and distress. The use of this approach to assessing the pain–mood relation has a precedent in Affleck, Tennen, Urrows, and Higgins’s (1992) study showing that pain and negative mood covary within day in rheumatoid arthritis patients.

Similar to Affleck et al.’s (1992) study, the present study used a daily diary approach to examine the pain–distress relationship in chronic pain patients. However, our study extended the Affleck et al. study in making use of the longitudinal nature of the diary data to examine prospective associations between pain and mood. Our study also capitalized on the longitudinal nature of the data to examine the specificity of the pain–depressed mood relationship and the impact of social support on this relationship.

Specificity of the Pain–Depressed Mood Relationship

Although most prior research has focused on the relationship between chronic pain severity and depressive symptomatology, there is evidence that chronic pain severity is also associated with anxiety and anger (Ackerman & Stevens, 1989; Gaskin, Greene, Robinson, & Geisser, 1992; Kerns, Finn, & Haythornthwaite, 1988; Linton & Goteastam, 1985; Summers, Rapoff, Varghese, Porter, & Palmer, 1991). Moreover, among chronic pain patients, those reporting depressive symptoms also report pain-related anxiety and anger (Wade, Price, Hamer, Schwartz, & Hart, 1990).

The link between pain and anger, especially suppressed anger, has long been of interest (e.g., Engle, 1959). There is some evidence that chronic pain patients may suppress anger more than other people (Braha & Catchlove, 1986; Pilowsky & Spence, 1976). Those chronic pain patients in greater pain report feeling more angry (Gaskin et al., 1992; Summers et al., 1991) but have greater difficulty expressing these feelings (Franz, Paul, Bautz, Choroba, & Hildebrandt, 1986; Kerns et al., 1994). It has been suggested that suppressed anger may contribute to pain development by deactivating the production of endogenous opioids (Beutler, Engle, Oró–Beutler, Daldrup, & Meredith, 1986). However, it is also possible that pain leads to anger suppression. For example, pain patients may be less likely than the typical person to express anger toward others because they fear compromising their relationships with the people on whom they depend or because they tend to attribute their anger to their pain rather than to the negative behavior of others (Downey, Purdie, & Schaffer-Neitz, 1999).

Most of the evidence implicating anxiety in pain perception comes from laboratory studies of acute-pain situations. These studies show that induced pain-related anxiety intensifies pain perception, whereas induced anxiety that is unrelated to pain reduces responsivity to pain (al Absi & Rokke, 1991; Arntz & De Jong, 1993; Arntz, Dreessen, & De Jong, 1994; Arntz, Dreessen, & Merckelbach, 1991; Bobey & Davidson, 1970; Weisenberg, Aviram, Wolf, & Raphaeli, 1984). Although the role of anxiety in chronic pain is less clear, there is evidence that patients with severe chronic pain experience more generalized anxiety (Ackerman & Stevens, 1989; Gaskin et al., 1992; Kerns et al., 1988; Linton & Goteastam, 1985) and that patients who experience pain-related anxiety report more pronounced pain, pain-related disability, and avoidance of pain-inducing situations (McCracken, Gross, Aikens, & Camrile, 1996; McCracken, Zayfert, & Gross, 1992).

These cross-sectional studies suggest that pain severity is linked with anger and anxiety, as well as with depressed mood. However, in the absence of prospective longitudinal evidence, it is uncertain whether these observed associations simply reflect an effect of pain on generalized negativity rather than specific contributions of these forms of negative mood to pain. To help reduce this uncertainty, the present study examined the cross-day associations between pain and these different types of negative mood as well as a general measure of negative mood.

Implications of Perceived Support for the Mood–Pain Relationship

Perceived social support has been identified as a potentially important influence on the relationship between pain and distress. Support may operate by directly affecting pain or mood (main-effects model) or by reducing (i.e., buffering) their impact on one another (Cohen & Wills, 1985). Both of these types of effects have been examined in cross-sectional studies.

Consistent with the main-effects model, those chronic pain patients who report high levels of perceived support and marital satisfaction experience less distress (Jamison & Virts, 1990; Manne & Zautra, 1989) and less severe pain (Brown, Wallston, & Nicassio, 1989; Jamison & Virts, 1990; Saartvjarvi, Rytkoski, & Karppi, 1990). However, support is not always associated with reduced distress and pain. Support in the form of spousal attentiveness and solicitousness to patient behavior is associated with heightened pain severity and overt pain behavior, perhaps because this form of support may unintentionally reinforce pain (e.g., Kerns, Haythornthwaite, Southwick, & Giller, 1990; Romano et al., 1992; for a review, see Turk, Kerns, & Rosenberg, 1992).

Cross-sectional data also provide evidence that support buffers the pain–distress association. Affleck, Pfeiffer, Tennen, and Field (1988) found that the positive association between social support satisfaction and physician-rated psychosocial adjustment was strongest for those arthritis patients with the greatest functional disability. Goldberg, Kerns, and Rosenberg (1993) also found weaker associations between depressive symptomatology and pain-related interference and activity reduction in chronic pain patients who reported high pain-related support from spouses. Finally, Brown et al. (1989) found that satisfaction with emotional support buffered the association between pain intensity and depressive symptomatology in arthritis patients. However, Brown et
al. failed to replicate this buffering effect in a longitudinal study. Rather, consistent with a main-effects model, satisfaction with support predicted a reduction in depressive symptomatology over 6-month periods, irrespective of pain intensity.

The cross-sectional nature of most of the research precludes differentiating among competing explanations for observed associations of support with distress and pain. Thus, concurrent negative associations between support and depressive symptomatology in chronic pain patients may indicate either that support protects against depression or that depression drives away support (Bolger, Foster, Vinokur, & Ng, 1996; Coyne, Burchill, & Stiles, 1991). Similarly, positive associations of pain and pain behavior with spousal solicitousness may indicate the reinforcing effect of such responses on pain (Fordyce, 1976, 1988). Alternatively, pain and pain behavior may evoke solicitous behavior.

The existing literature offers little guidance concerning the impact of discrete supportive interchanges, as opposed to global perceptions of support quality or type, on distress and pain. A goal of this study was to investigate whether interchanges perceived as supportive influence daily mood and pain in persons with chronic pain. We prospectively tested both main-effect and buffering models of the influence of perceived support on mood and pain.

Overview of the Present Study

This daily diary study of the relationships among pain, negative mood, and perceived social support was conducted with a community sample of people diagnosed with reflex sympathetic dystrophy syndrome (RSDS). Pain is the most troubling and debilitating symptom of this rare, unpredictable complication of soft-tissue trauma or illness (Schwartzman, 1993). The disorder is characterized by persistent burning pain, lowered pain threshold, and pain induced by nonnoxious stimuli, such as minor friction. Other symptoms include vasomotor disturbances (e.g., peripheral vasoconstriction evident in cold skin), edema, muscle weakness or atrophy, and trophic changes (e.g., osteoporosis).

Although the cause of RSDS is unclear, the disorder is believed to involve some form of sympathetic disregulation (but see Schott, 1994) and different pathophysiological mechanisms may underlie different forms of the disorder (Chard, 1991). Whereas sympathetic blockade may reverse the condition in the early stages, in the latter stages such treatments are considerably less effective. Presumably, this reflects a progressive sympathetic independence of the pain with the natural course of the condition. It may also reflect the contribution of psychological factors, such as maladaptive coping, to the course of the disorder (Chard, 1991; Eggle & Hoffmann, 1990; Poplawski, Wiley, & Murray, 1983; Sherry & Weissman, 1988; Van Houdenhove, 1986). Although the average pain intensity associated with RSDS is considerably more severe than that associated with most other chronic pain conditions (DeGood, Cundiff, Adams, & Shutty, 1993; Melzack & Katz, 1992), RSDS patients experience large day-to-day fluctuations in pain intensity. Thus, RSDS is particularly suited for daily diary studies of the causes and consequences of pain intensity in chronic pain patients.

The main questions addressed in this study were as follows: First, what is the nature of the relationship between pain and negative mood? Specifically, we examined (a) whether pain on the previous day predicted increased negative mood on the present day and (b) whether negative mood on the previous day predicted increased pain on the present day.

Second, is there a general relationship between pain and negative mood, or do the processes linking pain with depressed mood, anger, and anxiety differ? Specifically, we assessed the prospective relationship between pain and mood separately for overall negative mood, depressed mood, anger, and anxiety.

Third, what is the role of perceived support in the pain-negative mood relationship? Specifically, we examined (a) whether social support on the previous day predicted decreased negative mood on the present day and (b) whether social support on the previous day predicted reduced pain intensity on the present day. We also tested whether support buffered the effect of pain on negative mood and the effect of negative mood on pain. In addition to testing these models for overall negative mood, we also investigated whether support had a distinctive effect on the links (if found) between pain and depressed mood, anxiety, and anger.

Method

Participants

Participants were 109 currently married adults with RSDS recruited nationwide through advertisements posted in the RSDS Association newsletter and through announcements made at RSDS support group meetings. Participants were predominantly middle to lower middle class and were Caucasian, with 1 exception (a Native American). Their mean age was 39.8 years (SD = 8.84 years), and 89% were female, reflecting the greater prevalence of RSDS in women than in men (Chard, 1991). On average, participants had been diagnosed with RSDS 5 to 6 years prior to participating in the study (M = 5.62 years, SD = 4.58 years). Many experienced substantial disability: 39% reported the loss of use of at least one arm, and 9% reported being unable to walk. Largely because of their disability, 81% of the participants were not currently employed outside the home.

Procedure

Participants were asked to complete a brief structured questionnaire (diary) on 28 consecutive days. They were instructed to complete the diaries before going to bed each evening. If they forgot to complete their diary on a particular day, they were requested to complete it as soon as possible and to note the date and time when the diary was completed. A packet of seven questionnaires was mailed to participants for the beginning of each week, and participants returned the packets by mail at the end of the week. The first packet also contained a background questionnaire that included questions about demographics and medical status. On completion of the study, participants were given the option of receiving $5 compensation for their participation or having the money donated to the RSDS Association.

Measures

The daily structured questionnaire included items about mood, pain, support, and conflict.

Mood checklist. Each day, participants indicated the degree to which they had experienced each of 27 emotions over the previous 24 hr on a 3-point scale ranging from 1 (no) to 3 (very much). Items were adapted from the Depression, Anxiety, and Anger/Hostility subscales of the Affect Balance Scale (Derogatis, 1975). The list also included 7 positive mood

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1 Results were similar when the sample was restricted to women with RSDS.
items, which were not used in the present study. Scores on the items for each subscale were averaged to yield summary assessments of daily depressed mood, anxiety, and anger. Internal reliabilities (Cronbach’s alpha) for these subscales were .88, .80, and .86, respectively. As expected, daily reports of depressed, anxious, and angry mood were positively correlated (average within-subject rs ranged from .51 for depressed mood and anxiety to .61 for anxiety and anger). An overall negative mood score for each day was obtained by taking the mean of scores on all 20 negative mood items (α = .93).

Relative pain severity. Each day, RSDDS patients answered the question “Compared to the average day, how would you rate your level of physical pain today?” on a 5-point scale (1 = much lower than normal, 2 = lower than normal, 3 = normal, 4 = higher than normal, 5 = much higher than normal). Although pain measures typically ask people about absolute rather than relative levels of pain severity, we asked about relative pain severity because pilot work revealed a ceiling effect for daily ratings of absolute pain severity. This is consistent with findings that, relative to other chronic pain conditions, RSDDS pain is especially severe (e.g., DeGoed et al., 1993). The average daily pain rating was almost 4 (M = 3.96, SD = 0.47), suggesting a positive bias in reports of relative pain severity.

Perceived support. Participants were asked to check which, if any, of the following people had helped them with a worry, problem, or difficulty in the past 24 hr: (a) spouse, (b) parent or parent-in-law, (c) child, (d) other relative, (e) friend or neighbor, (f) coworker, or (g) someone else. They were told that help could be emotional (e.g., listening or comforting) or practical (e.g., doing something concrete). Pilot testing had shown that participants could easily generate examples of each type of support from their daily lives. Spouses were most commonly reported as providing support. They provided support on more than 60% of days for the average participant. Other common sources of support were children (30%) and friends or neighbors (25%). Support was provided on 10% to 15% of days by parents or parent-in-law, other relatives, and others. Support by a coworker was rare (3%), probably because few RSDDS patients were currently employed. The number of people from whom the participant received either practical or emotional support was summed to provide an overall index of daily perceived support. The average daily support for the present sample was 1.55 (SD = 0.85).

Conflict. Participants indicated whether over the past 24 hr they had experienced an argument, conflict, or tension with any of the following people: (a) spouse, (b) parent or parent-in-law, (c) child, (d) other relative, (e) friend or neighbor, (f) coworker, or (g) someone else. The number of individuals with whom the participant had argued was summed to obtain an index of daily conflict. Participants reported an average of 0.64 conflicts (SD = 0.52) per day.

Compliance and Attrition

A total of 153 RSDDS patients contacted us requesting information about participating in the study. Of these, 109 (70%) provided at least 1 week of diary data. These 109 patients completed the diary on 2,765 of 3,052 possible days (28 days × 109 participants), for a 91% completion rate. The vast majority of completed diaries were filled out on the appropriate day (98%). Two percent of diaries were completed 1 day late, and the remaining 0.4% of diaries were completed 2 or 3 days late. We used analyses of variance and chi-square tests to determine whether those participants completing more than 3 weeks of diaries (“completeers”) differed systematically from participants providing 5 or fewer weeks of data (“noncompleters”). We conducted analyses on demographic variables (participant gender, age, ethnicity, religion, education, and employment status; spouse age, education, and employment status; and family income and lawsuit involvement), medical variables (length of illness, RSDDS stage, and level of physical disability), and diary variables (mean daily mood, pain, support, and conflict). No systematic differences were found between completeers and noncompleters (i.e., for all tests, p > .05).

Diary Data Analyses

To assess the hypothesized associations among daily negative mood, pain, and support outlined in the introduction, we conducted analyses using a multilevel, or hierarchical, linear model (Bolger & Zuckerman, 1995; Bryk & Raudenbush, 1992; Kenny, Bolger, & Kashy, in press; Kenny, Kashy, & Bolger, 1998). This model allowed us to obtain separate estimates for each person of the associations among mood, pain, and support and to efficiently aggregate these estimates to provide results for the average person. It also allowed us to test the possibility that people would differ significantly from one another in the associations of interest and to take account of this variability when testing the significance of the associations.

The statistical model that we used had two levels, a within-person level and a between-persons level. For the within-person level, each person had their own equation specifying the distinctive association between variables of interest, such as depressed mood and pain, for that person. To help reduce ambiguity in the causal direction of the associations examined, we assessed the cross-day associations between variables (Kesseler & Greenberg, 1981). Specifically, we estimated the association between the independent variable on the previous day, t − 1, and change in the dependent variable from the previous day, t − 1, to the present day, t. This was achieved by including the lagged value of the dependent variable (i.e., the value at day t − 1) as a control variable. In such a model, the dependent variable can be viewed as residualized change in distress from day t − 1 to day t.

The Level 1 within-person model is as follows for the effect of previous day’s pain, P, on current day’s depressed mood, D:

\[
D_t = a_0 + a_1 D_{t-1} + a_2 P_{t-1} + q_t.
\]

The term \(D_t\) is the person’s depressed mood on day \(t\), \(D_{t-1}\) is the person’s depressed mood on day \(t - 1\), and \(P_{t-1}\) is the person’s pain on day \(t - 1\). The term \(q_t\) is a residual component of the person’s depressed mood on day \(t\) and is assumed to be a normally distributed random variable with a mean of 0 and constant variance. The term \(a_0\) is the mean level of the person’s depression across all days. The terms \(a_1\) and \(a_2\) denote the coefficients for the effect of the previous day’s depressed mood, \(D_{t-1}\), and pain, \(P_{t-1}\), respectively, on the present day’s depression for person \(i\). Estimates of \(a_0\), \(a_1\), and \(a_2\) were obtained for each individual, \(i\), in the sample. These estimates, \(a_{0i}\), \(a_{1i}\), and \(a_{2i}\), are dependent variables in the Level 2 equations. The Level 2 equations express each randomly varying effect as a function of a mean value (the effect for the average person) plus a deviation (the extent to which each individual’s effect is higher or lower than the effect for the average person). The equations are as follows:

\[
\begin{align*}
\alpha_0 & = \beta_0 + \epsilon_0, \\
\alpha_1 & = \beta_1 + \epsilon_1, \\
\alpha_2 & = \beta_2 + \epsilon_2.
\end{align*}
\]

In this analytic approach, it is assumed that the variables of interest were measured without error. To the extent that this is not warranted, the parameter estimates may not be accurate. Kenny and Zautra (1995) offered an alternative to the analytic approach used in the present article (the trait-state–error model for multivariate data). Their approach, which uses structural equation modeling, was used by Zautra et al. (1995) to address the pain–depression association with multivariate monthly data. Although the Kenny and Zautra approach can take account of measurement error, it has a number of limitations that do not apply to multilevel diary analyses approaches. As Kenny and Zautra noted, their approach is limited to bivariate relationships, does not allow for individual differences in slopes, and requires that no data are missing. Given these limitations of the Kenny and Zautra approach and the absence of other established ways of handling possible measurement error in multilevel data, we have adhered to standard time-lagged approaches to analyzing diary data (e.g., Bolger & Zuckerman, 1995; Larson & Almeida, 1999).

Because the association between variables of interest, such as pain and depressed mood, might reflect the influence of day of the study, we included day of study as a control variable in all of our analyses.
Level 2: \[ a_0 = c_0 + e_0 \]
\[ a_{ij} = c_1 + e_{ij} \]
\[ a_{2i} = c_2 + e_{2i} \]

Thus, \( e_0 \) signifies the predicted level of depressed mood for the average person on the average day. The term \( c_0 \) signifies person \( i \)'s deviation from that average. The term \( c_1 \) signifies the impact of the previous day’s depression on the current day’s depression for the average person. The term \( c_2 \) signifies the impact of the previous day’s pain on the current day’s depression for the average person. The term \( e_{2i} \) signifies person \( i \)'s deviation from that average. The terms \( e_0 \), \( e_{ij} \), and \( e_{2i} \) are assumed to be normally distributed random variables with a mean of 0 and constant variance.

The statistical model for the depressed mood–pain relationship explained above is an example of the main-effects models that we estimated to establish the effect of pain on mood (general, negative, depressed, anxious, and angry mood), mood on pain, and support on pain and on mood. We were also interested in testing whether the effect of mood on pain, and vice versa, was moderated by social support. The Level 1 within-person model estimated to test whether social support, \( S \), moderates (or buffers) the effect of pain, \( P \), on depressed mood, \( D \), is as follows:

\[ D = b_0 + b_1 S_{i-1} + b_2 P_{i-1} + b_3 S_{i-1} \times P_{i-1} + e_i. \]  

(2)

In this model, \( S_{i-1} \) refers to level of support on day \( t \) and \( P_{i-1} \) refers to the interaction between level of support and level of pain on day \( t - 1 \). We expected the coefficient for \( S_{i-1} \times P_{i-1} \) to be negative, indicating that support reduces the effect of pain on depressed mood. As above, separate equations were estimated for each person. The corresponding Level 2 between-persons equations can be derived in the same fashion that they were derived above for the example depicting the depressed mood–pain relationship.

We conducted these analyses using a weighted least squares approach, which was implemented using a modification of PROC GLM in SAS (see Kenny, Bolger, & Kashy, in press). This procedure is suitable for testing random effects models but may yield estimates that are slightly less efficient than maximum likelihood estimations of multilevel models.

**Results**

**Relating Pain and Mood Across Day**

The first goal of this study was to investigate the temporal relationship between daily pain and negative mood. Thus, we investigated the influence of the previous day’s pain on the present day’s mood and of the previous day’s mood on the present day’s pain.

**Effect of pain on mood.** We used the multilevel approach described above to assess the extent to which the present day’s negative mood was predicted by the previous day’s pain. Pain significantly predicted overall negative mood, \( B = 0.02, t(108) = 2.50, p < .05 \). Because the previous day’s negative mood was included as a control variable, this finding can be interpreted as showing that participants reported increases in negative mood following high-pain days.

Next, we examined whether the findings for overall negative mood would generalize to different forms of negative mood or whether there would be specificity in the associations of pain with depressed mood, anxiety, and anger. Previous day’s pain was a significant predictor of present day’s depressed mood, \( B = 0.03, t(107) = 2.94, p < .01 \). Significant effects of comparable magnitude\(^4\) were found on anxiety, \( B = 0.03, t(108) = 2.61, p < .01 \), and anger, \( B = 0.03, t(108) = 3.00, p < .01 \). These findings show that when participants experienced high pain, they showed increased levels of depressed mood, anxiety, and anger the following day.

Previous day’s pain was also a significant predictor of present day’s conflict, which may be viewed as expressed anger, \( B = 0.07, t(108) = 2.82, p < .01 \). To establish whether this effect was mediated by the present day’s anger, we reestimated the original model with present day’s anger included as an independent variable. The coefficient for previous day’s pain became nonsignificant, \( B = 0.04, t(107) = 1.66, p > .05 \), whereas the coefficient for present day’s anger was highly significant, \( B = 0.63, t(107) = 10.54, p < .001 \). Thus, these findings suggest that pain leads to increased conflict through its positive impact on anger.

**Effect of mood on pain.** The impact of previous day’s overall negative mood on present day’s pain was not significant, \( B = 0.04, t(108) = 0.73, ns \). We assessed whether increased pain was predicted by any of the three specific types of negative mood: depressed mood, anxiety, or anger. We conducted separate analyses for each mood type. Previous day’s depressed mood was a significant predictor of present day’s pain, \( B = 0.10, t(108) = 2.22, p < .05 \). Present day’s pain was not significantly predicted by either previous day’s anxiety, \( B = -0.04, t(108) = -1.02, ns \), or anger, \( B = 0.02, t(108) = 0.38, ns \). In addition, it was not predicted by previous day’s conflict, \( B = -0.03, t(106) = -1.06, ns \). Thus, only depressed mood predicted increased pain the following day.

Although neither anger nor conflict (i.e., expressed anger) were significant predictors of increased pain, suppressed anger could still predict increased pain. To test this hypothesis, we investigated whether high levels of feeling angry combined with low expressed anger, operationalized as conflict, would predict increases in pain. We conducted analyses to examine the impact on present day’s pain of the previous day’s anger and conflict and their interaction, controlling for the previous day’s pain. The Anger \( \times \) Conflict interaction term was not significant, \( B = 0.05, t(105) = 0.77, ns \). To test whether complete anger suppression would predict increased pain, we dichotomized the conflict variable into “no conflict” (43% of days) versus “some conflict” (57% of days). We then assessed the impact on present day’s pain of the previous day’s anger and dichotomized conflict and their interaction, controlling for the previous day’s pain. The Anger \( \times \) Conflict term was not significant, \( B = -0.03, t(105) = -0.24, ns \).

**Summary.** On the day following a high-pain day, participants showed elevated depressed mood, anxiety, anger, and conflict. Thus, pain appeared to elicit generalized emotional distress, which can emerge in conflict. Only depressed mood predicted increased pain across day, supporting the hypothesis that depressed mood plays a special role in the course of chronic pain. Although pain predicted increases in anxiety and anger, these aspects of mood were not significant predictors of increases in pain. Suppressed anger, operationalized as anger unexpressed in interpersonal conflict, did not predict increases in pain.

\(^4\) Note that unstandardized betas are presented. However, the three mood variables have similar standard deviations.
Role of Social Support

We conducted the next set of analyses to assess the influence of overall daily perceived support on mood and pain. Supplementary analyses, which are not reported, showed that the effects of perceived support from specific sources (e.g., spouses or children) showed trends similar to the effects for overall support reported here, although very few of the specific source effects met conventional levels of statistical significance.

Effects of support on mood. Previous day's social support predicted lower present day's negative mood, \( B = -0.03, t(107) = -3.82, p < .01. \) With regard to specific forms of negative mood, support predicted decreased depressed mood, \( B = -0.02, t(106) = -3.22, p < .01, \) and anger, \( B = -0.03, t(107) = -3.38, p < 0.1. \) Previous day's support did not significantly predict present day's anxiety, \( B = -0.01, t(106) = -1.44, \) ns.

Next, we investigated whether perceived support would buffer the impact of pain on distress by adding a Support \( \times \) Pain interaction term to the main-effects model tested above. For overall negative mood, the interaction term was statistically significant, \( B = -0.02, t(100) = -2.39, p < .05. \) To investigate whether perceived support buffered participants against negative mood in general or against only certain forms of negative mood, we conducted separate multilevel analyses for depressed mood, anxiety, and anger. Each model yielded similar results to the model for overall negative mood, although the interaction term was statistically significant only for depressed mood, \( B = -0.02, t(100) = -2.31, p < .05. \) Nonsignificant trends were observed for anxiety, \( B = -0.02, t(100) = -1.34, \) ns, and anger, \( B = -0.01, t(100) = -1.36, \) ns.

Figure 1 plots the predicted values of present day's depressed mood as a function of previous day's level of pain and support. Specifically, values are given for the depressed mood–pain relationship following high-support days (i.e., a level of perceived support 1 SD above the mean) and following low-support days (i.e., 1 SD below the mean). As the figure shows, differences in depressed mood as a function of whether support on the previous day was high or low were most pronounced under conditions of higher than average pain. Thus, there is evidence of a buffering effect of support on the pain–mood relationship.

![Figure 1](image-url)

**Figure 1.** Predicted values of depressed mood as a function of the previous day’s pain and support.

Effects of support on pain. Previous day’s support significantly predicted reduced present day’s pain, \( B = -0.04, t(106) = -2.12, p < .05. \) Perceived support did not moderate the influence of depressed mood on pain: for Support \( \times \) Depressed Mood, \( B = 0.04, t(106) = 0.70, \) ns.

Summary. Perceived support had both direct and buffering effects on negative mood. Significant main effects of support were observed on overall negative mood, depressed mood, and anger. Support also significantly buffered (i.e., moderated) the effect of pain on overall negative mood and on depressed mood. Thus, perceived support had a positive impact on mood in RSDS patients, and this beneficial effect was most pronounced when they were in the most pain. Support also predicted a reduction in perceived pain.

Discussion

This study used a daily diary methodology to address questions concerning the within-person associations among negative mood, pain, and social support in patients with the chronic pain condition RSDS. In the present study, pain predicted increases in depressed mood, anxiety, and anger. Depressed mood, but not anxiety or anger, predicted increases in pain. Perceived support helped reduce both negative mood and pain. In addition to having a direct salutary impact on negative mood, support helped reduce the effect of pain on overall negative mood and on depressed mood. As is common in daily diary studies, the cross-day associations that we documented among pain, mood, and support were relatively small. However, as Repetti and Wood (1997), drawing on Abele (1985), noted in defense of findings from daily diary studies, “when even small amounts of variance are explained in particular situations, the underlying processes can account for important long-term outcomes if the situations recur and the effects accumulate” (p. 191).

Pain and Negative Mood

The finding that pain leads to increases in depressed mood, anxiety, and anger suggests that pain is a trigger of generalized negative mood. This is consistent with a view of pain as a daily stressor in the lives of persons with chronic pain. Pain also contributed to increases in conflict to the extent that it induced anger. Despite this tendency for pain-related anger to spill over into interpersonal conflict, other findings from our research on RSDS patients indicate that, as a group, RSDS mothers’ anger is less likely to spill over into negative exchanges with their children than is the case for non-RSDS mothers (Downey et al., 1999).

 Whereas pain influenced the three types of negative mood in similar ways, the converse was not true. Depressed mood alone led to an intensification of pain. An influence of depressed mood on pain is consistent with general theoretical models that view pain perception as mediated by such psychological factors as mood, expectation, and appraisal (e.g., Melzack & Wall, 1982; Turk, Meichenbaum, & Genest, 1983). What specific mechanisms might underlie the unique influence of depressed mood on pain observed?

\(^5\) None of the measures of previous day’s mood were a significant predictor of present day’s support.
in this study? One possible way in which depressed mood can influence pain is through its impact on attention regulation. There is experimental evidence that depressed mood heightens attention to the self (Salovey, 1992; Wood, Saltzberg, & Goldsamt, 1990), particularly to negative aspects of the self (Carr, Teasdale, & Broadbent, 1991). Self-focused attention is associated with heightened perception of internal states, including somatic sensations (Pyszczynski & Greenberg, 1987). On the other hand, diverting attention from the emotionally distressing aspects of pain, such as through distraction or sensory redefinition, enhances pain tolerance and decreases pain sensitivity (see McAul & Malott, 1984; and Pearce, 1983, for reviews). Thus, by inducing self-focused attention, depressed mood may intensify pain sensitivity.

Depressed mood may also heighten pain sensitivity by fostering a sense of helplessness that encourages catastrophization about pain (Geisser, Melody, Gaskin, Robinson, & Greene, 1993; Geisser, Robinson, Keefe, & Weiner, 1994; Rosenstiel & Keefe, 1983). Besides directly increasing the aversiveness of pain, helplessness and catastrophization may disrupt the planning and enactment of strategies for coping with pain. This may lead to a self-perpetuating cycle, in which depressed mood impairs efforts to cope effectively with pain, which in turn leads to increased pain. The increased pain then leads to a greater sense of helplessness and threat and thus accentuates depressed mood.

This explanation is consistent with the reciprocal relationship observed in the present study between pain and depressed mood. It can potentially help account for the maintenance of pain and depressed mood over time in persons with chronic pain. In this context, it is worth noting that our findings on pain and depressed mood generalized across participants with varying levels of overall depressive symptomatology. Analyses not reported in the Results section revealed no difference in the time-lagged relationships between pain and depressed mood for those participants with and without clinically significant levels of depressive symptomatology (as defined by a T score of at least 70 on the Symptoms Checklist—90—R Depression subscale).

Neither anxiety nor anger led to increased pain on a day-to-day level, whereas pain led to increases in both anxiety and anger. This suggests that prior cross-sectional findings linking anger and anxiety with pain severity may reflect the influence of pain on generalized distress rather than a causal role for anxiety or anger in the course of chronic pain. In the case of anxiety, it may be that participants’ anxiety was focused primarily on non-pain-related concerns and this served to distract their attention from the pain. As noted in the introduction, evidence from laboratory pain studies demonstrates that anxiety aroused by pain-irrelevant environmental cues actually dampens pain perception through its influence on attentional regulation (e.g., al Abt & Rokke, 1991; Amst et al., 1994). In contrast, depressed mood elicits generalized negative self-focused attention, irrespective of the specific content of the mood-induction stimulus. Thus, Zelman, Howland, Nichols, and Cleeland (1991) found that induction of depressed mood by a non-pain-related stimulus (i.e., the Velten mood-induction procedure) leads to decreased pain tolerance. In additional analyses not reported in the Results section, we found that, controlling for the concurrent association between anxiety and depressed mood, anxiety predicted significantly decreased pain the following day, \( B = -0.13, \kappa(105) = -2.54, p < .05 \). The positive effect of depressed mood on pain, on the other hand, became stronger, \( B = 0.17, \kappa(105) = 3.10, p < .01 \), indicating suppression by anxiety.

In the present study, we did not find that either anger in general or suppressed anger in particular influenced daily pain intensity. As with anxiety, anger might be expected to contribute to increased pain intensity only when it is focused on the pain, as opposed to non-pain-related stimuli. Our data did not support the specific hypothesis that suppressed anger contributes to pain (e.g., Beutler et al., 1986). Nevertheless, our measure of suppressed anger was indirect, given that it was based on reports of low interpersonal conflict coupled with high anger. Moreover, our study design did not address the role of suppressed anger in the onset of chronic pain, as opposed to its maintenance over time.

**Role of Social Support**

There is a consensus in the stress-and-coping literature that perceived social support enhances psychological well-being in general and buffers the impact of life stress in particular (Cohen & Wills, 1985). The results of the present study are consistent with this view. Perceived supportive interchanges led to reduced negative mood both directly and by buffering the effects of pain on mood. Following days on which participants reported higher levels of support receipt, they showed decreased in depressed mood, anger, and overall negative mood, indicating salutary effects of support. Perceived support also moderated the effect of pain on general negative mood and depressed mood. That is, the contribution of pain to overall negative mood and to depressed mood was reduced when the individual reported a greater number of supportive interchanges. These findings highlight the potentially important role that members of an informal social network can play in helping a person cope with chronic pain.

In addition to its impact on negative mood, perceived support also significantly contributed to lower pain. Controlling for depressed mood did not appreciably alter these results. Moreover, perceived support did not moderate the influence of depressed mood on pain. Thus, support and depressed mood made relatively independent contributions to pain. The beneficial effect of supportive interchanges on pain found in this study is contrary to what would have been expected if these interchanges had involved solicitous, pain-reinforcing behavior (Turk et al., 1992). This raises the question of what occurred during these interchanges to decrease perceived pain, as well as to buffer the effect of pain on depressed and general negative mood. During the diary study, we did not collect data on the nature of the specific supportive interchanges reported by participants. Thus, in a follow-up questionnaire, we asked participants to answer open-ended questions about things that other people have said or done that were especially helpful or unhelpful to them in coping with RSIDS.

Participant responses indicated that typical interchanges that were perceived to be supportive involved encouraging active coping with the condition and discouraging helplessness and catastrophization. Supportive comments often included acknowledgment of the difficulties faced by participants coupled with reinforcement for past and present coping efforts (e.g., “My friend told me that I had to just keep fighting, even when the pain was bad. Remember how precious life is” and “My son said, ‘I am proud of you, Mom. I know what you are going through isn’t easy, but you are a fighter’ ”). Also common was a commitment of
continued support, thereby giving the person confidence that she or he would not be alone in confronting the difficulties associated with the condition (e.g., “My spouse said we can get through this, we’ve been through a lot over the last 20 years”).

These qualitative data suggest how illness-relevant support that is perceived as being helpful can be distinguished from solicitous behavior that may heighten pain perception and pain behavior (e.g., “takes over my job or duties” and “tries to get me to rest”; Kerns, Turk, & Rudy, 1985). The data are consistent with research and theory that suggest that support is effective to the extent that it increases active coping and helps diminish feelings of helplessness and catastrophization (e.g., Manne & Zautra, 1989; Thoits, 1986). They also indicate the importance of exploring people’s own definitions of support to help clarify its subjective impact.

Caveats and Conclusions

Several limitations of the present study should be noted. First, we relied exclusively on participants’ self-reports. Although self-reports are the most direct index of subjective states like pain, their augmentation with direct or indirect (e.g., spousal report) behavioral indexes would help rule out potential within-subject confounds (Dworkin & Whitney, 1992). For example, the finding that pain contributed to increases in reported conflict may reflect a heightened sensitivity to interpersonal negativity when in pain rather than an increase in conflicted interpersonal interaction per se.

Second, we used a global, unidimensional measure of pain intensity, rather than independently assessing its sensory, cognitive, and affective components (Melzack & Katz, 1992). Thus, in interpreting our finding of an effect of depressed mood on pain, we cannot determine which aspect of pain perception was affected by mood. Insofar as depressed mood increased self-focused attention, it may have increased the sensory intensity of the pain; however, depressed mood also may have led people to appraise the pain sensation as more distressing (cf. Fields, 1991; Geisser et al., 1993).

Third, we measured daily mood and perceived support without reference to whether they were pain related or pain unrelated. As discussed earlier for the case of anxiety, whether a particular negative mood state is focused on pain or on unrelated stimuli may substantially alter its influence on pain perception. It appears plausible that, by controlling for previous day’s pain severity when examining the effect of previous day’s anxiety on present day’s pain, we were effectively controlling for that component of anxiety that was pain related. Thus, our findings concerning the impact of anxiety on pain probably pertain to non-pain-related anxiety. In future research, it will be important to explicitly measure pain-related anxiety using approaches such as that developed by McCracken et al. (1992).

Fourth, as is common with most research on social support, we assessed support from the perspective of the recipient, as opposed to that of the provider (cf. Bolger et al., 1996). Although perceived support receipt is more directly related to its psychological impact than support provision, investigation of the latter might have more far-reaching practical implications. It would be useful to know whether intended supportive attempts generally have positive effects on the recipient’s pain and mood, as well as which types of support attempts by the provider benefit the person in pain. This information could be helpful to both formal and informal providers of support to chronic pain patients.

Fifth, the generalizability of our findings to persons with other chronic pain conditions needs to be established. RSDS is an idiopathic pain condition of uncertain etiology and pathophysiology (Chard, 1991; Schwartzman, 1993), in the course of which psychological factors have been hypothesized to play a prominent role (Egle & Hoffman, 1990). Furthermore, RSDS is characterized by high levels of perceived pain relative to many other chronic pain conditions (e.g., Melzack & Katz, 1992). These factors may have contributed to our finding significant influences of mood and support on pain, where these would not be apparent in persons with conditions with a more well-defined pathophysiology or less extreme levels of pain severity.

Finally, it is not possible to determine how representative our sample was of RSDS patients because there is no national registry for RSDS patients such as exists for some other disorders. RSDS patients who are currently married and involved in RSDS support groups or in reading RSDS newsletters may differ in unknown ways from patients who are not involved in RSDS networks. It is also possible that the most severely distressed or impaired patients were unable to participate in a diary study.

These caveats notwithstanding, this study illustrates how a daily diary methodology can help delineate the relationships among pain, mood, and social support in chronic pain patients. The results support a model of pain as a daily stressor whose impact on emotional distress is buffered by perceived supportive interchanges. These results also support a model of pain as having a cognitive-affective component that is modulated by depressed mood and perceived support. Daily diary studies and studies using even more frequent experience-sampling procedures can help identify mechanisms underlying changes that have previously been documented over longer spans of time. Moreover, different mechanisms may operate over different time spans. For example, pain may directly contribute to distress over short time periods, whereas longer term effects on distress may be mediated by the functional, practical, and interpersonal consequences of pain. Future prospective longitudinal and experimental research will help to clarify further the complex relationships observed in this study among pain, mood, and support.

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