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## The Effect of Laboratory-Induced Depressed Mood State on Responses to Pain

Scott G. Willoughby, PhD; B. Jo Hailey, PhD; Shazia Mulkana, MS; Jennifer Rowe, MS

Some researchers have suggested that a depressed mood state is associated with alterations in responses to pain. The authors examined cognitive, behavioral, and affective responses of 75 randomly assigned participants to depressed, neutral, or elated mood state induction conditions and subjected them to the cold-pressor task. Because they were unsuccessful in inducing elated moods, the authors used only the data for the depressed and neutral states as they measured pain threshold, tolerance, and unpleasantness during the test. After the task, the authors measured sensory, affective, and evaluative responses to the cold-pressor pain, as well as the participants' catastrophizing ideation about the painful procedure. The depressed mood state group, compared with the neutral group, had significantly lower cold-pressor tolerance times and higher pain catastrophizing scores. These results support previous findings that a depressed mood state may be associated with alterations in some pain responses.

Index Terms: depressed mood state, depression, pain, pain catastrophizing, pain tolerance

During the past few decades, as unidimensional models of pain have given way to more comprehensive multidimensional conceptualizations, researchers and clinicians in the field of pain management have increasingly recognized that important relationships exist between the experience of pain and psychological variables. One of the more frequently emphasized of these relationships is that between pain and depression.

There is little doubt that a high comorbidity exists between pain and depression. The prevalence of depressive symptoms is much higher in chronic pain populations than in the general population, and it is clear that chronic pain can lead to depression.<sup>1-3</sup> In addition, individuals who suffer from depression experience a greater number of acute and chronic pain complaints than nondepressed individuals

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do<sup>4-7</sup> and treatment with antidepressant medication often leads to reductions in pain in both depressed and nondepressed patients.<sup>4,8</sup> Furthermore, some studies suggest that depression serves as a risk factor for developing chronic pain problems<sup>9-12</sup> and that depression is a predictor of poor response to psychophysiological<sup>13</sup> and surgical treatment for pain.<sup>14-16</sup> Moreover, evidence that depressed mood prior to surgery is associated with increased complaints of post-operative pain is limited.<sup>17</sup>

Taken together, these findings suggest that a meaningful, and possibly reciprocal, relationship exists between pain and depression. That is, although depression can be a consequence of living with pain, depressed mood state or clinical depression may also significantly influence the experience of pain. Interestingly, studies that have directly addressed the question of whether depressed individuals experience greater sensitivity to experimental pain have generally not found increased pain sensitivity, as measured by pain threshold. In fact, one study found no difference in pain threshold, 18 and others have found an increased threshold. 19-22 Thus, evidence has emerged that although depres-

sion is associated with more complaints of clinical pain, it is apparently not associated with increased pain sensitivity. This conclusion was recently underscored by findings of Lautenbacher and associates,23 who demonstrated that although their sample of depressed psychiatric inpatients reported significantly more clinical pain during recent months than nondepressed controls, the same patients did not show greater pain sensitivity (ie, decreased pain threshold) when administered experimental pain stimuli. Rather, the depressed patients were less sensitive to pressure pain, relative to controls, and showed no significant difference in sensitivity to cold pressor and heat pain. Furthermore, they found no significant correlations between clinical pain complaints and experimental pain threshold. These researchers concluded that in contrast to earlier speculations, the high rate of pain complaints in depressed patients probably is not related to increased pain sensitivity as measured by pain threshold.

These findings appear to be somewhat contrary to results of studies of clinical pain that indicate an exacerbation of the experience of pain in depressive patients. For example, one study found higher affective pain intensity ratings, but not sensory intensity ratings, in depressed patients with clinical pain syndromes, relative to nondepressed pain patients. Another study found a positive relationship between pain intensity and depressive symptoms independent of level of disease activity and disability in rheumatoid arthritis patients. And still another found increased reports of pain intensity in the evening in depressed chronic pain patients compared with nondepressed chronic pain patients, as well as a relationship between degree of depression and degree of impairment in pain patients.

This discrepancy could suggest that depression has a different effect on clinical pain than on experimental pain. However, it also appears reasonable to hypothesize that this discrepancy reflects the notion that depression is related to different aspects of the pain experience in different ways. That is, depression may have a negative influence on an individual's psychological (eg, greater pain catastrophizing and affective distress) and behavioral (eg, decreased willingness to tolerate pain and greater propensity to complain about a given painful stimulus) reactions to pain, even if it is not related to greater pain sensitivity. This leads to an important question that has not been adequately addressed: Is depression associated with an alteration of various responses to pain beyond pain sensitivity or threshold? In the present laboratory analog study, we examined several different types of responses to a painful stimulus in participants whose mood was experimentally altered.

One experimental study has provided evidence that

depressed mood state influences reaction to a pain stimulus without influencing the perceived intensity of the pain.<sup>27</sup> In that study, participants were administered the cold-pressor task before and after a mood induction procedure designed to elicit either depressed, neutral, or elated mood states. Although the researchers found no significant differences in pain intensity ratings taken during the cold-pressor tasks, participants in the depressed mood induction condition showed significantly decreased pain tolerance time (number of seconds that the participant remains in the painful procedure) following mood induction; those in the elated mood induction condition showed significantly increased tolerance; and participants in the neutral mood condition showed no significant changes in tolerance. Thus, mood state was associated with an alteration of behavioral reaction (ie, amount of time the participant chooses to endure the pain), but it did not appear to affect reported pain intensity.

We designed the present study to investigate the influence of depressed mood state on the experience of pain further by examining the effect of laboratory-induced depressed mood, compared with neutral and elevated mood on various responses to cold-pressor pain. Our investigation expanded on previous research by using a more comprehensive, multidimensional evaluation of the pain experience that included evaluation of pain catastrophizing as well as sensory, affective, and evaluative dimensions of the pain experience. On the basis of previous findings suggesting that depressed mood influences tolerance for a painful stimulus<sup>27</sup> and affective pain intensity,<sup>24</sup> we predicted that, relative to neutral and elated mood, induced depressed mood state would be associated with lower tolerance time, higher scores on the Affective Pain Rating Index of the McGill Pain Questionnaire (MPQ), 28 and higher unpleasantness ratings. In addition, we hypothesized that induced depressed mood would be associated with altered cognitive-evaluative responses to the pain stimulus. That is, depressed mood would be associated with higher Evaluative Pain Rating Index scores on the MPQ and higher scores on a measure of catastrophizing cognitions about the pain experience. Although we were concerned with laboratory-induced depressed mood state rather than clinical depression, this hypothesis is consistent with Fields's<sup>29</sup> theory that depression affects the evaluative aspect of the pain experience, because individuals tend to evaluate events (including a pain experience) more negatively during depressive mood states. In addition, this prediction is somewhat consistent with research that has revealed a positive relationship between depression and pain catastrophizing in chronic pain patients.30,31

#### METHOD

#### **Participants**

The participants were 75 college students (21 men and 54 women) recruited from undergraduate psychology classes. Participants were excluded from the study if they reported conditions that might have been exacerbated by the experimental procedures, including painful conditions affecting the arms, history of frostbite, and significant depressive symptoms. Furthermore, because analgesic and antidepressant medications were likely to alter responses to experimental procedures, participants who reported taking antidepressant medications or who reported taking analgesic medications within 24 hours before the experimental procedure were excluded from the experiment. After completion of the experiment, individuals received extra credit toward their psychology course grade as compensation for their participation.

#### **Procedure**

Before we collected data, we received approval for the study from the University's Human Subjects Protection Review Committee. Three female research assistants who were unfamiliar with the hypotheses of the study collected all the data. They entered all relevant data on a data-collection form that was number coded so that the names of participants were not associated with any data.

#### Recruitment and Screening of Participants

Participants who volunteered were contacted by telephone so they could be screened for exclusion criteria. They were told that the study concerned mood and involved immersing one's hand in cold water, a painful experience. We scheduled those who met the inclusion requirements for the experimental session and told them to refrain from taking any analgesic medications during the 24 hours before the appointment.

We obtained written informed consent from participants when they arrived at the laboratory. We then asked them to complete a brief questionnaire that provided basic information about demographic backgrounds, recent use of medication, and painful conditions affecting their left arm. We disqualified 1 individual because of analgesic use within the previous 24 hours. Then we administered the Beck Depression Inventory (BDI), a 21-item self-report instrument<sup>32</sup> that we used to exclude individuals who reported a significant degree of depressive symptomatology and subsequently disqualified 10 individuals from participation because their BDI raw scores were above the exclusion cutoff.<sup>15</sup>

#### Mood Manipulation Procedure

We used a stratified random assignment procedure to ensure that the ratio of men to women was the same in each of the 3 mood conditions. In preparation for the mood induction procedure, the experimenter asked each participant to remain quiet and to relax for 2 minutes.

The Velten Mood Induction Procedure (VMIP)33 is a task designed to induce depressed, elated, or neutral mood states. The VMIP involves having participants read 9 instructional statements, then giving them a set of 60 cards, each with a statement typed in capital letters. Participants are asked to read and to "try to feel the mood suggested by" the statement on each card. Every 20 seconds, a sound on an audiotape cues them to go to the next statement. Thus, the mood induction procedure takes approximately 20 minutes. Cards used for the depressed mood state condition include self-referent statements suggestive of self-devaluation and of somatic characteristics of clinical depression. The elated mood condition contains self-referent statements suggestive of elevated mood. Cards for the neutral mood condition contain statements that are unlikely to be associated with depressed or elated moods.

The depressed mood state condition produces a subtle and short-lived mood state similar to that found in mild, naturally occurring depression.34,35 Both the depressed and elated conditions produce mood changes that tend to persist for several minutes after the procedure is completed but do not typically endure for more than 10 to 20 minutes.<sup>36</sup> Clark's<sup>37</sup> review indicates that a number of studies have demonstrated that participants who undergo the depressed mood induction report significantly higher levels of depressed mood than participants in the neutral and elated conditions. In addition, the Clark review indicated that, consistent with features of clinical depression, the depressed mood induction results in psychomotor slowing; decreased decision-making efficiency; lower ratings of pleasantness of activities; and altered social behavior, including reductions in eye contact, use of hand illustration, and helpfulness.

State Form of the Multiple Affect Adjective Check List-Revised (MAACL-R)

The State Form of the MAACL-R<sup>38</sup> is a 132-item instrument designed to measure affective state. Each item consists of only 1 adjective; therefore it is administered very rapidly (normally 2 to 4 minutes). Reliability and validity of MAACL-R have been well established.<sup>39,40</sup> In the present study, we administered it immediately after the mood induction procedure to evaluate its efficacy in producing the desired mood. The standard (T) score of the Positive Affect scale was subtracted from the standard score of the Depres-

Vol 28, Spring 2002 25

sion scale. This yielded a Depression-Positive Affect score (D-PA), with a higher D-PA indicating more depressed mood and lower D-PA indicating more positive mood. This procedure is similar to that Lassiter and colleagues<sup>41</sup> used in previous research.

#### Pain Stimulus

After the mood induction and administration of the MAACL-R, the experimenter presented the cold-pressor apparatus. The cold-pressor task (CPT) served as the painful stimulus. This procedure provides a continuous, nondamaging stimulus and is often used in studies of experimental pain. Although many trials are sometimes used to establish baseline performance, we elected to use only one administration of the task because of practical and ethical concerns. We wanted to limit participants' pain, and we were concerned about the likelihood of participants dropping out of the experiment if they were to be subjected to this painful procedure more than once.

The procedure involved immersion of the left hand and arm to 4" above the wrist in a container of ice water. A  $10'' \times 14'' \times 10''$  styrofoam ice chest with a screen partition in the middle was filled with water. One side of the container was filled with ice to maintain water temperature of 33 ± 1 °F. The water temperature was measured with a thermometer, and the water was circulated with a DC bilge pump so that there would be no local warming of the water around the hand and arm. The examiner then asked the participant to follow the instructions for the cold-pressor task provided on an audiocassette. The experimenter remained behind the participant during the entire procedure. Each participant was asked to immerse the left hand and forearm in the water up to a specified point marked by the examiner, and told to keep the hand still with palm face down and fingers pointed toward the bottom of the container. A picture demonstrating the proper hand position was posted in the experiment room. Participants were instructed to indicate when the water began to be experienced as "painful," and the examiner recorded the time from immersion to this report. We used this time period as a measure of pain threshold.

Participants were also cued at 15 seconds by a taperecorded tone to give a numerical rating (0–10) of "unpleasant at all and 10 indicating extremely unpleasant. Participants were also asked to leave the hand and arm in the water until they felt "forced to take their arm out," which was similar to the procedure Zelman et al<sup>27</sup> used. The examiner recorded the time from immersion to withdrawal of the hand, and used this as a measure of pain tolerance. Any participant

who maintained the hand in the water for 5 minutes was instructed to withdraw his or her hand at that time.

#### **Additional Measures**

Pain Rating Index of the MPQ

The Pain Rating Index of the MPQ28 was verbally administered immediately after completion of the CPT. The Pain Rating Index of the MPQ consists of 20 sets of pain-related adjectives, with each set containing 2 to 6 words. Within each set, the words are ranked by intensity. Word sets are grouped into 4 categories pertaining to various dimensions of pain. This grouping provides a basis for the following subscales: Sensory, Affective, Evaluative, and Miscellaneous. The words in each set were read to the participant, following the standard administration protocol, and he or she was asked to choose one of the words that best described the pain. They had the option of skipping a word set if none of the words applied. The MPQ was originally designed to assess clinical rather than experimental pain. In our study, the administration and scoring of the MPQ were standard,<sup>28</sup> except that the instructions were slightly modified so that the participant was asked to select words describing pain produced during the CPT rather than clinical pain. This was the only modification we made.

#### Modified Version of Pain Catastrophizing Scale (PCS)

Upon completion of the MPQ, the experimenter asked the participant to complete the PCS, a relatively new self-report instrument consisting of 13 statements that describe negative thoughts and feelings associated with pain experiences.<sup>42</sup> The individual is to "indicate the degree to which you have these thoughts and feelings when you are experiencing pain" by rating each statement on a 5-point scale. The ratings are summed to obtain a single score, with higher scores indicating a greater degree of catastrophizing.<sup>43</sup> Sullivan et al<sup>42</sup> reported good reliability (test-retest r = .75) and internal consistency (coefficient  $\alpha = .87$ ) for the instrument, as well as validity in measuring catastrophizing in both nonclinical and clinical samples. Because the PCS was designed to reflect the tendency to catastrophize about pain experiences in general rather than about a specific instance of pain, we modified the instructions and items slightly for the present study so that the individual could report on ideation during the CPT, as opposed to indicating thoughts and feelings associated with other painful experiences. We found good internal consistency of our version of the PCS (coefficient  $\alpha = .91$ ).

After completion of a postexperimental questionnaire designed to examine whether participants had been able to guess the purpose of the experiment, the experimenter dis-

missed participants in the elated and neutral mood conditions. To eliminate residual negative effects of the depressed mood induction, we administered 25 of the VMIP elated mood cards to the participants in the depressed mood condition.

#### **RESULTS**

Eighty-seven participants completed the full screening for the experiment, but we disqualified 10 of them before the mood manipulation because they had elevated BDI scores. One was disqualified because of recent analgesic use. Another 1 voluntarily withdrew from the experiment during the mood induction procedure, leaving 75 participants (21 men and 54 women) who completed the experiment. However, as we explain below, we used data from only 50 of the participants (14 men and 36 women, mean age = 23.2 years).

### Evaluation of Mood Differences Following Mood Induction (Manipulation Check)

We subtracted the standard (T) score from the standard score of the Depression subscale of the MAACL-R, producing a single index of current mood (D-PA), with a higher score indicating more depressed mood. One-way analysis of variance (ANOVA) revealed a significant difference in D-PA scores among conditions in the expected direction, F(2, 72) = 23.2, p < .001. Follow-up tests, however, indicated a significant difference between the depressed and neutral conditions, but no difference between the elated and neutral conditions, indicating that the elated mood was not ade-

quately induced. Thus, although the VMIP was successful in inducing depressed mood, it was not successful in inducing elated mood. The analyses that follow therefore include the depressed and neutral mood conditions as the experimental groups and exclude participants in the elated condition because this group was not significantly different from the neutral group on mood state.

#### **Equivalency of Conditions Analyses**

We conducted analyses to determine whether the experimental groups differed on demographic variables that might have influenced their scores on the dependent measures. A t test indicated no significant age difference between the 2 groups. Chi-square analyses indicated no significant differences between the groups in race, marital status, college classification, or smoking status. In addition, chi-square analyses showed that the female participants in each group did not differ in their use of oral contraceptives or phase in menstrual cycle.

#### **Main Analyses**

We used a priori *t* tests with alpha level set at .05 to test differences between depressed and neutral mood state groups on each dependent variable for which we made a priori hypotheses. We performed these a priori *t* tests for the following: (a) pain tolerance, (b) pain unpleasantness ratings, (c) MPQ Affective score (MPQ-A), (d) MPQ Evaluative score (MPQ-E), and (e) PCS score. Because we made directional a priori hypotheses for each of these variables,

TABLE 1						
Means and Standard Deviations of Mood State Induction Groups on Pain Measures						

Measure	Depressed		Neutral		
	M	SD	M	SD	t
Threshold (s)	21.36	25.53	18.24	17.70	.50
Tolerance (s)	42.60	33.15	61.08	41.78	-1.7*
Unpleasantness rating at	6.10	2.73	6.08	2.63	.03
15 s (0–10 scale)†					
McGill Pain Questionnaire					
Sensory	25.32	7.87	22.28	7.36	1.41
Affective	5.96	3.78	4.08	4.32	1.64
Evaluative	3.84	1.28	3.72	1.43	.31
Pain Catastrophizing Scale	29.24	12.78	23.24	11.29	1.76

*Note.* Depressed n = 25. Neutral n = 25.

\*p < .05.

Vol 28, Spring 2002 27

<sup>†</sup>Unpleasantness ratings were not available for 5 members of the depressed group.

we used 1-tailed t tests of significance (see Table 1 for means and standard deviations for each of these variables, as well as for other dependent variables.)

We found no significant differences between the groups on MPQ-A and MPQ-E scores or unpleasantness ratings obtained at 15 seconds into the cold-pressor task. However, the depressed mood state group (M = 42.60, SD = 33.15) showed significantly lower pain tolerance than the neutral mood group (M = 61.08, SD = 41.78), t(48) = -1.73, p < .05; and the depressed mood state group showed significantly higher scores (M = 29.24, SD = 12.78) on the Pain Catastrophizing Scale than the neutral group (M = 23.24, SD = 11.29), t(48) = 1.758, p < .05.

#### **Post Hoc Analyses**

In addition to the main analyses, we performed 2-tailed t tests to examine differences between the 2 groups on pain measures for which hypotheses were not made. We detected no significant differences in pain threshold time, t(48) = .502, p = .618 or MPQ Sensory score, t(48) = 1.411, p = .165.

#### **COMMENT**

In this study, we used a controlled laboratory experiment to investigate the influence of depressed mood state on responses to a painful stimulus. Our general finding was that induced depressed mood affected some aspects of the pain experience but not others. Specifically, participants subjected to laboratory-induced depressed mood state showed lower pain tolerance and greater pain catastrophizing than did participants in the neutral condition. Induced depressed mood did not, however, affect other aspects of the pain experience, including threshold, unpleasantness ratings, and MPQ-Sensory and Evaluative scores.

Our findings in this study are consistent with previous investigations that failed to find increased pain sensitivity in depression<sup>18–23</sup>; at the same time, they appear to replicate Zelman and associates' <sup>27</sup> finding that depressed mood state is associated with decreased tolerance for cold-pressor pain. Thus, our results may provide support for the idea that depressed mood has an influence on behavioral reaction to pain without actually enhancing sensitivity to pain.

The finding that individuals in the depressed mood state group showed greater catastrophizing during the pain procedure is particularly interesting because, to our knowledge, this is the first laboratory experiment to examine whether depressed mood directly influences degree of pain catastrophizing. This finding suggests that depressed mood may actually affect the way an individual thinks about pain. Thus, just as a clinically depressed individual tends to experience more negative and extreme cognitions about events

in general, as described in Beck's cognitive theory of depression,<sup>44</sup> an individual in a depressed mood may experience more negative and extreme cognitions about a specific pain experience. This supposition may also be viewed as consistent with Fields's<sup>29</sup> theory concerning the relationship between pain and depression, which holds that catastrophizing mediates the relationship between depression and the affective and evaluative experience of pain. Readers should recognize that we examined depressed mood and not clinical depression in the present study. Also, our results do not provide direct support for this theory because we did not examine a cause and effect relationship between catastrophizing and pain measures. Nevertheless, our results do provide some support for an assumption on which this theory is based, which is that depression is associated with greater catastrophizing about pain.

We expected but did not find the lack of significant differences between the mood conditions on measures of affective intensity (unpleasantness ratings and MPQ-A). The prediction that depressed mood would increase affective intensity was based on clinical research that revealed higher affective pain ratings in depressed patients with clinical pain syndromes relative to nondepressed pain patients.24 One may speculate that the failure to find these effects in a laboratory experiment suggests that an affective response to experimental pain is not affected by mood in the same way that affective response to clinical pain is affected or that induced depressed mood does not have the same effect as clinical depression on affective ratings of pain. It should be noted, however, that the nonsignificant trend in the predicted direction for MPQ-A, such that MPQ-A appeared greater for the depressed condition than for the neutral condition. Therefore, it is possible that this trend would have been significant if the statistical power had been greater.

Because we found no significant effects for the measures of affective intensity of the pain, we cannot conclude that depressed mood influenced affective response to the pain. What appears to be the most reasonable conclusion on the basis of the data is that depressed mood was associated with altered behavioral (lower tolerance) and cognitive responses (greater catastrophizing) to the pain experience. We used a controlled laboratory experiment in the investigation and therefore any clinical inference based on the results is clearly speculative. Nevertheless, this study may have implications for clinical practice by providing additional evidence for a negative influence of depressed mood on certain aspects of pain response.

Several limitations of this study are noteworthy. First, although the experimental nature of the work is considered

a strength because it allowed for causal conclusions about the influence of mood on pain responses, the fact that we used experimentally induced mood and experimental pain calls into question the degree to which the results may be generalized to situations involving clinical pain and clinical depression. Induced depressed mood is clearly not an exact replication of clinical depression; therefore, it is not a certainty that clinical depression or even depressed mood not resulting from induction would produce the same effects on pain that induced depressed mood produced. Similarly, the laboratory cold-pressor pain, although considered a useful simulation of clinical pain, may be experienced somewhat differently from clinical pain conditions. For example, coldpressor pain is probably less threatening than many pain conditions that are associated with serious injury or illness, or that are of unknown origin. Cold-pressor pain is also time limited, whereas clinical pain is of uncertain duration.

We hoped that 3 distinct groups would be produced by the mood induction procedure (ie, depressed, neutral, and elated). Unfortunately, although the manipulations for depressed and neutral mood state produced 2 distinct groups on the mood-state measure, we did not successfully produce an elated group. Future researchers in this area may consider using an alternative procedure for inducing elated mood (eg, music mood induction).

The study also has limitations related to the measures used to evaluate responses to the cold-pressor task. As could be expected when responses to laboratory pain are evaluated, (eg, Chen et al<sup>45</sup>) a high degree of variance was observed on some of the measures, particularly tolerance time and MPQ-A scores. This high variance, in conjunction with relatively small sample sizes, decreased statistical power. In addition, we slightly altered the instructions for 2 of the measures (MPQ and PCS) for this experiment so that they could be used to evaluate response to laboratory pain rather than to clinical pain. Of particular note is our use of the PCS to examine degree of catastrophizing during the painful procedure rather than evaluating pain catastrophizing as a trait. In the future, researchers should administer the PCS as a trait measure before performing any manipulations to determine whether there are between-group differences on this trait that may influence catastrophizing during the procedure.

Finally, we were somewhat concerned that demand characteristics would influence our results. To minimize this problem, we kept all research assistants blind to the purpose and hypotheses of the experiment. In addition, we administered a postexperiment questionnaire to participants to determine whether they were aware of the purpose or hypotheses of the experiment. Of the 75 original participants, only 9 made somewhat accurate guesses about the

general purpose of the experiment, and only 3 of those participants accurately guessed 1 of the hypotheses.

These findings underscore the importance of evaluating multiple aspects of pain response in experimental and clinical research investigating the influence of various factors on pain. Had this experiment evaluated only pain sensitivity or intensity, it could have appeared that laboratory-induced depressed mood was associated with no difference in pain response, compared with neutral mood. However, in this experiment, we considered differences in behavioral and cognitive responses, which may be more important than pain intensity when clinical inferences are made.

More research is needed to examine further the effect of depressed mood and clinical depression on catastrophizing about experimental and clinical pain. A particularly interesting study, from which clinical inferences could be more confidently made, would examine the influence of clinical depression on cognitive and behavioral responses to a clinical pain stimulus. For example, depressed patients could be compared with nondepressed patients on degree of catastrophizing during a painful medical procedure (eg, mammography) or after a pain-producing procedure (eg, surgery).

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#### NOTE

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Vol 28, Spring 2002 29

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All materials are to be sent to the Secretary of the Academy by February 1, 2003:

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