Chronic pain and depression:
toward a cognitive-behavioral mediation model

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Summary   Although considerable attention has recently been devoted to explaining why depression is a frequent concomitant of chronic pain, little empirical work has been conducted to test predictions based on these models. The present study was designed to test a cognitive-behavioral mediation model of pain and depression that proposes perceived reduction in instrumental activities along with a decline in perceptions of control and personal mastery are necessary prerequisites for the development of depressive symptomatology in pain patients. According to this model, in contrast to alternative models, the presence of pain is not sufficient condition for the subsequent development of depression. This model was tested and confirmed through the application of structural modeling with latent variables. Specifically, the direct link between pain and depression was found to be non-significant, however, measures of perceived life interference and self-control were found to be significant intervening variables between pain and depression. These results provide the first empirical demonstration that psychological mediators may be involved in the development of depression secondary to chronic pain. The findings of this study are contrasted with single-factor models that postulate both chronic pain and depression as resulting from a common cause.

Key words: Chronic pain; Depression; Mediation model

Introduction

Depression has been documented as a frequent concomitant of chronic pain (see reviews by Turner and Romano [60]; Roy et al. [49]). Reports of the incidence of depression among pain patients have ranged from 10% [45] to 100% [58], however, the majority of studies report the coexistence of depression in over 50% of chronic pain patients sampled [e.g., 28,31,37,63]. Although several theo-

retical models have been developed to describe or explain the apparent relationship between these clinical problems, surprisingly little empirical work has been conducted to test the relationships predicted by these models.

Three general models for conceptualizing the relationship between chronic pain and depression have been proposed. Non-specific biological models hypothesize that similar neurochemical mechanisms may be involved in both disorders. For example, abnormalities in the amount, turnover, or ratio of biogenic amines (e.g., catecholamines, serotonin) and endogenous opioids (e.g., β-endorphins) have been postulated to contribute to both depressive disorders and chronic pain [e.g., 20,62]. The demonstrated efficacy of tricyclic anti-

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depressants in the treatment of chronic pain is often cited as providing preliminary support for these models (see reviews by France et al. [17]; Lee and Spencer [34]).

A second model has been described by Engel [12] and recently extended by Blumer and Heilbronn [7]. These authors suggest that chronic pain in the absence of demonstrable organic pathology should be viewed as a variant of depressive disease. To support this model, authors describe the 'pain-prone patient' and cite reports that these patients manifest psychodynamic features similar to the 'depression-prone' individual [40,44]. Studies are also cited that report a higher than expected frequency of depression among relatives of a sample of pain patients [50], implying a genetic or learning basis for both disorders. This model has drawn considerable interest, but has been challenged as lacking empirical support [48,55]. Based on the results of the dexamethasone suppression test, France and his colleagues [16,18] have criticized the validity of this model. They suggest that viewing chronic pain as a variant of depression may be an oversimplification. Most recently, Large [33] evaluated 50 consecutive patients with chronic pain using DSM III nosology and also concluded that the case for viewing 'pain proneness' as a distinct psychobiological disorder could not be confirmed.

Finally, depression has been frequently explained as an understandable, even expected, secondary reaction to a chronic and incapacitating physical condition [e.g., 20,52]. A model integrating behavioral [14,36] and cognitive [4,47] formulations of depression has been proposed to explain the development of depression as a reaction to pain among a subset of chronic pain patients [28,53,55]. Generally, this model hypothesizes that depression is a function of a sustained reduction in instrumental activities and a concomitant decline in important social rewards, as well as a decline in perceptions of control over reinforcement contingencies and personal mastery.

The basis for the cognitive-behavioral mediation model comes from research on the contribution of cognitive and behavioral factors involved in the pain experience. Empirical support for the role of maladaptive thinking as a mediating variable has been reported by Lefebvre [35], who demonstrated a high frequency of cognitive distortions among depressed pain patients. Non-depressed pain patients, however, did not demonstrate the presence of these cognitive distortions. Declines in social, recreational, vocational, and domestic activities have long been documented to occur among chronic pain patients and have become a primary target in behaviorally oriented treatment programs [e.g., 15,56].

In the present paper, we assess a specific cognitive-behavioral model designed to predict the extent or degree of depressive symptomatology among a heterogeneous sample of chronic pain patients (see Fig. 1). According to this model, the experience of chronic pain is an insufficient condition for the development of depression. That is, the direct link between pain and depression should be small and minimally useful in accounting for the relationship between these two syndromes. Rather, the perceptions of significant declines in instrumental behavior and associated declines in life satisfaction, termed life interference, and lower perceptions of self-control, are hypothesized to act as necessary mediators or indirect links between the experience of pain and depressive symptoms. The methodology of structural modeling with latent variables (described below) is utilized because it permits a direct test of the degree of congruence between data provided by chronic pain patients and the predictions made by the proposed cognitive-behavioral model.

Method

Subjects

The participants in the study were selected from 127 consecutive referrals to the outpatient Pain Management Program at the West Haven, Connecticut VA Medical Center. Each referral was screened by examination of the patient's medical record, physician consultation, and a brief interview with the patient. The inclusion criteria were: (a) duration of pain 6 months or longer, (b) chronic pain other than headache or related to cancer, and (c) no evidence of active psychosis or acute suicidal risk.

Of 127 patients screened, 100 patients met the above inclusion criteria and provided complete data. The mean duration of pain was 10.7 years (range 6 months–40.6 years). The nature of the primary pain syndrome was quite varied (e.g., trigeminal neuralgia, tendenitis, herniated disc, rheumatoid arthritis), with the largest group of patients (36%) reporting low back pain. The mean age of patients was 50.8 (S.D. = 14.5), 78% were male, 66% were currently married, 56% had had at least one pain-related surgery, 52% were receiving disability compensation, and 67% were taking prescribed analgesic medications.

Measures

Pain severity. Three measures were used to assess current pain intensity. These measures were (a) the total score of the Pain Rating Index from the McGill Pain Questionnaire [MPQTOTAL; 43,57], (b) the Pain Severity Scale of the West Haven-Yale Multidimensional Pain Inventory [WHYMPI-Pain Severity; 29], and (c) the mean from 2 weeks of hourly self-monitoring of perceived pain intensity [PAINCARDS; 56]. This self-monitoring of pain intensity was based on a procedure adapted from Budzynski et al. [9]. Patients were instructed to keep hourly records of their levels of pain on a 6-point scale that ranged from 0, 'no pain,' to 5, 'severe pain.' For each patient, a mean hourly pain intensity rating was derived for all hours coded during the 2 week assessment period.

Depression. Level of depressive symptomatology was measured by 2 standard instruments, the Beck Depression Inventory [BDI; 5] and the Depression Adjective Check List [DACL; 41].

Interference. The amount of interference with life activities attributed by patients to pain was assessed with 3 scales from the WHYMPI [29]. These scales were the Social, Work, and Family Interference Scales (WHYMPI-Social, -Work, -Family), comprised of questions related to how much patients feel pain has affected their ability to participate in social and recreational activities, vocational activities, and family and domestic activities, as well as questions related to the amount of satisfaction that they derive from these activities.

Self-control. Patients' perceptions of self-control were measured by (a) items from the WHYMPI related to how much control they felt they had over their life during the past week (WHYMPI-Life Control), (b) how much they felt they had been able to solve their problems during the past week (WHYMPI-Problem Solving), and (c) the Internal Subscale from the Multidimensional Health Locus of Control [MHLC-Internal; 61].

In total, 11 measures were used. Previous psychometric analyses have indicated that each of these scales is reliable, with coefficient alphas greater than or equal to 0.70 and/or test-retest correlations of greater than or equal to 0.65. All summated scales and items were scored in the same direction, with higher scores indicating more depressive symptomatology, pain severity, perceived interference, and self-control.

Procedure

The scales used in this study were administered as part of a comprehensive assessment and treatment program offered by a hospital-affiliated outpatient pain clinic. Although the assessment instruments were self-administered, the clinicians conducting the assessment were available to respond to patients' questions.

Data analysis

The product-moment correlation matrix of the 11 measured variables was used to conduct maximum-likelihood structural modeling with latent variable procedures. The mathematical basis for these procedures is discussed by Joreskog [23] and good discussions of conceptual and technical issues are presented by Kenny [27] and Long [38,39].

The LISREL-V computer program [24] was used to obtain the maximum-likelihood estimates of all model parameters and a chi-square goodness-of-fit statistic. A normed goodness-of-fit index (Δ) suggested by Bentler and Bonett [6] was also computed. The delta index, which ranges from 0 to 1 (1 = the model completely explains the observed data), describes the degree of fit of a specific structural model relative to a given baseline. Although several types of baselines may be conceptually appropriate [6], the most frequently

used is a null model that postulates no covariation among the variables. In terms of the present study, the null model hypothesis was that depressive mood, pain severity, interference, and self-control measures were *independent*. Thus, this index estimated the improvement in variance accounted for by our specific *a priori* pain and depression model over a null model that assumed there were no common factors or interrelationships.

Specification of the cognitive-behavioral mediation model. Recent developments in the methodology of structural modeling with latent variables permit a direct test of the cognitive-behavioral mediation model of pain and depression described earlier. In contrast to the most popular form of 'causal' modeling, path analysis, structural models within the framework suggested by Joreskog [23] embed factor analysis within the path-analytic tradition. A major strength of this approach is that multiple indicators can be used to separate an error-free latent variable from the measurement error present in each of the observed variables [42]. Thus, unlikely many statistical approaches that view single measures and dimensions as iden-

tical (e.g., multiple regression analysis), measures within this framework no longer have to be pure indicators of theoretical variables because only the shared variance of measures within a dimension is extracted.

Structural modeling with latent variables consists of two portions that are estimated simultaneously. One portion, the measurement model, specifies the relationships between the measured or observed variables (e.g., scores on the BDI and DACL) and the latent (unobserved) variables (e.g., depressive symptomatology), the result of which is a set of factor scores. Thus, although conceptually similar to the factor analytic approach, the measurement portion of a structural model is also confirmatory in that relationships between measures and factors need to be defined *a priori* based on a theoretical rationale.

The second portion, the structural-relations or path model, specifies the pattern of relationships or influences among the latent variables. If no directionality is hypothesized for these relationships, then the structural model represents a confirmatory factor analytic model and, provided the
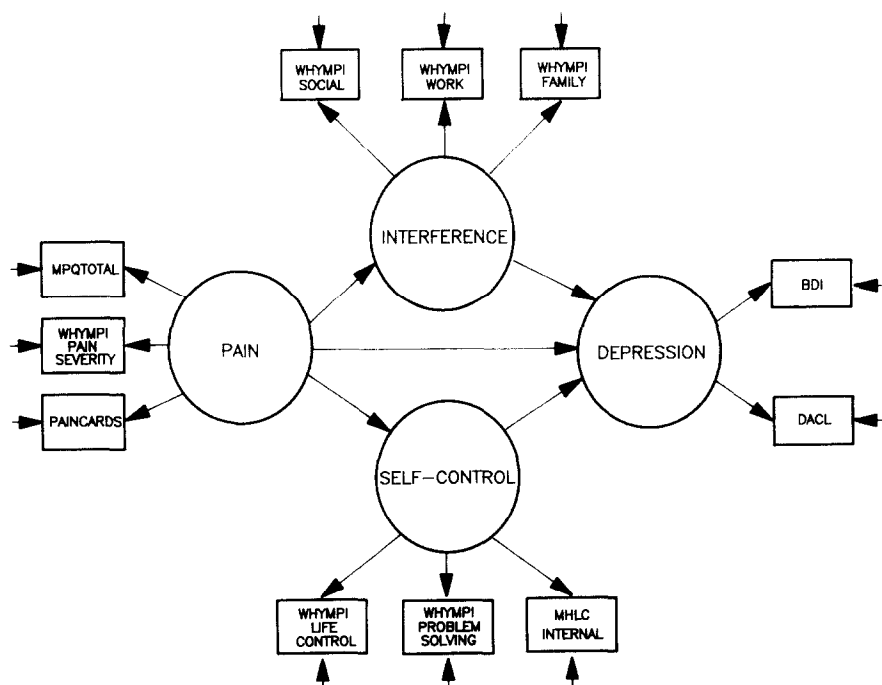

Fig. 1. Hypothesized cognitive-behavioral mediation latent variable structural model of chronic pain and depression.

measures or solution have been standardized, the coefficients between the latent variables are interpreted as correlations. If directionality or causal influences are specified among the latent variables, then the resulting parameters are regression coefficients. A further strength of this methodology is that a particular structural model can be a combination of hypothesized causal links, non-causal links, or no links at all between latent variables.

The structural model used to test the cognitive-behavioral mediation model of pain and depression is displayed in Fig. 1. For the measurement portion of this model, 4 factors or latent variables were hypothesized — Pain, Interference, Self-Control, and Depression. These factors are shown as circles in Fig. 1. The boxes in Fig. 1 indicate that the Pain Severity, Life Interference, and Self-Control factors were each operationalized with 3 measured variables and that 2 measures were used to operationalize the Depression factor. The factor loadings are shown as arrows originating at factors and pointing toward the measured variables. The other set of unidirectional arrows pointing toward measured variables denote the unique, residual variance (e.g., measurement error) for each observed variable.

The structural-relations portion of this model is shown in Fig. 1 as unidirectional arrows originating at one latent variable and pointing at another latent variable. As displayed in Fig. 1, Pain Severity, based on cognitive-behavioral theories of depression, was hypothesized to lead to increases in Life Interference, which in turn should increase a patient's level of depressed mood. Pain Severity was also predicted to have an inverse affect on a patient's perceived Self-Control in that, as a result of this decline in Self-Control, it should increase a patient's level of depressive symptoms.

To summarize the structural relationships depicted in Fig. 1, we hypothesized that the regression coefficients between Pain and Interference and between Interference and Depression should both be positive, and that the coefficients between Pain and Self-Control and Self-Control and Depression should both be negative. Additionally, although a direct link between Pain and Depression is shown in Fig. 1, we hypothesized that this link would not be significantly different from zero. In other words, our prediction was that pain, in and of itself, would not be a sufficient condition to predict depressed mood.

Results

The means, standard deviations, coefficients of kurtosis, and product-moment correlations for the

TABLE I

MEANS, STANDARD DEVIATIONS, COEFFICIENTS OF KURTOSIS, AND PRODUCT-MOMENT CORRELATIONS FOR THE MEASURED VARIABLES

Scale	1	2	3	4	5	6	7	8	9	10	11
1. BDI											
2. DACL	0.42										
3. PAINCARDS	−0.03	0.01									
4. MPQTOTAL	0.09	0.11	0.29								
5. WHYMPI-Pain Severity	0.23	0.18	0.52	0.44							
6. WHYMPI-Work	0.25	0.21	0.23	0.30	0.53						
7. WHYMPI-Social	0.28	0.11	0.31	0.26	0.46	0.74					
8. WHYMPI-Family	0.36	0.29	0.25	0.26	0.41	0.53	0.64				
9. WHYMPI-Life Control	−0.54	−0.30	−0.01	−0.18	−0.15	−0.12	−0.18	−0.30			
10. WHYMPI-Problem Solving	−0.53	−0.41	−0.03	−0.14	−0.28	−0.11	−0.20	−0.35	0.71		
11. MHLC-Internal	−0.18	−0.06	−0.09	−0.10	−0.27	−0.13	−0.10	0.03	0.23	0.31	
Mean	14.26	13.41	2.63	0.39	3.60	3.95	4.26	3.00	3.68	3.61	22.83
S.D.	8.78	5.37	1.09	0.20	1.16	1.50	1.59	1.45	1.78	1.68	6.32
Kurtosis	1.56	1.67	2.01	2.40	2.35	1.55	1.95	2.85	1.84	1.99	2.34

Note. Correlations $\geqslant 0.19$ or $\leqslant -0.19$ are significant at or below the 0.05 level.

measured variables used in this study are shown in Table I. As can be seen, all correlations among the indicators for each of the 4 latent variables (enclosed with triangles in Table I) were statistically significant. The summary statistics displayed in Table I indicated that approximately 50% of these pain patients reported at least moderate levels of depressive symptomatology and pain (scores above the midpoints on pain severity scales). Additionally, the coefficients of kurtosis contained in Table I indicate that none of these measures displayed a kurtose distribution, which can seriously affect the maximum-likelihood estimators and significance testing used in structural modeling with latent variables approaches [8].

The correlation matrix for the measured variables (Table I) was used to test the cognitive-behavioral model of pain and depression depicted in Fig. 1. The overall chi-square goodness-of-fit statistic indicated that this model had a satisfactory level of statistical fit to these data, χ^2 (39) = 49.59, P = n.s. Additionally, the Bentler–Bonett [6] normed index, with the null model employed as the baseline, was computed to be 0.91. Thus, the

goodness-of-fit indices demonstrated that not only did the cognitive-behavioral model statistically fit these data, but also explained a substantial proportion of the total covariation among the observed measures.

The parameter estimates (factor loadings) and standard errors for the measurement portion of this structural model are shown in Table II. Examination of this table indicated that the Pain, Self-Control, Interference, and Depression constructs (latent variables) were adequately operationalized by the measured variables because (a) all factor-loading estimates were statistically significant and (b) the correlations between a measured variable with its latent construct were high. Thus, based on the loadings displayed in Table II, we could infer that our hypothesized factors were measured appropriately by the indicators employed.

The estimates for the structural-relations portion of the cognitive-behavioral mediation model are presented in Table III. The standardized regression coefficients are also shown and are analogous to standardized regression coefficients in path

TABLE II

PARAMETER ESTIMATES AND STANDARD ERRORS FOR THE MEASUREMENT PORTION OF THE COGNITIVE-BEHAVIORAL PAIN AND DEPRESSION STRUCTURAL MODEL

Measure	Unstandardized loading	S.E.	Standardized loading
Pain			
WHYMPI-Pain Severity	1.000 [a]	0.000 [a]	0.888
PAINCARDS	0.644 **	0.116	0.572
MPQTOTAL	0.568 **	0.119	0.504
Interference			
WHYMPI-Social	1.000 [a]	0.000 [a]	0.885
WHYMPI-Work	0.936 **	0.108	0.828
WHYMPI-Family	0.796 **	0.111	0.704
Self-Control			
WHYMPI-Problem Solving	1.000 [a]	0.000 [a]	0.898
WHYMPI-Life Control	0.880 **	0.125	0.790
MHLC-Internal	0.365 *	0.124	0.327
Depression			
BDI	1.000 [a]	0.000 [a]	0.774
DACL	0.681 **	0.169	0.527

[a] Parameter fixed at indicated value during estimation.
* $P < 0.01$; ** $P < 0.001$.

TABLE III

PARAMETER ESTIMATES AND STANDARD ERRORS FOR THE STRUCTURAL-RELATIONS PORTION OF THE COGNITIVE-BEHAVIORAL PAIN AND DEPRESSION STRUCTURE MODEL

Dependent latent variable	Independent latent variable			
	I	II	III	IV
Unstandardized coefficients [a]				
I. Pain				
II. Interference	0.63 (0.11) **			
III. Self-Control	−0.31 (0.12) *			
IV. Depression	−0.13 (0.15)	0.29 (0.14) *	−0.66 (0.12) **	
Standardized coefficients [a]				
I. Pain				
II. Interference	0.64			
III. Self-Control	−0.31			
IV. Depression	−0.13	0.33	−0.76	
Inter-factor correlations				
I. Pain	1.00			
II. Interference	0.64	1.00		
III. Self-Control	−0.31	−0.20	1.00	
IV. Depression	0.30	0.39	−0.78	1.00

Note. Numbers in parentheses are standard errors.

[a] Regression coefficients fixed at zero are not shown.

* $P < 0.01$; ** $P < 0.001$.

analysis. These standardized coefficients indicate the relative magnitude of the direct effects between the different latent variables. In terms of Fig. 1, these coefficients are the values attached to the unidirectional arrows originating at one latent variable (circle) and pointing toward another. As displayed in Table III, all hypothesized regression coefficients among the latent variables were statistically significant *except* the direct path between the Pain and Depression factors. As predicted by the cognitive-behavioral mediation model, the coefficients linking Pain with Interference and Interference with Depression were in the positive direction. Also, as predicted, the coefficients linking Pain with Self-Control and Self-Control with Depression were found to be negative. To summarize, these findings indicated that increases in Pain Severity significantly predicted both increases in Life Interference and decreases in perceived Self-Control, which together significantly predicted an increase in depressive symptomatology.

Assessing the explanatory power of the cognitive-behavioral mediation model

Although the cognitive-behavioral mediation model of pain and depression was confirmed by the obtained data, several additional statistical indices helped to establish the overall quality or explanatory power of this model. They were (a) the coefficient of determination and (b) the total 'causal' effects of Pain and Depression. Taken together, these indices allowed us to assess how well the cognitive-behavioral mediation model explained the relationship between pain and depressed mood.

In classical regression theory, the coefficient of determination is defined as the percentage of the variation in the dependent variable that is explained by the regression. Because the primary purpose of the present study was to clarify the relationship between pain and depressive symptomatology in chronic pain patients, we were most concerned with how much variance in the Depression factor could be accounted for by the Pain,

Interference, and Self-Control factors. The LISREL program indicated that 68.1% of the variance in the Depression Factor could be accounted for by employing these three factors as predictors. Further, the residual variance for this structural equation was found to be non-significant, $\zeta = 0.19$, S.E. $= 0.13$, $z = 1.45$. Thus, although additional predictors of depressed symptomatology in pain patients could be hypothesized, the Pain Severity, Interference, and Self-Control factors appeared to be sufficient predictors of the Depression factor.

The correlation between 2 latent variables in a structural model can be decomposed into 2 basic components. These are (a) total 'causal' effects, which are composed of both direct and indirect effects and (b) unanalyzed association or 'spuriousness.' In terms of the present study, we were particularly interested in assessing the degree to which the cognitive-behavioral mediation model accounted for the correlation between the Pain and Depression factors.

The latent variable correlations are presented in Table III. The product-moment correlation between the Pain and Depression factors was computed to be 0.30. Using an option provided in the LISREL program, the total 'causal' effect of pain on Depression was calculated to be 0.26. This suggests that only a small component (0.04) of the total correlation between Pain and Depression was not explained by the cognitive-behavioral model. Thus, rather than being spuriously related to the Depression and Pain factors, the Self-Control and Interference factors appear to be intervening variables that accounted for a large proportion of the association between the Pain and Depression factors.

Although implicitly contained in the LISREL results reported above, it can also be demonstrated that the Self-Control and Interference factors are, indeed, intervening variables by examining partial correlations among the factors. Based on path-analytic models originally proposed by Simon [51], if the Self-Control and Interference are truly intervening variables between Pain and Depression, then the partial correlation between Pain and Depression, controlling for Self-Control and Interference, should be approximately zero. This statistical condition was confirmed, the corre-

lation between the Pain and Depression factors controlling for the effects of Self-Control was found to be 0.06, and the correlation between Pain and Depression controlling for Interference was 0.05.

Discussion

The results of this study provide initial support for a cognitive-behavioral mediation model of depressive symptomatology among chronic pain patients. Specifically, this model predicted that the direct relationship between pain and depressed mood would be non-significant and that the cognitive appraisal variables of perceived interference and lack of self-control would be significant mediators of the pain-depression relationship. Consistent with many other studies [e.g., 31,37], the prevalence of significant levels of depressed mood among chronic pain patients was present in approximately 50% of our sample. Although a Pearson correlation revealed a modest yet significant association between pain and depressive symptomatology ($r = 0.30$), through the use of structural modeling we were able to determine that the direct link between pain and depression was not significant, rather, the association between pain and depression was significantly mediated by perceived interference and lack of self-control. The structural equation that consisted of pain, perceived interference, and perceived lack of self-control accounted for over 68% of the variance in depressed mood.

The results described provide the first direct empirical demonstration that psychological mediators may be involved in depressive symptomatology among chronic pain patients. The cognitive-behavioral model tested can be contrasted with Blumer and Heilbronn's [7] 'pain-prone' model. Blumer and Heilbronn proposed that the prevalence of depression among chronic pain patients is based on a predisposition among certain individuals to develop chronic pain as a manifestation of an underlying depression. More specifically, their model would predict that pain and depression measures should be highly correlated. The data presented in this paper are contrary to

what would have been predicted from the view of pain-proneness as a distinct psychobiological entity as proposed by Blumer and Heilbronn.

An alternative single-factor model, derived from biological investigations, has been proposed by Ward et al. [62]. These investigators hypothesized that imbalances in CNS biogenic amines may produce depression as well as modulate pain perception. These imbalances may also either directly or indirectly affect endogenous opioid production (e.g., β-endorphins), known to have pain-modulating functions and potential involvement in depression [19]. This model also predicts that measures of pain and depression should be highly correlated. The results of the present study failed to provide support for either of the single-factor models described above, as the direct association between pain and depression was shown to be non-significant. Nonetheless, it is possible that biochemical or pre-existing psychological factors may be associated with the experience of pain in a subset of chronic pain patients.

Our cognitive-behavioral mediation model suggests that depressed mood among chronic pain patients can be parsimoniously explained by patients' appraisals of the degree to which pain interferes with important areas of functioning and perceptions of self-control. Adapting Lewinsohn's [36] behavioral model of depression, chronic pain patients may be expected to become depressed as they experience declines in response-contingent reinforcement associated with declines in the frequency and range of instrumental activities. Thus, as patients leave the work place, withdraw from social and recreational activities, and limit their activities in the home and with their families, they may be expected to experience declines in the level of contingent rewards in their lives.

As attempts at pain relief fail and as efforts to continue or resume activities are unsuccessful, it is understandable that perceptions of helplessness and reduced self-control evolve. Cognitive models of depression emphasizing the importance of attributions of helplessness [1] and reduced self-control [47] have been articulated and supported empirically. Results of the present study support the idea that a relatively global perception of lowered self-control may be an important mediator of the development of depression among chronic pain patients. Furthermore, the appraisal of self-control was found to be relatively independent of the appraisal of pain interference, suggesting that each variable should continue to be examined for its theoretical as well as clinical significance. Rehm's [47] adaptation of Kanfer's [25] self-control model may prove useful in further understanding the relationship between self-control and depression. Specifically, to what extent do pain patients manifest deficits in self-monitoring, self-evaluation, or self-reinforcement related to their appraisals of their pain and events in their lives and to what extent do these specific deficits contribute to the experience of depression?

It is important to note that not all pain patients report significantly reduced activity levels or diminished satisfaction with their lives. Several alternative reasons for patients' reports must be considered. First, some of these patients may be accurately reporting that their level of participation in and satisfaction with various aspects of their lives has not changed as a function of pain. Other patients may in fact have realized declines in one or more areas (e.g., disability, retirement), but may have compensated for these changes by developing new sources of reinforcement (e.g., improved family relationships). Alternatively, patients may objectively demonstrate declines in their behavioral repertoire, but continue to perceive themselves as functioning at an acceptable level. Thus, they may maintain the 'illusion of control' [32]. Although it may prove useful to distinguish among these and other alternatives by means of direct behavioral analysis, the theoretical and clinical utility of patients' reports of their subjective experience of the pain problem and its impact on their lives cannot be underemphasized [54,56]. Indeed, results of the present study suggest that additional inspection of patients' idiosyncratic appraisals of their pain problem and its effects may prove to be extremely important in the evaluation and treatment of coexisting depression [53].

Several potential limitations of this study should be acknowledged. The measures of depressive symptomatology employed were based on well-established, but nonetheless self-report measures (BDI and DACL). However, the BDI has been

shown to be significantly correlated ($r = 0.80$) with other diagnostic criteria of depression based on interviews [e.g., 13], and both the BDI and DACL have been found to be significantly correlated (all $rs > 0.65$) with the DSM III criteria for major depression [10,60]. Future research should confirm the relationships identified in the present study by cross-validating the findings with additional measures of depression.

A second potential limitation of these results concerns the sample of patients included in the study. The majority of patients in this study were patients at a Veterans Administration Medical Center. It might be argued that this sample is atypical and not representative of the more general population of pain patients. In a recent study, however, Holzman et al. [22] demonstrated that pain patients at 2 typical VAMCs were not significantly different from pain patients at 2 non-VA hospitals on any demographic or treatment variables with the exception of gender. Demographic and pain-related data from the present sample indicate that the subjects in this study appear to be comparable to other pain patients [e.g., 11,26].

A third limitation of these results is that although path analysis and other structural modeling approaches are often employed to assess 'causal' relationships, the data most frequently employed are based on concurrent correlational findings. Thus, strictly speaking the results of this study cannot be used to conclude that higher levels of pain 'caused' increased perceptions of life interference and decreased perceptions of self-control that together then 'caused' the development of depressive symptomatology. The structural modeling methodology employed, however, does allow the investigator to test the plausibility of competing theoretical models with the potential to demonstrate the inadequacies of some theories [42]. Although longitudinal structural modeling with latent variable approaches [e.g., 2,3] may be useful in more directly assessing the causal relationships between chronic pain and depressive symptomatology, ethical considerations (withholding treatment for a sufficiently long period of time for a large number of patients) makes this type of methodology prohibitive in clinical settings.

The results of this study are consistent with a cognitive-behavioral formulation of depression that emphasizes patients' appraisal of their current life situation. This perspective has important implications for both assessment of and interventions with chronic pain patients as it emphasizes the need to assess and directly address psychological variables, specifically patients' perceptions of their plight, in addition to attention to physiological factors [54]. A comprehensive cognitive-behavioral approach for the treatment of chronic pain patients has been described by Turk et al. [56]. Results from clinical studies with heterogeneous samples of pain syndromes (e.g., headache, low back pain, and arthritis) support the efficacy of this approach in treating pain patients [e.g., 21,30,46,59]. Despite the interest in employing cognitive-behavioral treatments with pain patients, few attempts have been made to test the theoretical rationale of these interventions for chronic pain. The present study provides preliminary support for the theoretical model upon which cognitive-behavioral interventions for chronic pain are predicated. Additional research is needed to provide further support for the assumptions implicit in cognitive-behavioral models of pain treatment.

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