

# Health Status, Cognitive Coping, and Depressive Symptoms: Testing for a Mediator Effect

MATTHEW P. MARTENS, JERRY C. PARKER, KAREN L. SMARR, JAMES E. HEWETT, BIN GE, KATHLEEN DONOVAN HANSON, JAMES R. SLAUGHTER, and SARA E. WALKER

**ABSTRACT. Objective.** Research has established a link between health status and symptoms of depression in persons with rheumatoid arthritis (RA), but the effects of “cognitive coping” variables have not been extensively studied. We examined the mediator effect of a cognitive coping variable (Pain Control and Rational Thinking factor score from the Coping Strategies Questionnaire) over the course of a pharmacological intervention.

**Method.** Data were analyzed from 54 persons with RA, all of whom met diagnostic criteria for major depression. Measures of depression, health status, and cognitive coping were collected at 4 different stages of a pharmacological (antidepressant) study as follows: (1) at baseline, (2) postintervention, (3) 6 month followup, and (4) 15 month followup.

**Results.** Results indicated that a direct relationship existed between health status and depression at all 4 time periods. However, this relationship was mediated by cognitive coping only at the postintervention and the 6 month followup.

**Conclusion.** A cognitive coping variable was found to mediate the relationship between health status and depression, but only at moderate levels of depression. (J Rheumatol 2005;32:1584–8)

## Key Indexing Terms:

HEALTH STATUS  
MEDIATOR EFFECT

PAIN

DEPRESSION  
RHEUMATOID ARTHRITIS

Depression is a common symptom in the US population<sup>1</sup>. For men the point prevalence of major depression ranges from 2% to 3%<sup>1</sup>; for women, the point prevalence ranges from 4% to 9%<sup>1</sup>. Chronic diseases, such as rheumatoid

arthritis (RA), have been identified as risk factors for depression<sup>2</sup>. Yet the true interrelationships among variables such as depression, pain, coping, health status, and chronic disease are not precisely known, and the directionality of these relationships is difficult to unravel. Experiments using randomization and the isolation of specific variables are not possible. Thus, research on the interrelationships among depression, pain, coping, health status, and chronic disease (among other potentially associated variables) requires theory-driven hypotheses and a careful conceptual analysis of the existing literature.

This study focuses on the core relationship between depression and health status and whether “cognitive coping” variables might mediate this relationship. As noted, none of these 3 variables (depression, health status, or cognitive coping) can be isolated in the context of a true experiment. Hence a path analysis approach was used in these analyses. As recommended by Jöreskog and Sörbom<sup>3</sup>, the strength of proposed models must be argued on the basis of theory or empirical evidence because equivalent models, given the same number of parameters, would yield the same goodness-of-fit measures.

With regard to the relationship between depression and health status, causality might be argued in either direction. In support of depression as the predictor variable, Anderson, *et al*<sup>4</sup> found that symptoms of depression were predictors of functional status in a sample of persons with RA. Similarly, Beckham, *et al*<sup>5</sup> reported that depression was a significant predictor of behavioral dysfunction in persons with RA. In

---

*From the Research Service, Harry S Truman Memorial Veterans' Hospital; Department of Physical Medicine and Rehabilitation; Department of Psychiatry; Department of Family and Community Medicine; Department of Immunology and Rheumatology, University of Missouri-Columbia, Columbia, Missouri; Department of Educational and Counseling Psychology, State University of New York, University at Albany, Albany, New York; and Department of Institutional Research, Buena Vista University, Storm Lake, Iowa, USA.*

*Supported in part by grants from the National Institute on Disability and Rehabilitation Research (NIDRR) of the US Department of Education under grant H133B30039; and from Pfizer, Inc. Support was also received from the Medical Research Service of the Department of Veterans Affairs.*

*M.P. Martens, PhD, Assistant Professor, Department of Educational and Counseling Psychology, State University of New York, University at Albany; J.C. Parker, PhD, Associate Chief of Staff, Research and Development, Harry S Truman Memorial Veterans' Hospital, Clinical Professor, Departments of Physical Medicine and Rehabilitation and Psychiatry, University of Missouri-Columbia; K.L. Smarr, PhD, Clinical Assistant Professor, Department of Psychiatry, University of Missouri-Columbia, Staff Psychologist, Harry S Truman Memorial Veterans Hospital; J.E. Hewett, MA, Director of Institutional Research, Buena Vista University; B. Ge, MD, MA, Statistician; K. Donovan Hanson, MEd, Research Associate; J.R. Slaughter, MD, Clinical Associate Professor, Family and Community Medicine; S.E. Walker, MD, Professor, Department of Medicine, Department of Immunology and Rheumatology, University of Missouri-Columbia.*

*Address reprint requests to Dr. J.C. Parker, Research Service, Harry S Truman Memorial Veterans' Hospital, 800 Hospital Drive, Columbia, MO 65201. E-mail: parkerjc@health.missouri.edu*

*Accepted for publication March 31, 2005.*

support of health status as the predictor variable, Smedstad, *et al*<sup>6</sup> reported no differences in psychological distress in persons with RA compared to control subjects after controlling for pain, disability, and fatigue, indicating that these health status variables were associated with depressive symptoms. Iosifescu, *et al*<sup>2</sup> have provided a review of the literature supporting health status (medical illness) as a predictor of depression.

However, one of the most impressive studies on the relationship between depression and health status was conducted by Hayes, *et al*<sup>7</sup>; this study was notable for the sample size ( $n = 856$ ), the longitudinal structure of the data (3 timepoints over 4 years), and the cross-lagged methodology. Hayes, *et al* replicated a frequent finding in the literature that there exists a moderate cross-sectional correlation between physical health and mental health; they also found marked stability effects over time for self-reports of both physical health and mental health (without intervention). Lastly, analyses by Hayes, *et al* suggested that the evidence for a causal effect was stronger in the direction from physical health to mental health (e.g., depression), rather than vice versa<sup>7</sup>.

Iosifescu, *et al*<sup>2</sup> concluded, based on their literature review, that antidepressant intervention for major depressive disorder for persons with chronic medical illness was less effective than for persons without chronic medical illness. Ciechanowski, *et al*<sup>8</sup> suggested that the relationship between medical illness and depression may be mediated by variables such as self-care, nutrition, and/or adherence to treatment. Another potential mediator of the relationship between health status and depressive symptoms pertains to cognitive processes such as coping or self-efficacy (i.e., the degree to which a person believes that he or she can cope with a stressor). In the arthritis literature, the work of Lorig and colleagues<sup>9</sup> and Parker and colleagues<sup>10,11</sup>, among others, suggested that cognitive coping variables are related to both health status and depressive symptoms. Therefore, a “cognitive coping” variable is a logical candidate for exploration as a potential mediator of the relationship between health status and depression.

Based on the available literature, the hypotheses for the current study were as follows: (1) that health status variables would predict symptoms of depression in a sample of RA patients; (2) that the predictive relationship would be mediated by “cognitive coping” variables; and (3) that the mediated effect of the cognitive coping variables would remain constant over time. If cognitive coping variables are found to mediate the relationship between health status and depression, they become important targets for clinical intervention in their own right.

## MATERIALS AND METHODS

**Subjects.** This project consisted of secondary analyses of data obtained from a previous study of persons with RA<sup>12</sup>. Subjects were 54 persons (39 women, 15 men) with a diagnosis of classic or definite RA. The mean age of the sample was 54.6 years (SD 11.4); the mean education level was 12.6

years (SD 2.3). The median annual income was between \$15,000 and \$19,999. The functional class of patients was as follows: 4% ( $n = 2$ ) Class I; 48% ( $n = 26$ ) class II; 48% ( $n = 26$ ) class III. Subjects were recruited from a midwestern Department of Veterans Affairs (VA) hospital ( $n = 8$ , 15%), a university medical center ( $n = 24$ , 44%), and a private rheumatology practice ( $n = 22$ , 41%). The diagnosis of RA was made by collaborating rheumatologists using the 1987 diagnostic criteria of the American College of Rheumatology<sup>13</sup>. Subjects also met the diagnostic criteria for major depression (MD) as diagnosed by a collaborating psychiatrist using the Structured Clinical Interview for DSM-III-R (SCID)<sup>14</sup>. Subjects were evaluated for MD after reporting depressive symptoms during a telephone screening for depression

## Measures

**Center for Epidemiologic Studies-Depression Scale (CES-D).** The CES-D<sup>15</sup> is a 20 item self-report measure that assesses depressive symptoms. Scores on each item range from 0 to 3, higher scores indicating more frequent experience of depression. The CES-D has demonstrated sound reliability and validity<sup>16</sup>, including studies that have assessed its psychometric properties on persons with RA<sup>17,18</sup>.

**Hamilton Rating Scale for Depression (HAM-D).** The HAM-D<sup>19,20</sup> is a 17 item, interview based inventory that yields a measure of depression severity; higher scores indicate greater depression severity. Studies indicate that the HAM-D is a reliable and valid measure of depression<sup>21-23</sup>.

**Pain Control and Rational Thinking (PCRT).** PCRT is a factor score from the Coping Strategies Questionnaire (CSQ)<sup>24,25</sup>, which is a 50 item measure designed to assess coping strategies and perceptions of coping effectiveness. PCRT assesses the degree to which an individual believes that he or she can manage pain; scores can range from 0 to 18, higher scores indicating a greater belief in one’s ability to manage pain. Studies indicate that PCRT correlates with measures of both health status and depression in persons with RA<sup>10,25,26</sup> and therefore was selected as a good representation of the “cognitive coping” construct under investigation.

**Arthritis Impact Measurement Scale-2 (AIMS-2).** The AIMS-2<sup>27</sup> is a 78 item questionnaire designed to measure health status for persons with arthritis. Five factor scores have been identified, but only the physical, symptom, and social scores were relevant to the research questions. The AIMS-2 has been shown to have adequate reliability and validity<sup>27</sup>.

**Procedures.** Complete information on subject selection can be found in Parker, *et al*<sup>12</sup>, but the key aspects will be summarized. Subjects were recruited from a university hospital, a VA hospital, and a private rheumatology clinic; they were screened for eligibility by administering the CES-D. Those subjects who scored  $\geq 11$  on the CES-D were then assessed for MD via the SCID, and subjects who met criteria for MD were invited to participate in the depression management study. A total of 638 persons with RA were screened; 254 persons with RA were invited to participate in the evaluation for MD; 84 consented to the diagnostic interview, and 54 subjects who met criteria for MD were enrolled in the study. Subjects were then randomly assigned to one of 3 treatment groups: (1) medication plus cognitive-behavioral intervention, (2) medication plus attention control, or (3) medication only. All subjects were prescribed the antidepressant medication (sertraline), although 3 subjects were subsequently prescribed nortriptyline (consistent with practice guidelines) after it was determined that sertraline did not induce a clinical response. Data were collected at baseline, postintervention, 6 month followup, and 15 month followup. There were dropouts during the course of the study; 41 of the original 54 subjects remained at the 15 month followup. Results from the study by Parker, *et al*<sup>12</sup> indicated no significant differences between groups at any time period for the depression, cognitive coping variables, or health status measures, but subjects in all 3 groups experienced a significant decrease in depressive symptomatology over time. Thus, for these analyses, all 3 groups were combined. The Parker *et al*<sup>12</sup> data (across time) for the variables under study in these analyses (i.e., depression, cognitive coping, health status) are shown in Table 1.

Table 1. Means (SD) for study variables at each measurement interval.

Measure	Baseline	Postintervention	6 month Followup	15 Month Followup
CES-D	28.9 (9.9)	14.9 (8.8)	15.8 (9.7)	11.9 (7.7)
HAM-D	42.0 (10.0)	15.9 (11.0)	13.8 (11.0)	10.3 (8.3)
PCRT	9.5 (2.6)	11.5 (2.2)	11.3 (2.4)	11.3 (2.4)
AIMS-2 Physical	3.0 (1.2)	2.7 (1.3)	2.5 (1.5)	2.6 (1.5)
AIMS-2 Symptom*	6.5 (1.8)	5.6 (2.1)	5.6 (2.1)	5.4 (2.1)
AIMS-2 Social	5.3 (1.6)	4.6 (1.8)	4.4 (2.1)	4.2 (1.7)

CES-D: Center for Epidemiologic Studies — Depression Scale, HAM-D: Hamilton Rating Scale for Depression, PCRT: Pain Control and Rational Thinking (factor score from Coping Strategies Questionnaire), AIMS-2: Arthritis Impact Measurement Scales-2. \*AIMS-2 Symptom score is a measure of pain.

To demonstrate a mediator effect, several conditions must be met<sup>28</sup>. The independent variable must be correlated with the mediator variable; the mediator variable must be correlated with the dependent variable, and when the mediator is added to the model, the path from the independent variable to the mediator to the dependent variable must be significant. To test for mediation, a 3 step regression process can be used as follows: (1) regressing the dependent variable on the independent variable, (2) regressing the mediator variable on the independent variable, and (3) regressing the dependent variable on both the independent variable and the mediator variable.

For the purposes of this study, individual scales were aggregated to form 2 composite variables: (1) a variable labeled “health status” (AIMS-2 physical, symptom, and social scores); and (2) a variable labeled “depression” (CES-D and HAM-D); the composite scores were derived by simple summation of the standardized scores for the measures being combined. PCRT was selected as the “cognitive coping” variable on the basis of previous analyses<sup>10</sup>. Figure 1 shows the hypothesized relationship among the variables, with the independent variable being health status, the mediator variable being PCRT, and the dependent variable being depression. This model was tested at baseline and sequentially at all followup periods. The bivariate relationship among all observed variables was established at baseline via inspection of correlation coefficients. In effect, this procedure established the first 2 conditions necessary for a mediator relationship to exist. Although the baseline correlations were known when establishing the model, the correlations among the variables at the later time periods were not known. The specific research questions were *a priori*; no adjustments to the alpha level of 0.05 were required.

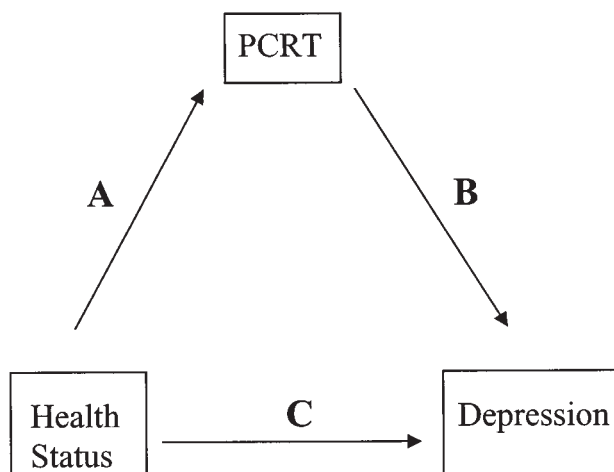


Figure 1. Hypothesized mediator model.

## RESULTS

**Relationship between health status (independent variable) and depression (dependent variable).** The first step in the test of a mediator relationship is to establish that a significant association exists between the independent and dependent variables, the health status and depressive symptoms, respectively (path C in Figure 1). Similar to previous studies involving persons with RA, results suggested that health status and depressive symptoms were related. The coefficient for the regression of depression on health status was 0.55 ( $p = 0.0008$ ) at baseline, 0.61 ( $p = 0.0001$ ) at postintervention, 0.85 ( $p = 0.0001$ ) at 6 month followup, and 0.52 ( $p = 0.0001$ ) at the 15 month followup. These results provide evidence of a direct effect between health status and depression at all 4 time periods. Thus, the relationship between the 2 variables remained consistent, even as the level of depression decreased (Table 2).

**Relationship between health status (independent variable) and PCRT (mediator variable).** The second step in the test of a mediator relationship is to establish that a significant association exists between the independent variable and the mediator variable, in this study, health status and PCRT, respectively (path A in Figure 1). Results indicated that health status and PCRT were correlated. The coefficient for the regression of PCRT on health status was  $-0.78$  ( $p = 0.0001$ ) at baseline,  $-0.44$  ( $p = 0.006$ ) at postintervention,  $-0.73$  ( $p = 0.0001$ ) at 6 month followup, and  $-0.59$  ( $p = 0.0001$ ) at 15 month followup. These results provide evidence that a significant relationship existed between health status and PCRT at all 4 time periods (Table 2).

**Mediator relationship of the full model.** The third step in the test of a mediator relationship is to establish that the path from the independent variable to the mediator to the dependent variable is significant. Thus, for this study, the path from health status to PCRT to depression must be significant (paths A and B combined in Figure 1). Thus, the applicable test statistic consisted of the product of 2 regression coefficients divided by their standard error, which has an approximate standardized, normal distribution ( $z$ ). Results indicated that the coefficient for this path was not significant at baseline ( $z = 1.50$ ,  $p = 0.13$ ) or at 15 month

Table 2. Path coefficients (SD) for the relationship between health status and depression across 4 time intervals.

Time	Health-Depression Direct Effect*	Health-PCRT Direct Effect**	Health-PCRT-Depression Mediator Effect***
Baseline	0.55 <sup>†</sup> (0.15)	-0.78 <sup>†</sup> (0.16)	0.16 (0.11)
Posttest	0.61 <sup>†</sup> (0.14)	-0.44 <sup>†</sup> (0.15)	0.18 <sup>††</sup> (0.08)
6 month	0.85 <sup>†</sup> (0.16)	-0.73 <sup>†</sup> (0.15)	0.26 <sup>††</sup> (0.13)
15 month	0.52 <sup>†</sup> (0.11)	-0.59 <sup>†</sup> (0.14)	0.06 (0.07)

\* Coefficient for regression of depression on health status. \*\* Coefficient for regression of PCRT on health status. \*\*\* Product of coefficient for regression of PCRT on health status and coefficient for regression of depression on PCRT and health status. <sup>†</sup> p < 0.01. <sup>††</sup> p < 0.05. PCRT: Pain Control and Rational Thinking (factor score from Coping Strategies Questionnaire).

followup ( $z = 0.87$ ,  $p = 0.38$ ), but was significant at post-treatment ( $z = 2.13$ ,  $p = 0.03$ ) and at 6 month followup ( $z = 2.01$ ,  $p = 0.04$ ). These results indicate that the mediator effect of PCRT was not present at baseline (highest depression score) or at 15 month followup (lowest depression score). However, a mediator effect was found at postintervention and at 6 month followup (moderate depression scores; Table 2).

To examine the possibility that the sample size might have been insufficient at baseline and at 15 months, *post hoc* power analyses were conducted. Given the observed, mediated path coefficient at baseline, 96 subjects would have been required to obtain statistical significance. Given the observed, mediated path coefficient at 15 month followup, 222 subjects would have been required to obtain statistical significance. Therefore, given the large sample sizes that would have been required to obtain statistical significance at baseline and at 15 months, the inference that the mediator variable had no meaningful effect at these 2 intervals appears to be warranted.

## DISCUSSION

The main purpose of our study was to determine if a variable that assessed cognitive coping strategy (i.e., PCRT) mediated the relationship between health status and depression over the course of a pharmacological intervention. Results indicated a direct relationship between health status and depression over the entire course of the intervention, but PCRT mediated this relationship only at postintervention and 6 month followup. PCRT did not mediate the relationship at baseline (preintervention) or at 15 month followup. Interestingly, the mediation effect was found at postintervention and 6 month followup despite the fact that there is inherent overlap, to some extent, between the symptoms of depression and the symptoms of RA (e.g., loss of energy).

A possible explanation for the inconsistent mediator relationship over the course of the intervention involves the clinical course of depression. The results suggest that cognitive coping strategies (e.g., PCRT) are more relevant for persons with RA who are experiencing moderate depressive symptoms. Specifically, at baseline, the mean CES-D score for the sample was 28.9, which is well above the common-

ly used cutoff score of 16<sup>15,29,30</sup>. Cognitive coping strategies may be largely ineffective for persons experiencing such severe symptoms of depression, which may explain why PCRT did not mediate a health status-depression relationship at baseline. At the 15 month followup, the mean CES-D score was only 11.9, which is roughly 4 points lower than the usual CES-D cutoff for depression. When persons are experiencing such low levels of depressive symptoms, their coping strategies may similarly be irrelevant from a mediation standpoint. In contrast, at postintervention and 6 month followup, the mean CES-D scores were 14.9 and 15.8, respectively. These scores are very close to the suggested cutoff for the CES-D, and are indicative of more moderate levels of depressive symptoms. Thus, cognitive coping strategies may be most relevant for individuals who are experiencing neither severe nor low levels of depression, but somewhere in between. These results suggest that psychological interventions (e.g., cognitive-behavioral intervention, coping skills training) may be most helpful to persons with RA who are experiencing moderate levels of depressive symptomatology. For example, in the case of persons with RA and comorbid major depression, cognitive-behavioral interventions may be best timed after antidepressive medication has reduced depressive symptoms to moderate levels.

Another interesting observation in the data (Table 1) is that the antidepressive intervention exerted a powerful effect on depressive symptoms as measured by both the CES-D and the HAM-D. At postintervention, 6 month followup, and 15 month followup, notable decreases in depressive scores were observed; these data are discussed in detail in Parker, *et al*<sup>12</sup>. Yet, the impact of the antidepressive intervention on the cognitive coping (PCRT) variable was quite modest in quantitative terms. Similarly, the antidepressive intervention yielded only a modest improvement in health status as measured by the AIMS-2 scores. Clearly, the lack of a mediation effect for PCRT in the models at baseline (high depression) and at 15 month followup (low depression) appears to be due to the variation in depression scores that occurred over the course of the measurement intervals, rather than to substantive quantitative changes on the other variables in the model.

Numerous potential pitfalls exist in the development of models of directional relationships. In our study, an argument was made on the basis of the empirical literature for a directional influence of health status on depression, but the converse relationship also could be postulated. In our opinion, the arguments for the proposed model are compelling, but there is recognition that goodness-of-fit statistics also could be generated for alternative conceptual models. Hence, the value of these particular analyses must be considered in light of the broader literature on the relationship between health status and depression.

The sample was collected from a single region of the US, which raises the issue of generalizability of results. In addition, all subjects met the diagnostic criteria for major depression; thus, the potential mediator properties of PCRT in a non-MD sample of persons with RA are not known. Finally, one recognized aspect of path modeling is that there are inherent limitations in the number of variables that can be examined simultaneously; the limitations in the number of variables derive from sample size considerations and the ability to directly test the research questions under investigation. In this study, the relatively small sample size was a limiting factor, which argues for cautious interpretation. Inevitably, the totality of variables that might potentially affect a given dependent measure cannot be included. Followup studies that build upon an exploratory path model are generally needed.

Our data suggest that cognitive coping strategies mediate the relationship between health status and depression, but only at moderate levels of depressive symptomatology. If these findings are confirmed in other studies, there would be support for focusing cognitive-behavioral interventions on persons with RA who present with moderate levels of depression, and possibly for timing cognitive-behavioral interventions sequentially after antidepressive medications have reduced the more severe depressive symptoms.

## REFERENCES

1. Depression Guideline Panel. Depression in primary care: volume 1. Detection and diagnosis. Clinical practice guideline, Number 5. Rockville, MD: US Department of Health and Human Services, Public Health Service; 1993:73.
2. Iosifescu DV, Bankier B, Fava M. Impact of medical comorbid disease on antidepressant treatment of major depressive disorder. *Curr Psych Rep* 2004;6:193-201.
3. Joreskog KG, Sörbom D. LISREL 7: A guide to the program and applications. 2nd ed. Chicago: SPSS Inc.; 1988.
4. Anderson KO, Keefe FJ, Bradley LA, et al. Prediction of pain behavior and functional status of rheumatoid arthritis patients using medical status and psychological variables. *Pain* 1988;33:25-32.
5. Beckham JC, D'Amico CJ, Rice JR, Jordan JS, Divine GW, Brook WB. Depression and level of functioning in patients with rheumatoid arthritis. *Can J Psychiatry* 1992;37:539-43.
6. Smedstad LM, Moum T, Vaglum P, Kvien TK. The impact of early rheumatoid arthritis on psychological distress. *Scand J Rheumatol* 1996;25:377-82.
7. Hays RD, Marshall GN, Wang EYI, Sherbourne CD. Four-year cross-lagged associations between physical and mental health in the medical outcomes study. *J Consult Clin Psychol* 1994;62:441-9.
8. Ciechanowski PS, Katon WJ, Russo JE. Depression and diabetes: Impact of depressive symptoms on adherence, function, and costs. *Arch Intern Med* 2000;160:3278-85.
9. O'Leary A, Shoor S, Lorig K, Holman HR. A cognitive-behavioral treatment for rheumatoid arthritis. *Health Psychol* 1988;7:527-44.
10. Parker JC, Smarr KL, Buescher KL, et al. Pain control and rational thinking: Implications for rheumatoid arthritis. *Arthritis Rheum* 1989;32:984-90.
11. Smarr K, Parker J, Wright G, et al. The importance of enhancing self-efficacy in rheumatoid arthritis. *Arthritis Care Res* 1997;10:18-26.
12. Parker JC, Smarr KL, Slaughter JR, et al. Management of depression in rheumatoid arthritis: A combined pharmacologic and cognitive-behavioral approach. *Arthritis Care Res* 2003;49:766-77.
13. Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988;31:315-24.
14. Spitzer RL, Williams JBW, Gibbon M, First MB. User's guide for the structured clinical interview for DSM-III-R: SCID. Washington, DC: American Psychiatric Press; 1990.
15. Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. *Appl Psychol Meas* 1977;1:385-401.
16. Orme JG, Reis J, Herz EJ. Factorial and discriminant validity of the Center for Epidemiological Studies Depression (CES-D) Scale. *J Clin Psychol* 1986;42:28-33.
17. Rhee SH, Petroski GF, Parker JC, et al. A confirmatory factor analysis of the Center for Epidemiologic Studies Depression Scale in rheumatoid arthritis patients: Additional evidence for a four-factor model. *Arthritis Care Res* 1999;12:392-400.
18. Sheehan TJ, Fifield J, Reisine S, Tennen H. The measurement structure of the Center for Epidemiologic Studies Depression scale. *J Pers Assess* 1995;64:507-21.
19. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry* 1960;23:56-62.
20. Hamilton M. Development of a rating scale for primary depressive illness. *Br J Soc Clin Psychol* 1967;6:278-96.
21. Bech P, Gram LF, Dein E, Jacobsen O, Vitger J, Bolwig TG. Quantitative rating of depressive states: Correlation between clinical assessment, Beck's Self-Rating Scale and Hamilton's Objective Rating Scale. *Acta Psychiatr Scand* 1975;51:161-70.
22. Hedlund JL, Vieweg BW. The Hamilton Rating Scale for Depression: A comprehensive review. *J Operational Psychiatry* 1979;10:149-65.
23. Williams JG, Barlow DH, Agras WS. Behavioral measurement of severe depression. *Arch Gen Psychiatry* 1972;27:330-3.
24. Rosenstiel AK, Keefe FJ. The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. *Pain* 1983;17:33-44.
25. Keefe FJ, Caldwell DS, Queen KT, et al. Pain coping strategies in osteoarthritis patients. *J Consult Clin Psychol* 1987;55:208-12.
26. Parker JC, McRae C, Smarr KL, et al. Coping strategies in rheumatoid arthritis. *J Rheumatol* 1988;15:1376-83.
27. Meenan RF, Mason JH. AIMS2 users guide. Boston: Boston University Arthritis Center; 1990.
28. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* 1986;51:1173-82.
29. Coulehan JL, Schulberg HC, Block MR. The efficiency of depression questionnaires for case finding in primary medical care. *J Gen Intern Med* 1989;4:541-7.
30. Schulberg HC, Saul M, McClelland M, Ganguli M, Christy W, Frank R. Assessing depression in primary medical and psychiatric practices. *Arch Gen Psychiatry* 1985;42:1164-70.