Stress Management in Rheumatoid Arthritis: What Is the Underlying Mechanism?

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Objective. *To test whether change in cognitive–behavioral variables (such as self-efficacy, coping strategies, and helplessness) is a mediator in the relation between cognitive behavior therapy and reduced pain and depression in persons with rheumatoid arthritis (RA).*

Methods. *A sample of patients with RA who completed a stress management training program (n = 47) was compared to a standard care control group (n = 45). A path analysis testing a model including direct effects of comprehensive stress management training on pain and depression and indirect effects via change in cognitive–behavioral variables was conducted.*

Results. *The path coefficients for the indirect effects of stress management training on pain and depression via change in cognitive–behavioral variables were statistically significant, whereas the path coefficients for the direct effects were found not to be statistically significant.*

Conclusion. *Decreases in pain and depression following stress management training are due to beneficial changes in the arenas of self-efficacy (the belief that one can perform a specific behavior or task in the future), coping strategies (an individual's confidence in his or her ability to manage pain), and helplessness (perceptions of control regarding arthritis). There is little evidence of additional direct effects of stress management training on pain and depression.*

INTRODUCTION

The risk for being diagnosed with major depressive disorder at any one point is 5% to 9% for women and 2% to 3% for men (1). In contrast, the prevalence of depression in persons with rheumatoid arthritis (RA) is approximately 20% (2). Depression is likely to have significant implications for the health status of persons with RA, given that the coexistence of depressive symptoms and a medical condition is associated with severe functional declines (3). For example, Katz and Yelin (4) found that, in persons with RA, depression is associated with clinical characteristics (e.g., greater number of painful joints), reduced functioning (e.g., greater number of days in bed), and a greater number of RA-related physician visits and hospitalizations.

The need for pain relief is also a chief concern for persons with rheumatoid arthritis. For example, McKenna and Wright (5) found that more than half of RA patients in their sample (n = 250) ranked pain as the most important symptom to be treated, and

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pain has been related to subsequent disability in persons with chronic rheumatic diseases (6). The relationship between pain and disability also has been supported by results of a laboratory experiment by Dardick, Basbaum, and Levine (7), who compared the morbidity (weight loss and decrease in activity) of arthritic rats whose pain receptors were eliminated with that of arthritic rats whose pain receptors remained intact. Although elimination of pain did not affect disease activity, it did have a positive effect on weight and activity, supporting the hypothesis that reducing pain may have a significant effect on the health status of persons with RA.

The fact that pain and depression are significant problems for persons with RA and that the prevalence of depression is higher in RA samples when compared with the general population is easily understandable given the potentially disabling nature of RA. There is convincing evidence, however, that disease activity or severity is not the main predictor of either the level of pain or of depression experienced by persons with RA. Parker and colleagues (8) found no significant relationships between pain and such medical variables as erythrocyte sedimentation rate, grip strength, and joint involvement. Hagglund and colleagues (9) also found no significant relationships between pain and measures of disease activity and severity (erythrocyte sedimentation rate, joint swelling, radiographic ratings). Similar results have been found for depression in persons with RA, with several researchers finding a lack of significant association between depression and disease activity, severity, or duration (e.g., 10–14). Pain and depression seem more related to psychological variables such as social stress and lack of social support (12), daily stress (8), and attitude toward illness (13). Several studies also found significant associations between pain/depression in RA and psychological variables addressed during cognitive behavior therapy (referred to as cognitive–behavioral variables in the present study), such as cognitive distortions (15), helplessness (9,16), catastrophizing (17), and coping strategies (9,18).

Given that pain and depression in persons with RA are significantly associated with general functioning and health status, alleviation of pain and depression could result in significant improvements in the lives of persons with RA. Studies indicating a lack of relationship between disease activity and pain/depression (e.g., 9,13) suggest that reduction in disease activity may not necessarily lead to the alleviation of pain and depression in persons with RA. On the other hand, cognitive behavior therapy is a good candidate for the treatment of pain and depression in persons with RA, given findings of significant associations between cognitive–behavioral variables and pain/depression in RA. Several studies examining the efficacy of cognitive behavior therapy in reducing depression or pain in RA have found positive results (e.g., 19–21).

McCracken (22) noted the paucity of empirical evidence regarding possible variables that mediate treatment effects of cognitive behavior therapy and suggested that increasing knowledge regarding mediating variables can clarify how psychological interventions effect change in pain and depression in RA. Increasing knowledge regarding mediating variables also has potential clinical value. That is, treatments can be refined to effect more change in the mediating variables known to have the most effects on pain and depression. An obvious candidate for such a mediating variable in cognitive behavior therapy is change in cognitive–behavioral variables (e.g., cognitive distortions, helplessness, coping strategies, self-efficacy). In their study examining the efficacy of cognitive behavior therapy in persons with RA, O'Leary, Shoor, Lorig, and Holman (23) found a positive correlation between the degree of self-efficacy enhancement and the magnitude of improvement on outcomes measures.

Parker and colleagues (21) examined the effects of cognitive behavior therapy (stress management training) on the clinical outcomes in persons with RA. They demonstrated that the stress management group had significant improvements on measures of pain and health status compared with a control group receiving patient education and a control group receiving only standard rheumatologic care. In contrast, there were no group differences in disease activity measured by joint counts. The possible reasons for the success of cognitive behavior therapy were not examined in this study.

Smarr and colleagues (24) re-analyzed the data from the Parker study in order to examine the effect of change in self-efficacy following cognitive behavior therapy. They found that change in self-efficacy was significantly related to changes in selected measures of depression, pain, health status, and disease activity. Smarr and colleagues cautioned that their results do not establish that change in self-efficacy is the underlying mechanism responsible for the positive changes following cognitive behavior therapy, however.

In the present study, the treatment efficacy of cognitive behavior therapy on pain and depression in persons with RA was examined with the goal of providing clearer answers regarding the way in which cognitive behavior therapy reduces pain and

depression in persons with RA. Specifically, the goal of the present study was to establish whether change in cognitive–behavioral variables (self-efficacy—the belief that one can perform a specific behavior or task in the future; coping strategies—an individual's confidence in his or her ability to manage pain; helplessness—perceptions of control regarding arthritis) is the underlying mechanism responsible for the positive results of cognitive behavior therapy. New analyses were performed on Parker and colleagues' (21) stress management study, testing a path analysis including both direct treatment effects on pain and depression and indirect treatment effects mediated by changes in cognitive–behavioral variables. The following alternative hypotheses were tested: (a) cognitive behavior therapy has direct effects on pain and depression but no statistically significant indirect effects mediated by changes in cognitive–behavioral variables (self-efficacy, coping strategies, and helplessness); (b) cognitive behavior therapy has indirect effects on pain and depression mediated by change in cognitive–behavioral variables but no statistically significant direct treatment effects; and (c) cognitive behavior therapy has both direct effects on pain and depression and indirect effects on pain and depression mediated by change in cognitive–behavioral variables.

If hypothesis (a) is supported, then change in cognitive–behavioral variables is not the underlying mechanism in the positive effects of cognitive behavior therapy and there are other unidentified underlying mechanisms (i.e., we do not know why cognitive behavior therapy reduces pain and depression in persons with RA). If hypothesis (b) is supported, then the decrease in pain and depression following cognitive behavior therapy is due to the positive changes in the self-efficacy, coping strategies, and helplessness of persons with RA. If hypothesis (c) is supported, both cognitive–behavioral variables and other unidentified variables are responsible for the positive effects of cognitive behavior therapy.

PATIENTS AND METHODS

Participants. One hundred forty-one persons with RA (diagnosed by a rheumatologist using the 1987 diagnostic criteria of the American College of Rheumatology [25]) participated in a study examining the efficacy of stress management training conducted by Parker and colleagues (21). The median age of the participants was 60, and the median years of education was 12. The median annual income was $15,000–$20,000. The participants were predominantly from the middle socioeconomic category. Sixty-three percent of the participants were unemployed, disabled, or retired, and 79 percent of the participants were married. According to the Steinbrocker criteria (26), 21 percent of the participants were functional class I, 69 percent of the participants were functional class II, and 10 percent of the participants were functional class III. Participants who were functional class IV were excluded because of the demands of the study.

Participants were randomly assigned to the stress management group, the attention control group, or the standard care control group, but there were equal proportions of participants in each functional class, clinic site, and degree of life stress in each group. Participants in the stress management group (n = 47) received stress management training in addition to ongoing rheumatologic care. They completed an outpatient program involving 10 weekly 2-hour outpatient visits. During this program, the participants learned cognitive behavior strategies including relaxation, stress management, coping strategies, problem solving, pain management, and improvement of interpersonal relationships and social support networks. A 15-month maintenance program (individual visits once every 3 months) followed the 10-week stress management program. Participants in the attention control group (n = 49) participated in a patient education program in addition to ongoing rheumatologic care. The same 3 counselors who administered the stress management training also administered the patient education program. The number of sessions and followup visits were identical in the stress management program and the patient education program. Participants in the standard care control group (n = 45) only received ongoing rheumatologic care.

In the present study, the direct effect of stress management on pain and depression and the indirect effect of stress management on pain and depression via change in cognitive–behavioral variables were assessed by comparing the 47 participants in the stress management group and the 45 participants in the standard care control group. Parker and colleagues (21) established that there were no significant differences between the standard care control group and the attention control group. Also, the focus of the present study was the identification of a specific mechanism that makes stress management training effective in reducing pain and depression. Therefore, the analyses in the present study were limited to a comparison between the stress management group and the standard care control group. The study was conducted in accordance with the Insti-

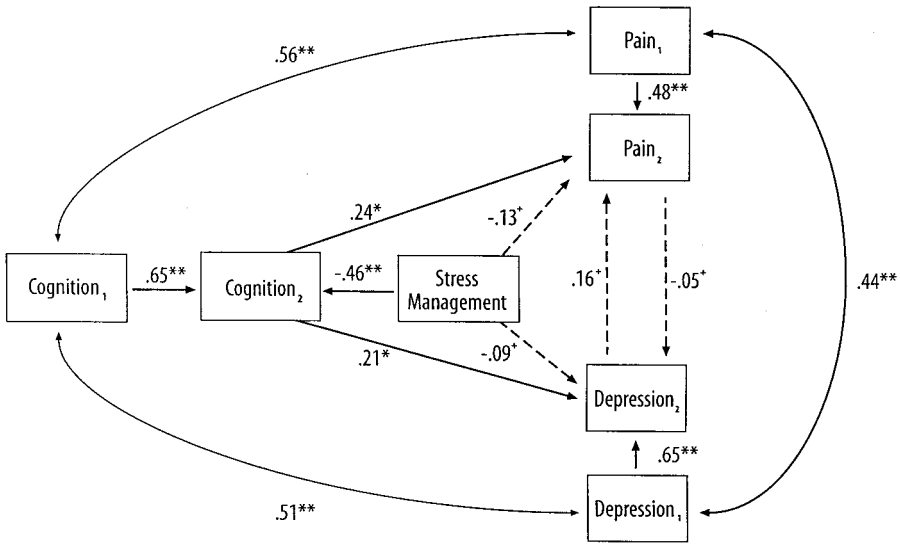

Figure 1. Path diagram with standardized path coefficients for the full model including both direct and indirect effects of stress management training on pain and depression. Cognition_1 = pre-intervention cognitive–behavioral variables; Cognition_2 = post-intervention cognitive–behavioral variables; Depression_1 = pre-intervention depression; Depression_2 = post-intervention depression; Pain_1 = pre-intervention pain; Pain_2 = post-intervention pain; + $P \geq 0.10$; *$P < 0.05$; **$P < 0.001$.

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Measures. *Depression.* Three valid and reliable measures were used to assess depression. The Center for Epidemiologic Studies–Depression (CES-D) Scale (27) is a 20-item scale designed to assess depressive symptoms in the general population. The Symptom Checklist-90–Revised (SCL-90-R) (28) is an instrument usually used to assess overall psychological functioning, and the Arthritis Impact Measurement Scales (AIMS) (29,30) are designed to assess the health status of persons with RA. The depression subscales from the SCL-90-R and the AIMS were used.

Pain. Four valid and reliable measures were used to assess pain. The visual analog scale for pain (VAS) (31,32) asks the participant to indicate a point on a 10-cm line (with anchors "no pain" at one end and "pain as bad as it can be" at the other end) that represents his or her pain level during the past week. The number of words chosen (NWC) and present pain intensity (PPI) are 2 subscales of the McGill Pain Questionnaire (MPQ) (33,34), a measure of self-reported pain. The AIMS (29,30) is a questionnaire designed to assess the health status of persons with RA, and the pain subscale from this measure was used.

Cognitive–behavioral variables. Three valid and reliable measures were used to assess cognitive–behavioral variables. The Arthritis Helplessness Index (AHI) (35) measures perceptions of control regarding arthritis. The Arthritis Self-Efficacy Scale (ASES) (36) measures the perception of ability to manage outcomes and problems associated with arthritis. Pain Control and Rational Thinking (PCRT) (37) is a factor score from the Coping Strategies Questionnaire (CSQ), which measures confidence in the ability to manage pain.

Analyses. Given the modest sample size, composite variables were created for depression, pain, and cognitive–behavioral measures. Exploratory factor analyses were conducted to evaluate the appropriateness of the composites. After the composite variables were created, multiple regression analyses were conducted in order to establish that stress management training resulted in significant improvements in both depression and pain. Next, structural equation methods were used to test the model shown in Figure 1. This model examines both the direct effects of stress management training on pain and depression at Time 2 as well as the indirect effects via the cognitive–behavioral variables. The model also includes reciprocal effects between pain and depression at Time 2. The effects of stress manage-

ment training on pain, depression, and cognitive–behavioral outcomes at Time 2 are adjusted for their initial (pre-intervention) values. After fitting the initial model shown in Figure 1, we retested the model after removing the statistically nonsignificant paths.

In model fitting, the adequacy of the model is indicated by the lack of discrepancy between the covariance matrix predicted by the model and the observed covariance matrix. This discrepancy is evaluated formally through the use of the chi-square goodness-of-fit test (χ^2) and descriptively through the use of one or more goodness-of-fit indices. With multivariate normal data and a large sample size, a statistically nonsignificant χ^2 (typically, $P > 0.05$) is indicative of a good fitting model. Violations of either of these assumptions tend to undermine the validity of the χ^2 test. Unfortunately, there is no general method for quantifying the effects of departures from normality or for determining at what point the sample size is large enough to justify asymptotic theory. Thus, formal tests of model fit are supplemented by the examination of descriptive measures of fit. Two such indices in popular use are the goodness-of-fit index (GFI) and the root mean square error of approximation (RMSEA). The GFI is a measure of absolute fit that represents the degree to which the fitted model reproduces the variances and covariances in the observed data. GFI is similar in spirit to the R^2 from multiple linear regression, and like R^2 it has a theoretical range of 0 to 1 with larger values indicating better fit. The RMSEA index includes a penalty for model complexity and thus can be interpreted as "a measure of discrepancy per degree of freedom" (38). Browne and Cudeck (38) suggest that RMSEA values of 0.05 or less are indicative of a good fitting model and that RMSEA values of 0.10 or greater signal a serious lack of fit.

In addition to an assessment of overall model fit, we are interested in making inferences about individual model parameters. As with the χ^2 test of fit, the standard errors of the path coefficients are strictly valid only in the case of multivariate normality and for large sample sizes. Given the modest sample size in the present study, bootstrap methods were used to reinforce the results produced by the normal theory methods.

Bootstrapping (39) is a simulation-based method for calculating standard errors that do not depend on specific distributional assumptions. The basic idea of the bootstrap, as it is applied to covariance structure models (40), is that the original random sample of observations is repeatedly sampled, with replacement, and the model is fit to each of the "bootstrap samples." Each of the bootstrap samples, also called replicates, is of the same size as the original sample. The empirical distribution of the bootstrap estimates is an approximation to the sampling distribution of the parameters estimates. The bootstrap distributions are then used to construct confidence intervals and to test hypotheses concerning the model parameters. The bootstrap replicates also can be used to construct a goodness-of-fit test (41).

As with all statistical methods, the bootstrap can break down when the sample size is too small. Because the bootstrap replicates are samples of size n drawn with replacement from the original sample of size n, the replicates will include some repeated observations. If the original sample size is too small, the bootstrap samples will underrepresent the true variability in the data. However, as noted by Chernick (42), the number of unique bootstrap samples grows very fast as the sample size increases. While not specifically discussing covariance models, Chernick (42) offers the "rule of thumb" that sample sizes of 50 or more are probably adequate for most purposes. In this study, the full and reduced models were each fit to 1,000 bootstrap samples. None of the samples were rejected due to insufficient variability. All model fitting was done via maximum likelihood estimation.

RESULTS

The CES-D total score, SCL-90-R depression, and AIMS depression loaded on 1 factor (depression) that explained 77% of the variance of the 3 measures, with factor loading ranging from 0.83 to 0.91. The PPI, VAS, NWC, and AIMS pain loaded on 1 factor (pain) that explained 66% of the variance of the 4 measures, with factor loadings ranging from 0.77 to 0.85. The ASES, PCRT, and AHI loaded on 1 factor (cognitive–behavioral variables) that explained 68% of the variance of the 3 measures, with factor loadings ranging from 0.82 to 0.83. An exploratory factor analysis of all of the measures resulted in 3 factors, with the CES-D total score, SCL-90-R depression, and AIMS depression loading on the depression factor (factor loadings ranging from 0.79 to 0.87), the PPI, VAS, NWC, and AIMS pain loading on the pain factor (factor loadings ranging from 0.56 to 0.94), and the ASES, PCRT, and AHI loading on the cognitive–behavioral variables factor (factor loadings ranging from 0.60 to 0.91). Given these results, the scores on the individual measures were standardized and then summed to create composite measures of depression, pain, and cognitive–behavioral variables. Time 1 means and standard deviations

were used for the standardization. Each composite variable had a unimodal and symmetric distribution.

The results of multiple regression analyses indicate that stress management training resulted in significant improvements in both depression and pain. Stress management training resulted in a significant decrease in pain at Time 2 controlling for pain at Time 1 ($\beta = -0.44$, $P = 0.002$). Stress management also resulted in a significant decrease in depression at Time 2 controlling for depression at Time 1 ($\beta = 0.26$, $P = 0.036$).

The initial model including both the direct and indirect effects (via changes in cognitive–behavioral variables) of the stress management training on pain and depression fit well, χ^2 (8) = 8.2, $P = 0.41$ (GFI = 0.97, RMSEA = 0.02). The path coefficients of this model are shown in Figure 1. In this model, the path coefficients for the indirect effects of the stress management training on pain and depression via cognitive–behavioral variables were significant. Stress management had a significant effect on cognitive–behavioral variables at Time 2, $P < 0.01$, and cognitive–behavioral variables at Time 2 had a significant effect on both pain at Time 2, $P = 0.02$, and depression at Time 2, $P = 0.05$. The path coefficients for the direct effect of the stress management training on pain at Time 2, $P = 0.12$, and the direct effect of the stress management training on depression at Time 2, $P = 0.27$, were not statistically significant. The reciprocal effects of pain on depression, $P = 0.68$, and depression on pain, $P = 0.16$, also were not statistically significant. The reduced model that eliminated the direct effects of the stress management training and the reciprocal paths between pain and depression fit the data nearly as well as the full model, χ^2 (12) = 13.4, $P = 0.28$ (GFI = 0.95, RMSEA = 0.05). The loss of fit from the full model was not statistically significant, χ^2 (4) = 6.14, $P = 0.19$. Significance levels for the Bollen-Stine goodness-of-fit statistic were 0.41 for the initial model and 0.28 for the reduced model. Bootstrapped significance levels for the individual path coefficients were uniformly consistent with the normal theory tests that are quoted here.

DISCUSSION

In the present study, the data from Parker and colleagues' (21) stress management training study were reexamined in an attempt to find the specific mechanism by which cognitive behavior therapy effects change in pain and depression in persons with RA. Three alternative hypotheses were tested: (a) cognitive behavior therapy has direct effects on pain and depression but no statistically significant indirect effects mediated by changes in cognitive–behavioral variables (self-efficacy, coping strategies, and helplessness); (b) cognitive behavior therapy has indirect effects on pain and depression mediated by change in cognitive–behavioral variables but no statistically significant direct treatment effects; and (c) cognitive behavior therapy has both direct effects on pain and depression and indirect effects on pain and depression mediated by change in cognitive–behavioral variables.

A model including both direct and indirect effects of the stress management training on pain and depression fit well, but the path coefficients indicating the direct effects were not statistically significant. The fit of the model dropping the direct effects was not significantly different from that of the original model. These results provide support for hypothesis (b) and evidence against hypotheses (a) and (c).

First, the results demonstrate the efficacy of cognitive behavior therapy in treating both pain and depression in persons with RA. Second, the results clarify how cognitive behavior therapy or stress management training effects change in pain and depression in RA. Stress management training helps persons with RA to improve coping strategies, lessen helplessness, and increase self-efficacy, and these positive changes lead to decreased pain and depression. Third, the results demonstrate that stress management training does not have an additional direct effect on pain and depression.

The results are consistent with those of O'Leary and colleagues (23) and Smarr and colleagues (24), who found that enhancements in self-efficacy were related to change in pain and depression measures. They also help explain why interventions without a focus on change in cognitive–behavioral variables (e.g., patient education in Parker and colleagues [43]; patient education group of Parker and colleagues [21]; structured group social support therapy in Bradley and colleagues [19]) are not as effective in decreasing pain or depression in persons with RA.

The methodologic limitations of the present study should be considered when interpreting the results. There is a possible limitation of the generalizability of the results, given that all of the participants came from a small Midwestern community, and a sizeable proportion of the participants were veterans (41%) or male (57%).

The first implication of the results is the need for health care professionals to become more aware of the benefits of psychological interventions in reducing pain and depression, which are significantly re-

lated to the health status of persons with RA. Second, cognitive behavior therapy for persons with RA should emphasize increased change in cognitive–behavioral variables (e.g., perceived coping effectiveness, decreased helplessness, and enhanced self-efficacy). For example, Parker and colleagues (21) did not find a statistically significant group difference between the stress management group, the attention control group, and the standard care control group in post-intervention depression. On the other hand, the present re-analyses of their data suggest that the participants in the stress management group experienced significantly more positive changes in cognitive–behavioral variables than those in the control group and that positive changes in cognitive–behavioral variables were significantly related to decrease in depression. Refining stress management training to maximize positive changes in cognitive–behavioral variables may increase its therapeutic efficacy on depression. Further research examining such refinements will contribute to both theoretical and practical knowledge regarding the treatment of pain and depression in persons with RA.

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