

Biopsychosocial functioning and pain self-efficacy in chronic low back pain patients

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Abstract—The aim of this study was to examine the relationship between biopsychosocial functioning and pain severity and to evaluate whether pain self-efficacy (PSE) mediates this relationship. This study used archival data from a multidisciplinary pain management program. Participants were 99 individuals (69% female) with chronic low back pain who completed measures of biological, psychological, and social functioning; pain severity; and PSE at admission. They ranged in age from 18 to 72 yr (mean = 42.6, standard deviation = 12.1). Structural equation modeling and bootstrapping techniques were used to test the significance of the mediated model. As we predicted, lower biological functioning (beta = -0.011 ; 95% confidence interval [CI] = -0.019 to -0.004 , $p = 0.002$) and social functioning (beta = -0.009 ; 95% CI = -0.016 to -0.003 , $p = 0.007$) were found to significantly predict higher pain severity, and lower social functioning was found to significantly predict lower PSE (beta = 0.196 ; 95% CI = -0.130 to 0.273 , $p = 0.002$). PSE did not mediate the relationship between biopsychosocial functioning and pain severity, and psychological functioning did not significantly predict pain severity or PSE. These findings suggest that social functioning is an important factor in predicting outcomes and has a number of treatment implications.

Key words: biopsychosocial, chronic low back pain, chronic pain, multidisciplinary rehabilitation, pain behaviors, pain self-efficacy, pain severity, physical/biological functioning, psychological functioning, social support.

INTRODUCTION

Chronic low back pain (CLBP) is a widespread and expensive problem. CLBP is the primary cause of disability and absenteeism in the workplace, and the cost of CLBP to society is staggering [1–2]. In order to further understand the experience of CLBP, it is important to consider the biopsychosocial model, which views physical illnesses as the result of the dynamic interaction among biological, psychological, and social factors [3]. Poor biopsychosocial functioning has been found to predict higher pain severity in CLBP patients [4]. Biological functioning refers to general physical functioning and the extent to which health limits physical activities. This model acknowledges the biological bases that underlie pain conditions, but also notes that psychosocial factors contribute to the experience and effect of pain [3].

Abbreviations: CLBP = chronic low back pain, MI = multiple imputation, PSE = pain self-efficacy, PSEQ = Pain Self-Efficacy Questionnaire, SD = standard deviation, SEM = structural equation modeling, SF-36 = Short Form-36 Health Status Questionnaire, SF-MPQ = Short-Form McGill Pain Questionnaire.

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Biopsychosocial conceptualizations of chronic pain have received increasing support in the broader pain literature [5–8]. However, little research involving CLBP patients includes an investigation of all three levels of biopsychosocial functioning. Specifically, studies on CLBP often investigate only two of the three levels (usually biological and psychological) of biopsychosocial functioning [9–16]. Though these studies have provided important building blocks for a biopsychosocial understanding of chronic pain, they are all incomplete. It is important to include all three areas of functioning in order to fully understand the heuristic model of CLBP and to best target interventions that address those areas.

Research has documented that biopsychosocial functioning and pain exist in a mutually reinforcing relationship [3,17], such that chronic pain predicts biopsychosocial functioning and that biopsychosocial functioning predicts chronic pain [3,18–19]. However, the influence of biopsychosocial functioning on pain severity is of particular interest in the present study because of the treatment implications of using biopsychosocial functioning as a point of intervention for chronic pain patients.

Pain self-efficacy (PSE) is a psychological variable that has been shown to be associated with all three aspects of biopsychosocial functioning [20]. PSE is a complex construct that consists of an individual's confidence in his or her ability to tolerate pain, cope with pain, and participate in daily activities despite pain [21–23]. It has been reported that individuals with higher PSE tend to report higher rates of biopsychosocial functioning and lower pain severity [21–23]. Self-efficacy beliefs are often used to predict pain tolerance and psychological aspects of the pain experience [2,9,16,24–27]. Specifically, those with higher self-efficacy rate pain stimuli as much less unpleasant than those with lower self-efficacy [26] and exhibit fewer pain behaviors [24–25]. In addition, researchers have indicated that increases in PSE for pain control may increase the usage of coping strategies, which results in reduced levels of disability [16]. Furthermore, lower levels of PSE are associated with lower levels of social support and higher levels of anxiety and depressive symptoms [20]. Research has also supported the positive influence of PSE on disease severity and physical functioning [28]. Overall, PSE positively affects biopsychosocial functioning in chronic pain patients [9,16,20]. However, the role of PSE within the relationship between biopsychosocial functioning and pain severity is unclear.

PSE also has important treatment implications. Specifically, chronic pain patients who have higher levels of self-efficacy are more motivated to adhere to treatment recommendations and engage in healthy behaviors because they believe they are more likely to succeed [3]. In addition, these individuals are less likely to give up when faced with pain. Thus, they are also less likely to succumb to activity avoidance, which leads to depression, loss of social reinforcers, and physical deconditioning [3]. Therefore, we suggest that PSE may be the mechanism influencing the relationship between biopsychosocial functioning and pain severity.

The present study has two primary hypotheses. First, we predicted that biological, psychological, and social functioning would separately predict pain severity such that lower biological, psychological, and social functioning would be associated with higher pain severity. Second, we predicted that PSE would mediate the relationship between biological, psychological, and social functioning and pain severity. We predicted biological, psychological, and social functioning would separately predict PSE, such that lower biological, psychological, and social functioning would be associated with lower PSE.

METHODS

Participants

We utilized archival data from a multidisciplinary treatment program for pain management at Unsted Park Rehabilitation Hospital in Surrey, England, to address these hypotheses. Individuals were eligible for participation if they (1) were 18 yr or older; (2) were able to read, write, and understand English; (3) had persistent low back pain for at least 6 mo; (4) had pain associated with a medical condition that was not expected to improve with medical or surgical treatment; and (5) were referred by their general physician in England. Individuals were ineligible for participation if they were (1) under 18 yr of age; (2) non-English speaking or illiterate; (3) medically unstable (nonambulatory); (4) in need of further medical attention (e.g., needed further surgery); (5) likely to benefit from further medical investigation or treatment; (6) experiencing a severe personality disorder, psychosis, or suicidal ideation; (7) unable to manage their own affairs; or (8) experiencing substance use addiction and/or needed detoxification (e.g., addicted to pain medications). All individuals were asked questions regarding inclusion and exclusion criteria during a telephone call with research staff.

Participants in this study were 114 CLBP patients, 99 of whom completed enough data to allow for adequate analysis. These CLBP patients included all those referred and eligible for rehabilitation. All patients included in this study completed a demographic questionnaire prior to treatment with questions asking about their age, sex, and the number of surgeries they have had in the past. Additionally, all participants also completed measures of each construct (as described in the “Measures” section) as a standard component of intake evaluation before treatment and then again when discharged from treatment. Rehabilitation for CLBP patients at this pain center typically lasted 4 wk, with patients attending rehabilitation 4 d/wk. Though participants were assessed at admission and 4 wk later at discharge, for the purposes of the present study, we are only using data collected at admission in order to focus on the relationships among pretreatment variables in patients with CLBP. Sixty-nine percent of the participants were female and 31 percent were male. They ranged in age from 18 to 72 yr (mean \pm standard deviation [SD] = 42.6 \pm 12.1), and they ranged in number of surgeries from 1 to 28 (mean \pm SD = 1.26 \pm 2.86).

Sampling Procedures

The present study used archival data collected at a multidisciplinary pain rehabilitation center at Unsted Park Rehabilitation Hospital in Surrey, England, between May 1995 and December 1997. This site’s institutional review board approved this study. All eligible CLBP patients who were referred for treatment were included. If patients met the criteria for participation, they were informed that participation in the rehabilitation program was voluntary prior to admission and that they had the option of seeking treatment elsewhere or withdrawing from treatment at any time. Patients completed the informed consent process and paperwork at a pretreatment assessment interview. Patients were not monetarily compensated for their participation.

Sample Size, Power, and Precision

Power analysis procedures for structural equation modeling (SEM) analyses are widely debated; however, Thompson [29] suggested 10–20 participants per observed variable or about 100 participants for a full analysis, consistent with guidelines from other researchers [30–31]. Given these guidelines and the fact that our preliminary model has five observed variables, we predicted that our available sample of 114 participants, with 99 participants who completed enough data for analysis, would be adequate for the planned analyses.

Measures

All measures were obtained before treatment during a pretreatment assessment interview and were conducted by a research assistant who was not a treating clinician. The research assistant used a standardized script to collect data on the variables of interest. Due to the archival nature of the data, which were obtained at the scale score level, alpha coefficients are not reported.

Biopsychosocial Functioning

The Short Form-36 Health Status Questionnaire (SF-36) was developed to assess health attributes during the past month [32]. The measure includes 36 items that are summarized into eight subscales: Physical Functioning, Role Limitations-Physical, Bodily Pain, Self-Reported General Health, Vitality, Social Function, Role Limitations-Emotional, and Mental Health. Each item is scored differently using yes or no; true or false; or 3-, 5-, or 6-point Likert scales, with higher scores indicating higher quality of life. Sample items include “Compared to one year ago, how would you rate your health in general now?” and “Have you felt downhearted and blue?” Scores are calculated for each subscale by summing responses to individual items and converting to a scale score from 0 (“poor health”) to 100 (“good health”). For the purposes of our study, three subscales were used: Physical Functioning (10 items), Mental Health (5 items), and Social Functioning (2 items), which respectively represent the biological, psychological, and social functioning variables in our study. We chose the Physical Functioning scale to represent biological functioning because of its emphasis on overall health, as opposed to Role Limitations-Physical and Bodily Pain, which are more specific measures assessing how physical health interferes with work and pain intensity. These subscales have been used to measure health attributes in past research [32]. A number of studies support the validity and other psychometric properties of the SF-36 [33–41].

Pain Self-Efficacy

The Pain Self-Efficacy Questionnaire (PSEQ) is a measure of generalized PSE that assesses the patient’s confidence in performing daily activities despite experiencing pain, tolerating pain, and coping with pain [22–23]. The PSEQ contains 10 items rated on a 7-point Likert scale from 0 (“not at all confident”) to 6 (“completely confident”). The sum of all items is the total score, which ranges from 0 (“no PSE”) to 45 (“the most PSE”) and was used in this study. Sample items include “I can enjoy things, despite the pain”

and “I can still accomplish most of my goals in life, despite the pain.” Studies of the psychometric properties of the PSEQ have demonstrated its reliability and validity [22–23].

Pain Severity

The Short-Form McGill Pain Questionnaire (SF-MPQ) was developed to assess pain [39]. The measure contains 15 items that describe different aspects of pain and two subscales: Sensory Pain (11 items) and Affective Pain (4 items). Each item is rated on a 4-point Likert scale from 0 (“none”) to 3 (“severe”), and sample items include “burning,” “throbbing,” and “sickening.” The sum of all items is the evaluative pain score, which ranges from 0 (“no pain”) to 45 (“severe pain”) and was used in this study. The evaluative component of the pain experience consists of a subjective overall intensity combining both the sensory and affective qualities of pain. The SF-MPQ has been widely used in studies related to chronic pain and has sound psychometric qualities [42–45].

Analyses

SEM was used to test whether PSE mediated the relationship between biological, psychological, and social functioning and pain severity. In order to test the hypothesized model (**Figure**), we used a bootstrap sampling procedure. Researchers have recently recommended using bootstrap resampling methods to test for the significance in mediated models [46–47]. A strength of the bootstrapping technique is that it maximizes the statistical power, reducing the chance of type II error [46]. In the present study, we specified 1,000 bootstrap iterations and used 95 percent bias-corrected confidence intervals and bootstrap estimates of indirect, direct, and total effects [46].

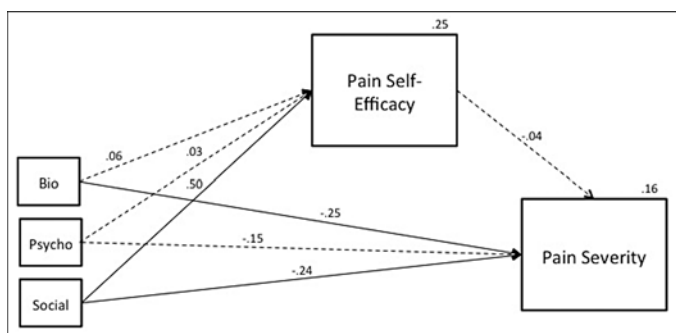


Figure. Significant (bold lines) and nonsignificant (dashed lines) paths in hypothesized model. Bio = biological, Psycho = psychological.

SPSS version 16.0 (IBM Corporation; Armonk, New York) was utilized to conduct a preliminary examination of the data. Cases missing more than 24 percent of the data were eliminated ($n = 15$), as has been recommended in similar research [48]. A sufficient amount of data for analysis (>76% [48]) was present for a total of 99 participants. In order to proceed with the planned analyses, multiple imputation (MI) [48] was used to manage missing data. Scale-level imputation was used because item-level scores were not present in the databases. Research suggests that although item-level imputation may have a power advantage, parameter estimates are not biased by scale-level imputation [49]. All variables were included in the imputation models, and variables were constrained to their scale-appropriate maximum and minimum possible values. Pooled values from five MI data sets were used for preliminary data analysis; however, because analyses using MI data sets will not comply with SPSS Amos programming, the first imputed data set was used for SEM analysis. Several steps were taken to assess the normality of the data for the 99 participants by using procedures outlined by Byrne [50] and Field [51].

RESULTS

Descriptive Statistics

Descriptive statistics, including the ranges, means, SDs, and bivariate correlations, for each of the measures employed in this study are shown in **Tables 1** and **2**. No sex differences were observed across measures of interest.

Table 1.

Possible range of values compared with range of values in present sample.

| Measure | Possible Range | | Sample Range | |
|---------|----------------|---------|--------------|---------|
| | Minimum | Maximum | Minimum | Maximum |
| SF-MPQ | 1 | 5 | 1 | 5 |
| PSEQ | 0 | 60 | 6 | 57 |
| SF-Bio | 0 | 100 | 0 | 80 |
| SF-Psy | 0 | 100 | 8 | 88 |
| SF-Soc | 0 | 100 | 0 | 100 |

PSEQ = Pain Self-Efficacy Questionnaire, SF-36 = Short Form-36 Health Status Questionnaire, SF-Bio = SF-36 Biological subscale, SF-MPQ = Short-Form McGill Pain Questionnaire, SF-Psy = SF-36 Psychological subscale, SF-Soc = SF-36 Social subscale.

Table 2.Mean \pm standard deviation and Pearson correlation values for SF-MPQ, PSEQ, SF-Bio, SF-Psy, and SF-Soc.

| Measure | SF-MPQ | PSEQ | SF-Bio | SF-Psy | SF-Soc |
|---------|-----------------|-------------------|--------------------|--------------------|--------------------|
| SF-MPQ | 3.47 \pm 0.92 | -0.23* | -0.31 [†] | -0.30 [†] | -0.35 [†] |
| PSEQ | — | 21.66 \pm 10.04 | 0.15 | 0.26 [†] | 0.51 [†] |
| SF-Bio | — | — | 33.19 \pm 20.74 | 0.16 | 0.16 |
| SF-Psy | — | — | — | 49.33 \pm 17.88 | 0.44 [†] |
| SF-Soc | — | — | — | — | 34.00 \pm 25.01 |

* $p < 0.05$.[†] $p < 0.01$.

PSEQ = Pain Self-Efficacy Questionnaire, SF-36 = Short Form-36 Health Status Questionnaire, SF-Bio = SF-36 Biological subscale, SF-MPQ = Short-Form McGill Pain Questionnaire, SF-Psy = SF-36 Psychological subscale, SF-Soc = SF-36 Social subscale.

Tests of Mediation

The unstandardized results from the hypothesized model are shown in **Table 3**. Only one direct effect was significant between social functioning and PSE ($\beta = 0.196$; 95% CI = -0.130 to 0.273, $p = 0.002$). Additionally, there were no significant indirect effects for biological functioning ($\beta = 0.000$; 95% CI = -0.002 to 0.000, $p = 0.58$), psychological functioning ($\beta = 0.000$; 95% CI = -0.002 to 0.001, $p = 0.70$), or social functioning ($\beta = -0.001$; 95% CI = -0.006 to 0.003, $p = 0.74$). However, there were significant total effects for social functioning ($\beta = -0.009$; 95% CI = -0.016 to -0.003, $p = 0.007$) and biological functioning ($\beta = -0.011$; 95% CI = -0.019 to -0.004, $p = 0.002$). These results suggest that higher social functioning predicts higher PSE and lower pain severity. Similarly,

higher biological functioning predicts lower pain severity. However, the results of this analysis also suggest that the relationships between biological, psychological, and social functioning and pain severity are not mediated by PSE.

DISCUSSION

The overall goal of the present study was to examine the relationship between biopsychosocial functioning and pain severity and also to evaluate whether PSE indirectly affects this relationship. Our findings offer partial support for our first hypothesis that biological, psychological, and social functioning would separately predict pain severity. Specifically, lower social and biological functioning both

Table 3.

Hypothesized model: Bootstrap results to test significance of mediation (unstandardized values).

| Path/Effect | β | SE | 95% CI | | p -Value |
|--|---------------|--------------|---------------|---------------|--------------|
| | | | Lower | Upper | |
| <i>a</i> SF-Bio \rightarrow PSEQ | 0.029 | 0.042 | -0.053 | 0.116 | 0.48 |
| <i>a</i> SF-Psy \rightarrow PSEQ | 0.018 | 0.049 | -0.097 | -0.106 | 0.71 |
| <i>a</i> SF-Soc \rightarrow PSEQ | 0.196 | 0.035 | 0.130 | 0.273 | 0.002 |
| <i>b</i> PSEQ \rightarrow SF-MPQ | -0.004 | 0.010 | -0.028 | 0.015 | 0.76 |
| <i>c</i> (total effect) SF-Bio \rightarrow SF-MPQ | -0.011 | 0.004 | -0.019 | -0.004 | 0.002 |
| <i>c</i> (total effect) SF-Psy \rightarrow SF-MPQ | -0.008 | 0.005 | -0.018 | 0.002 | 0.13 |
| <i>c</i> (total effect) SF-Soc \rightarrow SF-MPQ | -0.009 | 0.003 | -0.016 | -0.003 | 0.007 |
| <i>c'</i> (direct effect) SF-Bio \rightarrow SF-MPQ | -0.011 | 0.004 | -0.019 | -0.004 | 0.003 |
| <i>c'</i> (direct effect) SF-Psy \rightarrow SF-MPQ | -0.008 | 0.005 | -0.018 | 0.002 | 0.12 |
| <i>c'</i> (direct effect) SF-Soc \rightarrow MPQ | -0.008 | 0.004 | -0.016 | -0.001 | 0.03 |
| <i>a</i> \times <i>b</i> (indirect effect) SF-Bio \rightarrow SF-MPQ | 0.000 | 0.001 | -0.002 | 0.000 | 0.58 |
| <i>a</i> \times <i>b</i> (indirect effect) SF-Psy \rightarrow SF-MPQ | 0.000 | 0.001 | -0.002 | 0.001 | 0.70 |
| <i>a</i> \times <i>b</i> (indirect effect) SF-Soc \rightarrow SF-MPQ | -0.001 | 0.002 | -0.006 | 0.003 | 0.74 |

Note: Bold font indicates significant effects.

CI = confidence interval, PSEQ = Pain Self-Efficacy Questionnaire, SE = standard error, SF-36 = Short Form-36 Health Status Questionnaire, SF-Bio = SF-36 Biological subscale, SF-MPQ = Short-Form McGill Pain Questionnaire, SF-Psy = SF-36 Psychological subscale, SF-Soc = SF-36 Social subscale.

significantly predicted higher pain severity. However, psychological functioning did not significantly predict pain severity. The present findings that lower social functioning and biological functioning predict higher pain severity seem to be well reflected in previous literature [3,52].

Our second hypothesis that PSE would mediate the relationship between biopsychosocial functioning and pain severity was not supported. Biological and psychological functioning did not predict PSE, which was surprising given the support in the literature for this relationship. However, we did find that social functioning significantly predicted PSE. Our results correspond with previous research, which has found that lower social functioning predicts PSE [13]. Specifically, lower levels of PSE are associated with lower levels of social support and higher levels of anxiety and depressive symptoms. It is possible that our sample size was too small to detect mediation effects, so future research should utilize larger sample sizes.

Our finding that psychological functioning did not significantly predict pain severity or PSE is a surprising one. Previous studies have documented the relationship between psychological functioning and pain severity [9,12–13,16]; however, our study is the first to our knowledge to have examined both PSE and psychological functioning in CLBP patients in the same model. It is likely that in this study, PSE and psychological functioning shared more variance, because these are very similar constructs, and therefore, the unique contribution of psychological functioning was difficult to disentangle from PSE and no significant effects were found. Future research should seek to clarify the role of psychological functioning within the biopsychosocial mode and its unique influence on pain severity.

Limitations

Although the present study offers valuable findings, it is important to recognize several potential limitations. First, due to the cross-sectional nature of the data analysis, we cannot draw strict conclusions about the nature of these relationships. Second, we did not control for factors such as socioeconomic status or education, so we cannot rule out the possibility that other constructs might be affecting our findings. Third, the results of this study cannot be generalized beyond the characteristics of the current sample. Fourth, the findings are based on self-report measures that are susceptible to a variety of threats to

validity. Fifth, our sample size of 99 participants was adequate for the planned analyses; however, a larger sample would likely be more representative.

Valuable Findings

Despite these limitations, this study has several strengths that make it a unique and important contribution to this area of literature. Little research involving CLBP patients includes all three levels of biopsychosocial functioning [52]. Studies on CLBP often investigate behavioral and psychological functioning (e.g., disability, pain behaviors, self-efficacy, and catastrophizing [9,12–13,15–16,24–25]), but few studies include social functioning (e.g., social expectations, relationships, or social support [10,52]). Hence, our finding that social functioning predicts PSE and pain severity reiterates the importance of understanding social functioning in the overall model of chronic pain.

Future Research

One logical extension of the present research would be for researchers to investigate and implement possible strategies that aid in increasing social functioning through social support groups and/or family therapies in multidisciplinary rehabilitation programs. These programs could also target deficits in interpersonal and communication skills through techniques like assertiveness training. In addition, further research should investigate how deficits in social functioning may represent barriers to treatment. Furthermore, the present study suggests that there is a need for more research targeted at understanding the relationship between social functioning, PSE, and pain severity. In other words, it is necessary to disentangle how higher social functioning lowers pain severity, if not through higher PSE, possibly through lower catastrophizing, for example. Moreover, future research should broaden the investigation of social functioning on pain severity within the biopsychosocial model of CLBP.

CONCLUSIONS

In sum, social functioning is an important factor in predicting pain severity and PSE and is necessary to consider when trying to understand the development of patient difficulties in patients with CLBP and when designing and implementing rehabilitation programs. These results provide valuable insight into the role of social functioning

within the biopsychosocial model that contributes to the expense of healthcare, patient distress, and increased pain severity. These findings might be utilized to better understand and predict which CLBP patients will develop greater difficulties and to improve social functioning for the clinical care of patients in multidisciplinary rehabilitation programs.

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Author Contributions:

Study concept and design: A. L. Koenig, A. E. Kupper, J. R. Skidmore, K. M. Murphy.

Analysis and interpretation of data: A. L. Koenig, A. E. Kupper, J. R. Skidmore, K. M. Murphy.

Drafting of manuscript: A. L. Koenig, A. E. Kupper, J. R. Skidmore, K. M. Murphy.

Critical revision of manuscript for important intellectual content: A. L. Koenig, A. E. Kupper, J. R. Skidmore, K. M. Murphy.

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