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The contribution of pain and depression to self-reported sleep disturbance in patients with rheumatoid arthritis

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ABSTRACT

The objective of this article is to assess the contribution of disease activity, pain, and psychological factors to self-reported sleep disturbance in patients with rheumatoid arthritis (RA), and to evaluate whether depression mediates the effects of pain on sleep disturbance. The sample included 106 patients with confirmed RA who participated in an assessment of their disease activity, pain, psychological functioning, and sleep disturbance during a baseline evaluation prior to participating in a prospective study to help them manage their RA. Self-measures included the Rapid Assessment of Disease Activity in Rheumatology, the SF-36 Pain Scale, the Helplessness and Internality Subscales of the Arthritis Helplessness Index, the Active and Passive Pain Coping Scales of the Pain Management Inventory, the Center for Epidemiological Studies Depression Scale, and the Pittsburgh Sleep Quality Index. Hierarchical multiple regression analysis confirmed that higher income, pain, internality, and depression contributed independently to higher sleep disturbance. A mediational analysis demonstrated that depression acted as a significant mechanism through which pain contributed to sleep disturbance. Cross-sectional findings indicate that pain and depression play significant roles in self-reported sleep disturbance among patients with RA. The data suggest the importance of interventions that target pain and depression to improve sleep in this medical condition.

1. Introduction

Rheumatoid arthritis (RA) is an inflammatory autoimmune disorder that is characterized by joint pain, joint swelling, fatigue, and, in many patients, poor sleep [1,10,32]. Several studies have found sleep fragmentation in RA patients, contributing to low sleep efficiency, awakenings from sleep, and reports the next day of poor sleep quality [8,16]. Studies have found that between 50% and 75% of RA patients report problems with their sleep, as indicated by difficulty falling asleep, staying asleep, nonrestorative sleep, and excessive daytime sleepiness [1]. Sleep complaints in RA are 2–3 times more prevalent than in the general population, and significantly higher than in patients with other medical conditions such as obesity, hypertension, and respiratory illness [27]. Importantly, sleep disturbance may contribute to greater pain, disease activity, and mood disturbance, creating a cascade of dysfunction for afflicted patients [20,34].

A variety of variables have been suggested as possible causes of sleep disturbance in RA, including inflammatory disease activity, joint pain, other sleep disorders such as sleep apnea and restless legs, and psychological distress, most notably depression and/or anxiety [1,34]. A key issue in this research concerns the relative influence of these variables on sleep. A clearer understanding of the factors contributing to sleep disturbance in RA would inform the development of rational, empirically based treatments to improve sleep in this patient population.

Because 20% to 30% of RA patients have significant mood disturbance [7], investigators have examined the role of depression, along with pain and disease activity, in RA-related sleep disturbance [1,22,34]. Nicassio and Wallston [22] showed that a 2-item scale of sleep disturbance was associated with depression independently of pain, and that prior pain predicted worsening sleep disturbance

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over time. More recently, Wolfe et al. [34] found that pain and depression contributed independent variance to sleep disturbance in RA, as measured by a visual analogue scale and Medical Outcomes Study sleep problem indexes. However, these studies did not address the possibility that pain may lead to sleep disturbance through its effect on depression [4].

Several studies in the RA literature have shown that illness beliefs such as helplessness and internality (perceived control) and pain coping mechanisms have very significant influences on pain, mood disturbance, and disability in both cross-sectional and longitudinal studies [5,23,31]. To date, research has not examined the potential contribution of these factors to sleep disturbance in this population beyond their effects on pain outcomes. Thus, current knowledge of the role of psychological factors in RA sleep disturbance is very limited.

1.1. Study objectives

This research adopted a multidimensional approach to understanding sleep disturbance in RA [9,17], in which the major objective was to determine whether psychological variables would contribute independently to sleep disturbance beyond the effects of disease activity and pain, in a sample of RA patients recruited from the greater Los Angeles area. A secondary objective was to determine whether depressive symptoms would mediate the relationship between pain and sleep disturbance. We assessed sleep disturbance with the Pittsburgh Sleep Quality Index [6], a valid, well-established self-report measure of sleep disturbance for medical patients.

2. Materials and methods

2.1. Patient recruitment

The research was approved by the Institutional Review Board Committee for the Protection of Human Subjects at both the University of California, Los Angeles (UCLA) and Cedars Sinai Medical Center (CSMC). RA patients were recruited through advertisements placed in local newspapers and written announcements posted in clinic offices in the Departments of Rheumatology at UCLA and CSMC, Los Angeles, to participate in a clinical research program designed to help them manage their RA. The project coordinator at UCLA conducted a telephone screening with prospective patients, who were referred to CSMC, where the study rheumatologist (MW) conducted a diagnostic evaluation that included assessments of tender and swollen joints and disease activity using the Disease Activity Score Calculator for Rheumatoid Arthritis to confirm a diagnosis of RA. Eligible participants (1) were 18 years of age or older; (2) met American College of Rheumatology revised criteria for RA; (3) were stable on a disease-modifying drug regimen for 3 months prior to study entry; (4) had a stable disease course for 3 months; (5) did not have serious comorbid medical conditions such as diabetes, congestive heart failure, renal failure, or cancer; and (6) were not pregnant. Eligible patients were referred to UCLA for an evaluation of psychiatric status, physical functioning, and psychosocial adjustment. The project team held a consensus meeting to evaluate results from the Structured Clinical Interview for DSM (*Diagnostic and Statistical Manual of Mental Disorders*) Disorders [29]. Those patients who had a serious psychiatric condition such as bipolar disorder, psychosis, or posttraumatic stress disorder, or who were at risk for suicide, were excluded from the research.

2.2. Data collection

The findings of this research are derived from a baseline evaluation of participants who agreed to be followed over a 1-year period during their participation in a clinical trial that evaluated behavioral interventions for managing RA. The project coordinator administered measures of self-reported disease activity, pain, illness beliefs, pain coping, and depression, which are the focus of analyses. Patients also completed self-report measures of quality of life, disability, and social functioning, which are not included in this report. In addition, participants provided information regarding their age, gender, and socioeconomic status (ie, years of education and annual income).

2.2.1. Medication use

Data were obtained from each patient on the following types of medication: (1) nonsteroidal antiinflammatory drugs (NSAIDs)/analgesics; (2) biologic disease-modifying antirheumatic drugs (DMARDs), including abatacept (Orencia; Bristol-Myers Squibb, Princeton, NJ, USA), adalimumab (Humira; Abbott Laboratories, Abbott Park, IL, USA), etanercept (Enbrel; Immunex Corporation, Thousand Oaks, CA, USA), and infliximab (Remicade; Janssen Biotech Inc, Horsham, PA, USA); (3) synthetic DMARDs, including azathioprine (Imuran; Aspen Pharmacare Australia Pty Ltd, St. Leonards, NSW, Australia), hydroxychloroquine (Plaquenil; Sanofi-aventis, Bridgewater, NJ, USA), leflunomide (Arava; Sanofi-aventis), methotrexate (Rheumatrex, DAVA Pharmaceuticals, Inc, Fort Lee, NJ, USA; Trexall; Barr Laboratories, Inc, Pomona, NJ, USA), and sulfasalazine (Azulfidine; Pfizer, New York, NY, USA), as well as corticosteroids (prednisone); and (4) "other" (drugs for other medical conditions, including psychotropics).

2.2.2. Disease activity and pain

The Rapid Assessment of Disease Activity in Rheumatology (RADAR) assessed self-reported measure of disease activity [17]. The RADAR consists of questions about past and current disease activity, pain, morning stiffness, and the degree of pain/tenderness in 10 joints on the right and left sides of the body. Research has shown the RADAR to be an efficient, valid proxy for physician assessments of disease activity and joint pain in RA [35].

In addition to the RADAR, the SF-36 Pain Scale [33] was adopted to assess global pain reported by patients over the preceding 4 weeks. The scale consists of 2 items, "How much bodily pain did you have?" (0 = none to 5 = very severe), and "How much did pain interfere with your normal work (including both work outside the home and housework)? (0 = not at all to 4 = extremely).

2.2.3. Psychological functioning

2.2.3.1. Illness beliefs. The Helplessness and Internality Subscales of the Arthritis Helplessness Index [23] were used to evaluate patients' beliefs about their ability to manage RA. The 5-item Helplessness and 7-item Internality Subscales of the Arthritis Helplessness Index provide an assessment of contrasting beliefs about RA, with helplessness assessing the perceived inability to affect RA outcomes (eg, pain, disease course), and internality assessing perceived control over RA ("managing arthritis is my own responsibility") [30].

2.2.3.2. Pain coping. Pain coping strategies reported by patients were assessed with the Pain Management Inventory, developed by Brown and Nicassio [5]. The Pain Management Inventory consists of Passive and Active Coping Subscales, reflecting divergent styles of managing pain when pain reaches a moderate or high level of intensity. The 11-item Passive Coping Subscale measures tendencies to limit functioning or avoid pain, while the 7-item Active Coping Subscale measures tendencies to reduce pain (eg, distraction) or to function in spite of the pain (engaging in exercise, physical activity).

2.2.3.3. Depressive symptoms. The Center for Epidemiological Studies Depression Scale (CES-D) [26] was used to assess the severity of

depressive symptoms. The CES-D is a 20-item scale in which patients report the existence of depressive symptoms over the past month. The measure was designed to assess the existence of depressive symptomatology in community samples and nonpsychiatric groups, and has been used extensively in studies with patients with RA and other chronic illnesses because very few of its items overlap with medical symptoms that can artificially elevate the total score [3,7].

2.2.4. Sleep disturbance

The Pittsburgh Sleep Quality Index (PSQI) [6] assessed sleep disturbance reported by patients over the previous month. The PSQI consists of 19 individual items that assess subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. A score of 5 or greater has high diagnostic specificity for detecting clinical sleep impairment as defined by the diagnostic criteria for insomnia in the general population. The PSQI has been used to detect sleep disturbance in a range of psychiatric and medical populations [11,14].

2.3. Statistical approach

Data analyses were conducted using SPSS 19.0 software (SPSS Inc, Chicago, IL, USA). Descriptive statistics for variables of interest in this study are presented as percentages, means, and SDs. To establish the best set of predictor variables, bivariate correlation analyses were conducted. Variables shown significantly associated with sleep disturbance ($P < 0.05$) were evaluated using a hierarchical multiple regression approach in which blocks of variables were entered according to the following sequence: (1) the control variables of sociodemographic factors and medication use, (2) disease activity/pain, (3) illness beliefs and pain coping, and (4) depressive symptoms. The purpose of this strategy was to isolate the contribution of each block of factors to sleep disturbance after controlling for all variables entered on preceding blocks. Finally, a model that examined whether depressive symptoms mediated the effects of pain on sleep disturbance was evaluated.

3. Results

Data on demographic characteristics, model variables, and sleep are presented in Table 1. The sample (n = 106) was predominantly female (85%), with an average age of 56.2 years. The sample consisted of patients of mixed ethnicity from Caucasian, Latino, African-American, Asian, and Native-American backgrounds. Participants reported having RA for an average duration of 12 years at study onset. Participants reported a combination of biologic DMARDs, synthetic DMARDs, NSAIDs/analgesics, and other medications (eg, psychotropics) used to manage their RA and comorbid medical/psychiatric problems. Synthetic DMARDs were the most commonly used medication reported by patients. A large range of scores was found on the PSQI, although significant sleep disturbance overall was found in the sample, as reflected by a mean score of 5.93.

Correlations among model variables and PSQI scores are presented in Table 2. Income ($P = 0.001$), RADAR scores ($P = 0.037$), SF-36 pain ($P < 0.001$), helplessness ($P = 0.005$), and depressive symptoms ($P < 0.001$) were all correlated positively with PSQI, while internality was correlated negatively with PSQI ($P = 0.014$). No significant associations emerged between PSQI and education, gender, illness duration, biologic DMARDs, synthetic DMARDs, NSAIDs/analgesics, other medications, passive coping, or active coping, thus these variables were not included in subsequent analyses.

Next, variables significantly associated with sleep disturbance were evaluated using a hierarchical multiple regression approach.

Table 1
Summary statistics on sample and model variables.

Variable	Mean ± SD or n (%)	Range
Sample characteristics		
Age (years)	56.22 ± 12.45	22–79
Annual income ($)	51,142 ± 18,631	17,644–141,527
Education (years)	15.96 ± 2.39	12–21
Gender		
Female	90 (85)	
Male	16 (15)	
Ethnicity		
Caucasian	56 (52.8)	
Latino	15 (14.2)	
African American	11 (10.4)	
Asian/Pacific Islander	6 (5.6)	
American Indian	1 (0.01)	
Other ethnicity	5 (4.7)	
Not specified	12 (11.3)	
Medications use		
Biologic DMARDs	44 (41.5)	
Synthetic DMARDs	77 (72.6)	
NSAIDs/Analgesics	64 (60.4)	
Other medications	69 (65.1)	
Illness duration (years)	11.97 ± 11.39	<1–53
Model variables		
RADAR	11.47 ± 9.30	0–36
SF-36 Pain	28.27 ± 9.99	10–55
Helplessness	14.80 ± 3.94	7–28
Internality	30.16 ± 5.97	7–42
Active coping	22.80 ± 4.72	12–35
Passive coping	24.90 ± 6.99	12–43
Depression (CES-D)	8.89 ± 9.19	0–39
PSQI	5.93 ± 3.64	0–17

DMARD, disease-modifying anti-rheumatic drug; NSAID, nonsteroidal antiinflammatory drug; RADAR, Rapid Assessment of Disease Activity in Rheumatology; CES-D, Center for Epidemiological Studies Depression Scale; PSQI, Pittsburgh Sleep Quality Index.

Specifically, a 4-step approach was used to assess the unique contribution of the set of predictors: income was entered by itself in step 1, followed by the disease activity/pain variables in step 2 (RADAR and SF-36 pain), the illness belief variables in step 3 (helplessness and internality), and depression in step 4 (CES-D). At step 1, higher annual income was associated with sleep disturbance ($\beta = .31$) and accounted for 9.7% of the variance in PSQI scores ($F = 11.23$, $P = 0.001$). The entry of RADAR scores and SF-36 pain at step 2 significantly improved the predictive ability of the model ($F_{inc} = 7.98$, $P < 0.001$); however, only higher SF-36 pain was uniquely related to sleep disturbance ($\beta = .33$). At step 3, the addition of illness beliefs (ie, helplessness and internality) added significantly to the model ($F_{inc} = 3.90$, $P = 0.023$), although only lower internality emerged significant as an individual predictor of sleep disturbance ($\beta = -.20$). On the last step, higher depression was significantly associated with sleep disturbance and accounted for 6.8% of the variance in PSQI scores ($F = 10.20$, $P = 0.002$). After all variables had entered the regression equation, SF-36 pain ($\beta = .21$), internality ($\beta = -.17$), depressive symptoms ($\beta = .30$), and income ($\beta = .29$) retained significance as individual predictors. The final regression model, taking into account the contribution of all variables, explained 34.4% of the variance in PSQI scores ($F = 8.64$, $P < 0.001$). Hierarchical multiple regression analysis findings are summarized in Table 3.

Finally, we examined whether depressive symptoms would mediate the effects of pain on sleep disturbance. A series of regression analyses were conducted following the criteria described by Baron and Kenny [2] to establish mediation. In order to demonstrate mediation, the paths from pain to depressive symptoms and from pain to sleep disturbance would have to be confirmed. Then, the path from pain to sleep disturbance would be either

Table 2
Correlations among study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Age	–															
2. Income	−.09	–														
3. Education	−.01	.14	–													
4. Gender	.18	−.13	.17	–												
5. Illness duration	.17	.05	−.13	.04	–											
6. Biologic DMARDs	−.13	.17	.02	−.12	.15	–										
7. Synthetic DMARDs	.13	.07	.02	−.12	.13	.01	–									
8. NSAIDs/analgesics	.00	.10	−.12	−.03	.15	.19	−.02	–								
9. Other medication	.11	.08	−.05	−.15	−.06	.12	.29**	.11	–							
10. RADAR	.01	−.06	.05	−.09	.04	.04	.09	−.04	−.02	–						
11. SF-36 Pain	−.04	.06	.17	−.06	−.02	−.09	.03	.01	−.20*	.58***	–					
12. Helplessness	−.09	.00	−.08	−.07	−.02	.02	−.02	−.11	−.02	.31**	.41***	–				
13. Internality	.12	−.07	−.01	.03	.03	.18	−.06	.14	.01	.07	.00	−.21*	–			
14. Active coping	−.01	−.06	−.02	−.06	.09	.10	.07	.05	.10	.10	−.06	−.24*	.42***	–		
15. Passive coping	−.13	.06	−.03	−.18	−.14	.01	−.05	−.12	−.07	.25*	.42***	.51***	−.17	−.24*	–	
16. Depression	−.08	−.01	−.14	−.28**	−.08	−.07	−.03	−.01	−.15	.26**	.39**	.44***	−.17	−.19	.42***	–
17. PSQI	−.18	.31**	.06	−.13	.12	.04	.14	.10	.11	.20*	.37***	.27**	−.24*	−.02	.13	.42***

DMARD, disease-modifying anti-rheumatic drug; NSAID, nonsteroidal antiinflammatory drug; RADAR, Rapid Assessment of Disease Activity in Rheumatology.
Note. Gender was dummy coded 0 = female, 1 = male; the gender-medication and medication-medication figures are phi coefficients; all other coefficients involving gender or medication are point biserial correlations.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

eliminated for full mediation, or significantly reduced for partial mediation, after accounting for the effects of depressive symptoms on sleep disturbance.

This framework was tested in a series of regressions. First, the path from SF-36 pain to sleep disturbance was examined while controlling for annual income. Pain made a significant contribution to sleep disturbance ($F = 16.03$, $P < 0.001$) at this step. In the second regression examining the path between pain and depressive symptoms, higher pain was associated with higher depressive symptoms ($F = 18.14$, $P < 0.001$). Finally, the third regression examined the contribution of depressive symptoms to sleep disturbance when it was entered jointly with pain to determine if depressive symptoms would account for the relationship between pain and sleep disturbance. The contribution of these variables to sleep disturbance was highly significant ($F = 15.70$, $P < 0.001$). Both pain ($\beta = .22$, $P = 0.016$) and depressive symptoms ($\beta = .34$, $P < 0.001$) contributed independently to PSQI scores. However, while pain retained its significance, when the Preacher and Hayes [25] bootstrapping method was applied to test the model, depressive symptoms significantly reduced the relationship between pain and sleep disturbance. Thus, depression partially mediated the effects of pain, substantiating an indirect path from pain to sleep disturbance through depressive symptoms. This indirect effect ($b = .20$, $SE = .07$) accounted for 37.46% of the total effects in the model tested. A summary of the mediational analysis, depicting direct and indirect effects, is presented in Fig. 1.

4. Discussion

This research confirmed that patients with RA have a high prevalence of sleep disturbance, converging with evidence from other studies in patients with RA and other chronic pain populations

Table 3
Hierarchical multiple regression analysis of PSQI scores.

Variable	β	*t*	sr^2 (%)	R^2	*df*	F	ΔR^2	ΔF
Step 1				.097	1, 104	11.23**		
Income	.31	33.35**	9.73					
Step 2				.220	3, 102	9.57***	.112	7.98***
Income	.29	3.34**	8.53					
RADAR	.03	0.28	0.06					
SF-36 Pain	.33	3.08**	7.24					
Step 3				.276	5, 100	7.63***	.056	3.90*
Income	.28	3.30**	7.90					
RADAR	.04	0.38	0.10					
SF-36 Pain	.29	2.60*	4.88					
Helplessness	.10	1.02	0.74					
Internality	−.20	−2.28*	3.76					
Step 4				.344	6, 99	8.64***	.068	10.20**
Income	.29	3.55**	8.35					
RADAR	.03	0.29	0.06					
SF-36 Pain	.21	1.98*	2.59					
Helplessness	.01	0.05	0.002					
Internality	−.17	−2.00*	2.62					
Depression	.30	3.19***	6.76					

PSQI, Pittsburgh Sleep Quality Index; RADAR, Rapid Assessment of Disease Activity in Rheumatology.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

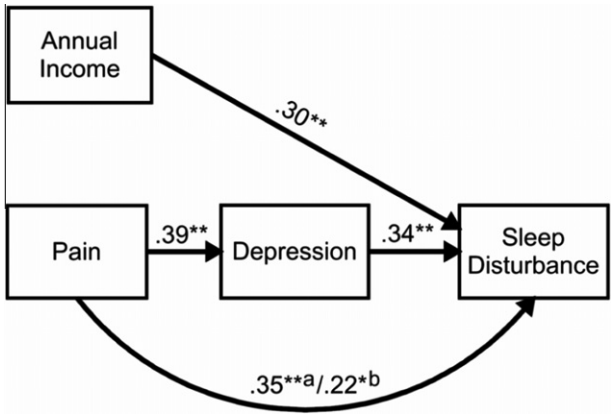

Fig. 1. Relationship between pain, depression, and sleep disturbance. *Note.* The figure illustrates direct relationships between pain and sleep disturbance, and between depression and sleep disturbance. The relationship between pain and sleep disturbance is partly mediated by depression, as reflected by the path from pain to depression, and the path from depression to sleep disturbance. Although attenuated, the relationship between pain and sleep disturbance remains significant after controlling for depression. [a]Before accounting for depression; [b]after accounting for depression. $^{*}P < 0.05$; $^{**}P < 0.001$.

[1,12]. More than half of the sample had scores on the PSQI that exceeded the cutoff for determining sleep disturbance. Importantly, this study evaluated an integrated framework [9] for examining potential determinants of sleep disturbance in this population. Previous studies have raised questions about the origins of sleep disturbance in patients with this medical condition [1,34], and this research sought to clarify this question by identifying individual variables that contribute to poor sleep.

Hierarchical multiple regression findings demonstrated that SF-36 pain and depressive symptoms were the most important predictors of sleep disturbance, each being individually associated with higher PSQI scores. Also, lower internality and higher income were associated with greater sleep disturbance. It is unclear why patients with higher income would have greater sleep disturbance, but it is possible that this finding could have reflected the unique features of this urban sample of RA patients. Income was unrelated to other variables in the model. Importantly, self-reported disease did not predict sleep disturbance when it competed with the SF-36 pain scale in the multivariate analysis. Differences between the 2 scales in the approach to the measurement of pain could have been responsible for this result. As a more general index of pain, capturing both pain intensity and impairment, the SF-36 may be more sensitive to sleeping difficulty than the RADAR, which assesses pain specifically in the joints. These findings corroborate the results of previous studies that have demonstrated a positive relationship between self-reported pain and sleep disturbance in other chronic pain populations [28].

Despite the importance of pain, depressive symptoms accounted for more variance than pain in explaining sleep disturbance. Since depression is a highly prevalent problem in RA [18], its role in sleep disturbance assumes considerable importance, and adds to the numerous health-related comorbidities that are already known to be associated with RA, such as disability, adherence problems, and limitations in quality of life [10,32]. Compounding the problem of depression in RA is evidence that mood disturbance is often not detected or managed in rheumatology care [21]. Thus, sleep problems, caused or exacerbated by depression, may similarly go unaddressed.

Prior research has documented a significant relationship between chronic pain and depression in RA, with evidence showing that pain is more likely to contribute to depression over time than to be the result of depression [4]. Adopting procedures recommended by Baron and Kenny [2], the analyses showed that depressive symptoms largely mediated the relationship between pain and sleep disturbance, but that pain continued to be independently related to PSQI scores. A test of mediation confirmed the existence of a direct and indirect relationship between pain and sleep disturbance through depressive symptoms [25]. The results suggest that sleep disturbance in RA may result, in part, from a cyclical pattern in which heightened pain creates risk for mood disturbance that, in turn, interferes with sleep. The fact that pain continued to contribute to sleep disturbance independently also suggests that pain may affect other potential mechanisms such as sleep fragmentation and nightly awakenings, leading to reduced sleep efficiency [1] and a feeling of nonrestorative sleep.

It is noteworthy that low internality was associated with greater sleep disturbance, while helplessness, passive coping, and active coping were unrelated to PSQI scores in the regression analysis. The finding that internality, as a measure of low perceived control over arthritis, may reflect sleeping difficulty in RA, is a novel result. In contrast, while being unrelated directly to sleep disturbance, helplessness and passive coping may affect sleep through their indirect effects on pain and depression [5,23], which this research has shown, affect sleep directly. More complex models exploring the direct and indirect mechanisms through which illness beliefs and pain coping contribute to sleep disturbance could be developed to explore this issue in future studies.

The results of this research provided evidence of the importance of pain and depressive symptoms in explaining sleep disturbance in patients with RA. Although the findings supported an initial test of the model that was examined, some methodological limitations of the study prevent a conclusive interpretation of the data. The cross-sectional nature of the findings precluded an understanding of the directionality between pain, depression, and sleep disturbance. Thus, other models that examine the dynamic relationship between pain, mood, and sleep in RA warrant further examination. Longitudinal studies incorporating mediational models are needed to determine whether pain over time contributes to depression, which then leads to poor sleep. While the model and results of this study suggest the plausibility of this mechanism, it is also possible that an alternate model in which poor sleep triggers subsequent pain, depression, and functional problems may be viable as well [15,24]. Also, since the research relied on self-reports of sleep disturbance, the findings require further clarification using objective polysomnographic assessment. Finally, the participants in this research, who volunteered from the surrounding urban area, may not have been representative of RA patients who either were not receiving active medical care or who were not as motivated to enroll in research of this type.

4.1. Conclusions

Despite these limitations, the data have implications for the management of sleep disturbance in patients with RA. Specifically, the treatment of sleep disturbance in RA should incorporate strategies to reduce pain and depressed mood, along with other established behavioral approaches such as cognitive behavior therapy, stimulus control instructions, and sleep restriction that have proven to be effective in other sleep-disordered populations [13,19]. This comprehensive approach would address the relevant factors that either cause or maintain sleep disturbance in this medical condition.

Conflict of interest statement

The authors have no conflicts of interest in the conduct or publication of this research.

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