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Evaluating Evidence for the Role of Sleep in Fibromyalgia: A Test of the Sleep and Pain Diathesis Model

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Abstract

The Sleep and Pain Diathesis (SAPD) Model predicts that sleep quality is related to Fibromylagia (FM) outcomes such as disability and depression and that these relationships are mediated by both pain and impaired emotional dysregulation. The purpose of this paper is to provide a preliminary test of this model using cross-sectional data. 35 adult women, who had been living with FM for an average of 13 years, completed a battery of questionnaires that included reports of pain, sleep, affect, and disability. Consistent with this model, FM patients who reported more disrupted sleep also reported higher levels of psychological disability (i.e., BDI depression symptoms) and physical disability. Moreover, the trajectory of the relationship between sleep and pain appears to be mediated by cognitive processes such as increased pain helplessness and, thus, the relationship between sleep and disability appears to be mediated via pain. These data are consistent with the SAPD model, and lend support for the need to include sleep related factors as a critical contributor to our understanding of FM.

Fibromyalgia (FM) is a chronic pain condition that affects close to 2% of the general population (F. Wolfe, Ross, Anderson, Russell, & Hebert, 1995). Although the symptom profile of FM is well understood to include widespread, intense pain combined with extreme fatigue, poor sleep quality, gastrointestinal complaints, cognitive deficits, and depression (e.g., Bennett, Jones, Turk, Russell, & Matallana, 2007; F. Wolfe et al., 2000; F Wolfe & Skevington, 2000), little is known about the etiology of the disorder. Pain is often the most salient symptom of FM and thus FM has traditionally been conceptualized as a pain disorder, with other somatic symptoms (e.g., fatigue, sleep problems) conceptualized as downstream of pain. However, the Sleep and Pain Diathesis (SAPD) model would suggest that for many patients with FM, sleep should be modeled upstream, as an etiological factor and also a “driving force” activating a cognitive feedback loop that serves to maintain a broad range of FM symptoms. The purpose of this paper is to provide a preliminary test of this model using cross-sectional data.

FM has traditionally been treated as a pain disorder. However this approach has failed to yield effective treatment outcomes. One of the largest studies of Rheumatology clinic FM patients found that FM patients were frequent users of health care services (an average of 10 health care visits per year), but did not show improvements in symptoms over time (F. Wolfe et al., 1997a; F. Wolfe et al., 1997b). Moreover, there is ample evidence that analgesics and opiate pain medications that are efficacious for management of other pain disorders do not improve functioning in FM patients. Antidepressant medications have proven to provide improvement in symptoms, but the most robust effect sizes are found using tri-cyclic antidepressants, a class of antidepressants that also improve sleep (Hauser, Bernardy, Uceyler, & Sommer, 2009). Clearly, there is a need for greater understanding of

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the functional interaction between the symptoms that makeup this disorder in order to inform more effective treatment options for FM.

Although sleep disruption is not among the core diagnostic symptoms of FM (F. Wolfe et al., 1990), FM patients almost always report disrupted sleep (e.g.,White, Speechley, Harth, & Ostbye, 1999) and unlike pain symptoms, multiple facets of the disrupted sleep have been routinely, throughly documented. For instance, FM patients have been found to have an EEG abnormality (known as alpha-delta sleep) that correlates with fatigue and achiness (Moldofsky & Scarisbrick, 1976; Moldofsky, Scarisbrick, & England, 1975) (e.g.,A. Drewes et al., 1995; A. M. Drewes et al., 1995; Roizenblatt, Moldofsky, Benedito-Silva, & Tufik, 2001), a high frequency of arousals (Jennum, Drewes, Andreasen, & Nielsen, 1993; Kooh et al., 2003; Rizzi et al., 2003; Sergi et al., 1999), more light sleep (Rizzi et al., 2003; Roizenblatt et al., 2001; Shaver, Lentz, Heitkemper, Buchwald, & Woods, 1997), less deep, slow-wave sleep (Rizzi et al., 2003), and more fragmented sleep (Shaver et al., 1997). In addition, FM patients show a high prevalence of sleep disorders such as Obstructive Sleep Apnea (OSA) (May, West, Baker, & Everett, 1993; Shah, Feingberg, & Krishnan, 2006), a less severe form of respiratory related sleep problem, Upper Airway Resistant Syndrome (UARS) (A. Gold, Dipalo, Gold, & O'Hearn, 2003), Restless Leg Syndrome (RLS) (Yunus & Aldag, 1996), and Periodic Limb Movements (PLM) (Tayag-Kier et al., 2000). As suggested by this review, not all FM patients have one specific type of sleep problem, rather across studies of FM patients we see a heterogeneous constellation of problems that are more generally experienced as “non-refreshing sleep.”

The SAPD model is an attempt to specify a common causal trajectory of FM using a diathesis-stress formulation that begins with sleep disruption. This model proposes that a wide range of biopsychosocial stressors can set the stage for FM by activating diatheses for sleep disruption. Sleep disruption, in those most sensitive to pain, then initiates a cascade of symptoms, including pain and fatigue. Once this process is initiated, the symptoms of FM are perpetuated and aggravated by cognitive variables such as vigilance and perceptions of helplessness that increase perceptions of threat to a broad range of stimuli. These threat related schema would also be expected to manifest in diminished emotion regulation. Final endpoints for patients with FM are known to include disability and elevated levels of depression symptoms, if not clinical depression. Note that the SAPD model does not claim to provide an etiological model for ALL patients with FM. We do however argue that onset of chronic sleep disruption is sufficient in those with low pain tolerance to account for the symptom profile that is described as FM.

The purpose of the present study was to evaluate preliminary evidence for the role of sleep in FM. The SAPD model would be supported if sleep were related to FM outcomes such as disability and depression and if those relationships were mediated by pain and by emotional dysregulation. Although we have imposed a specific causal trajectory on these data, it should be noted that treatment studies and prospective longitudinal data will be needed to disconfirm these causal models.

Method

Participants

Participants were part of two ongoing projects designed to investigate the relationship between sleep and FM symptoms. Sleep Neuroscience Affect and Pain (Project SNAP) participants were community members recruited either in-person at community events, through advertisements in the university newspaper, or through referral from friends. Participants were included if they had a physician diagnosis of FM, and did not have a history of cancer, autoimmune illness, or head injury that resulted in unconsciousness.

Fibromyalgia and Sleep Treatment (Project FAST) were recruited mainly from physician referral, through the study's in-person recruitment at the hospital's rheumatology clinic, and through online advertising. Project FAST is an NIH clinical trial of a sleep treatment for patients with FM and a comorbid sleep disorder. Participants were screened out if they had a comorbid autoimmune illness or significant history of another chronic pain condition. Data used here were pre-treatment and collected from the initial participant questionnaire.

Measures

Participants in both studies completed a battery of questionnaires that included reports of pain, sleep, affect, and disability.

McGill Pain Questionnaire, short form (MPQ-S). The MPQ-S is a shortened version of the original MPQ-long from. Participants rate a list of 15 descriptors of pain on a Likert scale ranging from 0 (no pain) – 3 (severe pain). Scores on items are averaged to produce two subscales: 15 adjectives comprise the sensory dimension (e.g., throbbing, stabbing) and four adjectives comprise the affective dimension (e.g., cruel-punishing) of pain experience (Melzack, 1987). Correlations between the short and long forms ranged between 0.67 and 0.87 and Internal consistency reliability was 0.73 to 0.89 in repeated testing of rheumatoid arthritis and fibromyalgia patients (Burckhardt, 1994).

Pittsburgh Sleep Quality Inventory (PSQI). The PSQI is a self-report questionnaire that retrospectively assesses sleep quality and frequency of sleep disruptions from a one-month period. The measure is comprised of 14 multiple-choice items and 5 open-ended items that are rated from 0 (“No difficulty”) to 3 (“Severe difficulty”). Seven composite scores are derived from these responses to yield a total global score. Composite score areas include: sleep quality, latency, duration and disturbances, habitual sleep efficiency, daytime dysfunction, and use of sleep medication. An optional 5 questions can be asked of the spouse or housemate on sleep habits to contribute to the overall clinical picture but do not additively contribute to the global score (Bussye, Reynolds, Monk, Berman, & Kupfer, 1989). Good to excellent construct and content validity have been reported, as well as high internal consistency (α = .83), and excellent treatment sensitivity (Hunsley & Mash, 2008).

Fibromyalgia Health Assessment Questionnaire (FHAQ). FM related disability was assessed via the FHAQ. Items from the FHAQ were drawn from the Health Assessment Questionnaire (Fries, Spitz, Kraines, & Holman, 1980) and revised to fit the symptom profile of FM. The FHAQ was developed as an internally consistent alternative to other functional disability measures such as the Fibromyalgia Impact Questionnaire (Burckhardt, Clark, & Bennett, 1991) that were found to underestimate disability in individuals with FM (F. Wolfe et al., 2000).

Beck Depression Inventory (BDI). The BDI-II (Beck, Steer & Brown, 1996) assesses 21 symptoms of depression relative to the DSM-IV diagnostic criteria. Symptoms include sadness, pessimism, past failure, loss of pleasure, guilty feelings, punishment feelings, self-dislike, self-criticalness, suicidal thoughts or wishes, crying, loss of interest, indecisiveness, changes in sleeping pattern, irritability, changes in appetite, tiredness or fatigue, loss of interest in sex, agitation, worthlessness, and concentration difficulty. Symptoms are measured on a 4 point scale with range of 0 –3. Scores 13 and above are indicative of high risk for depression. Scores 12 or below are at lower risk for depression. Psychometrics of the BDI-II were generated from a sample of 500 psychiatric outpatients (alpha = .92) and a sample of 120 college students (apha = .93) (Beck, Steer, & Brown, 1996). Dozois, Dobson, and Ahnberg (1998) examined the sensitivity and specificity of the BDI-II in a sample of 1,022 college students and found .81 and .92, respectively.

Rheumatology Attitudes Index (RAI). Pain helplessness was assessed using the helplessness items from the RAI (Callahan, Brooks, & Pincus, 1988; Nicassio, Wallston, Callahan, Herbert, & Pincus, 1985). Items from the RAI were drawn from the Arthritis Helplessness Index and revised to fit the symptom profile of FM. The RAI helplessness subscale has been found to have adequate internal consistency and has been found to have stronger correlations with measures of functional distress than the full RAI (Callahan et al., 1988; Nicassio et al., 1985).

The Emotion Amplification and Reduction Scales (TEARS). The TEARS is an 18-item self-report questionnaire that assesses perceived ability to change the trajectory of an emotional response through amplification or reduction (Hamilton, Karoly, Gallagher, Stevens, Karlson, & McCurdy, 2007). The TEARS has been found to correlate with well-established measures, such as the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) and the Beck Depression Inventory (BDI; Beck & Steer, 1993). TEARS-Amplification is correlated with higher PANAS-Positive Affect ($\beta = .34$, $p < .05$) and TEARS-Reduction is correlated with PANAS-Negative Affect ($\beta = -.36$, $p < .05$) and BDI total score ($\beta = -.15$, $p < .05$). Hamilton et al., demonstrated that both scales have internal consistency ratings that exceed .80.

Data analysis and Results

Participants were 35 adult women, who had been living with FM for an average of 13.02 years (SD = 9.18). Participants exceeded clinical cut-offs for PSQI sleep (*M*=13.91) and BDI depression symptoms (*M*=16.86). Table 1 contains demographic data. Table 2 contains a correlation matrix that includes relevant predictors and possible covariates.

Data Analytic Strategy. Data were analyzed using Ordinary Least Squares Regression (OLS). Zero order correlations were examined to identify potential covariates and to determine whether the necessary preconditions for testing mediation were present in the data (Baron & Kenny, 1986). Multiple Imputation (using 10 model iterations) was used to adjust for missing data. A total of 14 data points were missing, meaning that 4% of the data shown in Table 2 were imputed. Note that correlations between the RAI and other variables are based solely on the FAST data (N=22).

Zero order correlations were examined for possible covariates and to determine whether it would be appropriate to test the meditational models specified by the SAPD model (Table 2). The potential covariates, age and years with FM were correlated only with sensory pain. Although years with pain was correlated with MPQ sensory pain, when included with other predictors years with pain was no longer significant and did not substantially change the results reported in Tables 3 and 4. The conditions to test the hypothesis that pain mediated the relationship between sleep and outcome variables (disability and depression) were met. 1) The predictor, PSQI sleep was correlated with the outcomes of interest, BDI depression symptoms and FHAQ disability. 2) The mediating variables pain (MPQ affective pain and MPQ sensory pain) were correlated with BDI depression symptoms and FHAQ disability. 3) Finally, both MPQ subscales were correlated with PSQI sleep. Thus, tests of mediation included pain and emotion amplification as mediators of the relationship between PSQI sleep disruption and the outcomes depression symptoms and disability. The two TEARS subscales were not related to sleep and, thus, were not tested as mediators. However, the TEARS-reduction (TEARS red)subscale was correlated with depression symptoms and was, thus, included as a predictor.

Predictions made by the SAPD model were tested using OLS regression. Specifically, the SAPD model predicts that sleep disruption will be associated with significantly higher pain

(shown in Table 2) and that pain will be associated with outcomes such as disability and depression. As can be seen in Table 3, PSQI sleep problems were related to higher levels of BDI depression symptoms. Note that values presented in the table are averages of 10 imputations used to replace missing data. Ranges of imputed values are presented in text. Sleep problems alone accounted for an estimated 20% (range: $r^2\Delta$ =.17 to .26) of variance in BDI depression symptoms, F(1,33) 9.00, p<.02. Thus, every 1 unit increase in PSQI sleep correlated with nearly a two point increase in BDI depression symptoms (b's range 1.47–1.93). In the next step, we added MPQ sensory (b's all ns) and MPQ affective dimensions of pain (b's range 8.82–10.73), accounting for an estimated 48% (range: $r^2\Delta$ =.45 to .52) of depression symptoms, F(2,31) 23.62, p<.00. Thus, every one unit increase in affective pain corresponded to a higher BDI depression symptom scores of approximately 10 points. Adding pain ratings mediated the effect of sleep on BDI depression symptoms (*Sobel* =2.669, *p*<.05). Finally, TEARS red (b's range −2.87 to −5.03), accounted for an additional 7% (range: $r^2\Delta$ =.04 to .11) of the variance in BDI depression symptoms, F(1,30) 9.82, p<.05. In contrast to the negative predictors, for every one unit increase in the TEARS red (representing the ability to reduce negative emotions) was correlated with a four unit reduction in the BDI depression symptom score. Collectively, these data accounted for the majority of variance in depression scores r^2 total=.73 (range .68–.80), F(4,30) 24.69, p<.00.

A similar pattern of effects was found for FHAQ disability. There were no missing data in these equations. Thus, data in the table reflect observed, non-bootstrapped estimates of the observed relationships. PSQI sleep accounted for 30% of the variance in FHAQ disability (range: $r^2\Delta$ =.27 to .31), *F*(1,33) 14.22, *p*<.00. Adding the MPQ sensory and MPQ affective pain accounted for an additional 27% (range: $r^2\Delta$ =.26 to .9), *F*(2,31) 9.49 *p*<.00 and partially moderated the effect of PSQI sleep on FHAQ disability. Similar to the BDI, poor sleep was related to a greater degree of disability and at least part of that relationship was accounted for by higher ratings of affective pain (*Sobel*=2.745, *p*<.01).

Finally, we investigated the relationship between PSQI sleep and MPQ pain (Table 4) and whether this relationship was mediated by RAI helplessness. The SAPD model predicts that sleep disruption will be related to increased pain, in part because sleep disruption is associated with dysfunctional cognitions about the ability to cope with pain. RAI pain-helplessness was available only in the FAST data. Thus, this hypothesis was tested in this smaller data set (N=23). Consistent with these predictions PSQI sleep disruption was related to MPQ sensory pain, with sleep disruption accounting for 22% of the variance, F(1,22) 7.004, p=.02. In the second step of this model we added RAI pain helplessness, which accounted for an additional 20% of the variance in MPQ sensory pain F(1,22) 6.835, p=.02. These two measures in total accounted for 46% of the variance in MPQ sensory pain F(2,22) 7.941, p=.003. Adding pain helplessness to the model significantly attenuated the relationship between PSQI sleep disruption and sensory pain (Sobel=2.367, *p*<.01), so that after adding RAI pain-helplessness, PSQI sleep was only a marginally significant predictor of MPQ affective pain F(1,23) 2.84, p=.10. RAI helplessness, however accounted for a significant amount of variance in MPQ affective pain F(1,23) 19.71, p=.00. Collectively these variables accounted for 53% of the variance in MPQ affective pain. These data suggest that the effect of sleep disruption on pain was substantially determined by cognitive factors such as the biased appraisal that one cannot manage pain.

Discussion

FM may best be conceptualized as an endophenotype, or a constellation of symptoms that may result from multiple causes. The SAPD model is an attempt to capture one causal trajectory. Consistent with this model, FM patients who reported more disrupted sleep also reported higher levels of psychological disability (i.e., BDI depression symptoms) and

physical disability. Moreover, the trajectory of the relationship between sleep and pain appears to be mediated by cognitive processes such as increased pain helplessness and, thus, the relationship between sleep and disability appears to be mediated via pain. These data are consistent with the SAPD model, and lend support for the need to include sleep related factors as a critical contributor to our understanding of FM.

From a clinical perspective, it is difficult to treat FM. Patients present with relatively severe levels of disability, but without obvious markers of pathology. Moreover, outcome trajectories are unclear. Recently, longitudinal data from 1555 FM patients showed that over the course of 10 years, most patients experienced fluctuation in symptom severity, 35–40% of patients steadily worsened, and only 25% improve steadily over time (Walitt et al., 2011). Lack of a clear symptom trajectory combined with the absence of objective metabolic and physical markers with FM are likely to enhance perceptions that the disease is mysterious (Reich, Johnson, Zautra, & Davis, 2006), and that effective coping is not possible (Johnson, Zautra, & Davis, 2006). Coping may be particularly problematic for individuals with the worst quality of sleep and for any individual on days following a night of disturbed sleep because of general effects of fatigue, but also because chronic sleep problems are associated with increased arousal and vigilance (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006).

Consistent with this hypothesis are theories of both FM (Turk, 2002) and insomnia (Spielman, Caruso, & Glovinsky, 1987) that emphasize that attention to symptoms maintain and exacerbate perceptions of symptom severity. This is a very reasonable assertion given that normally functioning attention mechanisms have evolved to help shift cognitive resources to stimuli in ourselves and in our environment that might represent threat to our well being. This early threat detection system has been labeled the “motivated attention network” by researchers of neuroaffect and neurocognition (for a description of model of motivated attention see Bradley, Codispoti, Cuthbert, & Lang, 2001). In short it is argued that the motivated attention system, which is controlled by early, posterior attention and limbic system structures, is designed to allow for biased detection of highly arousing and negative internal and external stimuli. This type of bias is argued to be primarily involuntary, normal, and often desirable. In fact this biased attention plays an important evolutionary role because it allows for early detection of threatening stimuli and it allows for the early activation of response plans which might help us to avoid serious injury or trauma (Bradley et al., 2001). Pain and fatigue act as clear, internally-localized signals of threat to our physical selves, which capture attentional resources leaving positive and neutral stimuli relatively ignored.

A perfect example of this role of motivated attention in FM is the phenomena of symptom vigilance which might exacerbate pain helplessness (as measured in the current study). Two fMRI studies have now shown that FM patients do not differ qualitatively from healthy adults in their response to stimuli that both groups categorize as clearly painful. However, unlike the healthy adults, FM patients show a similar pattern of response across neuroanatomical structures associate with motivated attention that is matched to perceived pain intensity rather than actual stimulus intensity (e.g., Cook et al., 2004; Gracely, Petzke, Wolfe, & Clauw, 2002). Thus, the neurophysiological experience of pain in FM patients is clearly more subjective.

Furthermore, unlike healthy controls, FM patients show a heightened response to non-painful levels of blunt thumb nail bed pressure (Gracely et al., 2002) and heat stimuli (Cook et al., 2004). These kinds of stimuli would better be characterized as threatening rather then painful, and they capture greater motivated attention in patients with FM. Vigilance to negative stimuli in general may explain how low-level aches and pains normally associated

with chronic sleep deprivation could activate pain processing centers that are not normally activated by such low intensity stimuli. Furthermore, this experience of more consistent perceived pain, again, likely increases feelings of helplessness. Symptom vigilance may also partially explain the lack of treatment efficacy in FM. A recent meta-analysis of FM-medication trials showed that FM patients have a higher rate of "nocebo-response" (mening placebo patients reporting adverse events similar to side effects of the active medication such as nausea, insomnia, fatigue, headache) than drug trials for migraine medications and Multiple Sclerosis medications (Mitskiostas, Chalarakis, Mantonakis, Delicha, & Sfikakis, 2011)

It should be noted that there are significant limitations to our findings. First and foremost, these are cross sectional data. In order to test the SAPD model, it will be necessary to examine waxing and waning of sleep and pain over time. Although the SAPD model predicts that sleep is the primary driving force in the relationship between sleep and pain, in day to day practice we expect for the relationship to be bidirectional (Affleck, Urrows, Tennen, Higgins, Abeles, et al., 1996). However, the SAPD model goes further than postulating that sleep is driving symptoms of FM. The SAPD model suggests that sleep disruption activates a cognitive schema that activates our motivated attention system and, thus, promotes vigilance to threat related stimuli and amplifies the perceived threat and/or pain value of existing negative stimuli. Again, it will be necessary to examine state related changes in motived attention to determine whether it is sleep or pain that drives increased threat processing.

Is sleep upstream of pain? Is there a bidirectional relationship between sleep and pain? Or does pain drive sleep disruption? There are certainly data to support each of these assertions. However, this is more than an academic-scientific argument. Approaching FM as though it were a pain disorder has provided only partial relief to patients with FM (e.g., Walitt et al., 2011). If the clinical community were to act on the hypothesis that sleep disruption is upstream, then this new understanding opens additional avenues of intervention. As noted in this special issue, there are well validated interventions for sleep disorders like insomnia. Moreover, behavioral interventions for insomnia have been shown to produce benefits for FM patients (Edinger, Wohlgemuth, Krystal, & Rick, 2005) and treatment of respiratory related sleep disorders have produced marked improvements in FM symptoms such as pain and fatigue (Gold, Dipalo, Gold, & Broderick, 2004). Thus, attending to the role of sleep in FM may well offer concrete treatment benefits to a class of patients who have treatment options with limited efficacy.

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Table 1

Demographics

	FAST (N=23)	SNAP (N=12)	Combined (N=35)
Age	*M* = 45.97 (*SD* = 10.49)	*M* = 49.00 (*SD* = 10.60)	*M* = 47.08 (*SD* = 10.47)
Ethnicity	61.9% Caucasian	91% Caucasian,	72.7% Caucasian
	28% African-American	8.3% African-American	21.2% African-American
	8% Hispanic		6.1% Hispanic
	8% missing		5.7% Missing
Years with Pain	*M* = 11.38 (*SD* = 8.99)	*M* = 15.87 (*SD* = 9.19)	*M* = 13.02 (*SD* = 9.18)
Marital	65.22% married	58.3% married	62.86% married
Status	17.39% divorced	33.3% divorced	22.86% divorced
	17.39% single	8.3% single	14.28% single
Annual Income	*M* = $40,000–59,000	*M* = $60,000–79,999	

Table 2

Zero Order Correlations

	PSQI	Age	Years	FHAQ	MPQ Sensory	MPQ Affect	TEARS Amp	TEARS Red	BDI	RAI
PSQI	1									
AGE	−.115	1								
Years pain	−.054	.604**	1							
FHAQ	**.553** **	.081	−.012	1						
MPQ S	**.503** **	−.194	**−.360** *	**.656** **	1					
MPQ A	**.434** **	−.082	−.188	**.677** **	**.710** **	1				
TEARSamp	−.256	.047	.215	−.114	−.210	−.149	1			
TEARSred	−.196	.122	.305	.002	−.086	−.249	**.650** **	1		
BDI	**.466** **	−.206	−.261	**.618** **	**.574** **	**.819** **	**−.304** †	**−.479** **	1	
RAI	**.547** **	.173	.004	**.562** **	**.649** **	**.752** **	−.108	−.125	**.535** *	1

Note:

Note: PSQI=Pittsburgh Sleep Quality Inventory, FHAQ=Fibromyalgia Health Assessment Questionnaire, MPQ S and A=McGill Pain Questionnaire, Sensory and Affective Dimension, TEARSamp and red=The Emotion Amplification and Reduction Scales, amplification and reduction subscales, BDI=Beck Depression Inventory, RAI=Rheumatology Attitudes Index

† **p<.10,**

* ***p*<.05,**

** ***p*<.01**

Table 3

Sleep, Pain, and Emotion Regulation Predict Symptoms of FM

Depression Symptoms		**b**	**SE**	**t**	**R2Δ**
Set 1	(Constant)	−7.061	8.411	−.839	
	PSQI	1.720	.594	2.896**	.17*
Set 2	(Constant)	−2.919	5.804	−.503	
	PSQI	.568	.454	1.253	
	MPQ Sensory	−1.227	2.508	−.489	
	MPQ Affective	10.506	1.879	5.591**	.48**
Set 3	(Constant)	8.039	6.472	1.242	
	PSQI	.393	.404	.973	
	MPQ Sensory	−.080	2.267	−.035	
	MPQ Affective	9.207	1.736	5.304**	
	TEARS red	−3.894	1.509	−2.581**	.07*
Disability Symptoms		***b***	**SE**	**t**	***R2Δ***
Step 1	(Constant)	.519	.280	1.855	
	PSQI	.075	.020	3.812**	.306**
Step 2	(Constant)	.602	.229	2.627	
	PSQI	.035	.019	1.883†	
	MPQ Sensory	.152	.106	1.436	
	MPQ Affect	.185	.081	2.285**	.264**

Note:

Note: PSQI=Pittsburgh Sleep Quality Inventory, MPQ S and A=Multidimensional Pain Questionnaire, Sensory and Affective Dimension, TEARSred=The Emotion Amplification and Reduction Scales

† **p<.10,**

* ***p*<.05,**

** ***p*<.01**

Table 4

Sleep and Helplessness predict Pain

Sensory Pain		**b**	**SE**	**t**	**R2Δ**
Step 1	(Constant)	.269	.568	.474	
	PSQI	.102	.039	2.647**	.259
Step 2	(Constant)	−.286	.543	−.527	
	PSQI	.044	.041	1.086	
	RAI	.362	.139	2.614**	.196
Affective Pain		***b***	**SE**	**t**	***R2Δ***
Step 1	(Constant)	.169	.875	.194	
	PSQI	.100	.059	1.686†	.124
Step 2	(Constant)	−1.018	.683	−1.489	
	PSQI	−.024	.051	−.468	
	RAI	.775	.174	4.440**	.446

Note:

Note: PSQI=Pittsburgh Sleep Quality Inventory, RAI= Rheumatology Attitude Index

† **p<.10,**

* ***p*<.05**,

** ***p*<.01**