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Fibromyalgia: The Role of Sleep in Affect and in Negative Event Reactivity and Recovery

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Objective: Fibromyalgia (FM) syndrome is a chronic pain condition characterized by diffuse muscle pain, increased negative mood, and sleep disturbance. Until recently, sleep disturbance in persons with FM has been modeled as the result of the disease process or its associated pain. The current study examined sleep disturbance (i.e., sleep duration and sleep quality) as a predictor of daily affect, stress reactivity, and stress recovery. ***Design and Measures:*** A hybrid of daily diary and ecological momentary assessment methodology was used to evaluate the psychosocial functioning of 89 women with FM. Participants recorded numeric ratings of pain, fatigue, and positive and negative affect 3 times throughout the day for 30 consecutive days. At the end of each day, participants completed daily diary records of positive and negative life events. In addition, participants reported on their sleep duration and sleep quality each morning. ***Results:*** After accounting for the effects of positive events, negative events, and pain on daily affect scores, it was found that sleep duration and quality were prospectively related to affect and fatigue. Furthermore, the effects of inadequate sleep on negative affect were cumulative. In addition, an inadequate amount of sleep prevented affective recovery from days with a high number of negative events. ***Conclusions:*** These results lend support to the hypothesis that sleep is a component of allostatic load and has an upstream role in daily functioning.

Keywords: sleep, affect, negative events, reactivity, recovery

Fibromyalgia (FM) syndrome is a chronic pain condition that has pervasive effects on daily functioning. Although muscle pain is FM's cardinal symptom, nearly all people suffering from FM complain of disabling fatigue and poor sleep quality (e.g., Moldofsky, Scarisbrick, & England, 1975; Wolfe, Hawley, & Wilson, 1996; Wright, 1985). In their initial characterization of the relationship between FM and sleep disturbance, Moldofsky et al. (1975) noted a correlation between disrupted non-REM sleep and symptoms of FM. Although this marker of disrupted sleep did not prove to be unique to FM patients (Moldofsky, Lue, & Smythe, 1983; Moldofsky et al., 1975), complaints of insomnia and nonrefreshing sleep play a central role in FM patients' symptom reports (Wolfe et al., 1996) and may exacerbate the symptoms and psychosocial problems they report.

The role of sleep in daily functioning may best be thought of as a component of allostatic load (Hamilton, Catley, & Karlson, 2007). Allostatic load has been defined as the accumulated wear and tear on the body that is caused by repeated stress-related demands on metabolism and organ systems (McEwen & Stellar, 1993). Allostatic load varies across individuals and is determined by genetic, developmental, behavioral, and psychosocial factors (McEwen, 1998). Within this framework, the inability to obtain adequate amounts of sleep is not conceptualized as a stressor per se, but as a limited resource that diminishes an individual's ability to withstand repeated adjustive demands. This in turn may alter the ability to manage stress and also influence the onset of disease and the resulting disease course (McEwen, 1998; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). In this way, the allostatic load model is consistent with the theory that the function of sleep is to restore health and promote vigor (Adam & Oswald, 1977). In the case of FM, inadequate sleep may diminish metabolic, cognitive, or affective resources and the ability to respond and recover from psychosocial stress.

Conceptualizing sleep as a component of allostatic load has the potential to inform rather than compete with extant theories of FM pathology. For instance, it has been theorized that pain symptoms in FM are not the result of injury or a disease process, but rather a central process that leads to greater reactivity to pain. People with FM have shown lower central and peripheral pain thresholds, lower peripheral pain tolerance, and slower pain recovery than healthy adults (for a review, see Bennett, 2005). However, it is also the case that people with FM show more intense affective reactions to a wide variety of negative stimuli. When compared with people with other chronic pain conditions such as osteoarthritis, people

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with FM report greater pain and show greater reactivity to negative events (Zautra, Fasman, et al., 2005; Zautra, Hamilton, & Burke, 1999), report greater goal interference from family and friends (Hamilton, Karoly, & Zautra, 2005), and have social networks that they characterize as critical and unreliable (Davis, Zautra, & Reich, 2001). Thus, central sensitization could be thought of as a more pervasive phenomenon of increased allostatic load, driven by a hypersensitivity to a broad range of negative stimuli.

Consistent with the central sensitization framework, the allostatic load model would suggest that poor sleep would promote hypersensitivity to a wide range of affective stimuli, including but not limited to pain. Ecological momentary assessments of people with either FM or rheumatoid arthritis show that sleeping less than 8 hr per night and poor sleep quality predicted stronger affective reactions to negative events and to pain (Hamilton, Catley, Karlson, 2007). Similarly, the FM patients studied here showed a prospective relationship between sleep quality and FM pain and the ability to distract one's self from pain (Affleck et al., 1996). To our knowledge, the role of sleep in stress recovery remains unexamined in patients with FM. A speedy recovery may be more central to the allostatic load concept than is reactivity. A quick response to negative stimuli may enhance the probability of survival, but a prolonged response would increase systemic wear and tear (i.e., allostatic load). Thus, poor sleep appears to contribute to the allostatic load associated with a stressful or painful event by promoting greater reactivity to events and pain and would also be predicted to impede recovery from stressful or painful events.

In addition to reactivity and recovery from negative events, the allostatic load model would also predict that inadequate sleep would compromise affective resources. The allostatic load theory overlaps with research indicating that FM patients show a basic deficit in positive affect (PA) and high levels of fatigue (Davis et al., 2001; Zautra, Fasman, Parish, & Davis, 2007; Zautra, Fasman, et al., 2005; Zautra et al., 1999; Zautra, Smith, Affleck, & Tennen, 2001). A similar pattern of data emerges with regard to sleep. For instance, ecological momentary assessment data gathered from healthy young adults showed that poor sleep quality was associated with more dysphoric mood and greater fatigue (Totterdell, Reynolds, Parkinson, & Briner, 1994). Pain-related sleep disruption also appears to predict fatigue in FM patients (Nicassio, Moxham, Schuman, & Gevirtz, 2002). Furthermore, a 2-year longitudinal study of people with rheumatoid arthritis found that pain and sleep problems acted synergistically to predict an increased number of depressive symptoms (Nicassio & Wallston, 1992).

For patients with FM, deficits in PA are thought to be part of a more profound change in the structure of affective space. As interpersonal stress and pain increase, affective space appears to become constrained and thus the negative correlation between PA and negative affect (NA) increases. This conditional relationship between NA and PA has been termed the dynamic model of affect (Zautra, Johnson, & Davis, 2005; Zautra et al., 2001). It seems clear that stressors such as increased pain and negative events constrain affective space. However, the relationship of sleep to affective complexity remains unclear. The allostatic load framework construes sleep disruption as a resource for managing stress rather than as a stressor. However, it is an empirical question as to whether sleep disruption also reduces affective complexity.

Although the relationship between sleep quality and outcomes such as affect and fatigue is likely to be linear, the relation of sleep duration may be more complex. For instance, experiments involving partial sleep restriction across several consecutive nights (i.e., sleep debt) have shown increasing impairment over time in alertness, mood, and cognitive performance in healthy young adults (Dinges et al., 1997). Furthermore, there is also strong evidence for a curvilinear relationship between sleep duration and outcomes such as mortality (Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002) and diminished psychosocial well-being (Hamilton, Nelson, Stevens, & Kitzman, 2007). Although there may be a nonlinear relationship between sleep and biopsychological functioning in the aggregate, it remains unclear whether the effects of long and short sleep can be detected in terms of variations in day-to-day affective well-being. Furthermore, nonlinear effects of sleep duration have never been evaluated in a population in which sleep is already disrupted and in which individuals are potentially in need of more sleep. Thus, data suggest that the effects of sleep duration may be complex, determined not only by linear and nonlinear duration effects, but also by an additive effect of sleep debt.

Summary

Sleep problems and FM have been linked to reactivity, affective dysregulation, and fatigue. Furthermore, sleep problems are ubiquitous among people with FM. However, few studies have examined the intersection between FM and sleep. The allostatic load model would predict that the inability to obtain an adequate amount of restorative sleep would compromise daily affect and also diminish FM patients' ability to manage daily events. If sleep were a component of allostatic load, we would expect that sleep disruption would increase allostatic load via three relationships: (a) increased NA and fatigue and decreased PA, (b) promoting a stronger relationship between negative events and NA as well as fatigue and weakened relationship between negative events and PA, and (c) impeding recovery from the previous day's negative events. These hypotheses are informed by allostatic load theory. Consistent with experimental and epidemiological data, it would be important to examine both sleep quality and sleep duration and also nonlinear effects of sleep on functioning such as sleep debt and curvilinear relations to biopsychosocial outcomes. In addition to these empirically supported hypotheses, we were also able evaluate whether sleep was related to affective complexity. Thus, our purpose in the current study was to investigate the role of sleep as it relates to affective functioning in a sample of women with FM. Data from a larger study investigating the relationship between psychosocial functioning and FM symptoms were used to test these hypotheses (Affleck et al., 1996).

Method

Sampling Procedures

Study participants were 89 women who met American College of Rheumatology criteria for primary fibromyalgia syndrome (Wolfe et al., 1990): pain in all body quadrants of at least 3 months duration, pain in 11 of 18 tender point sites, and the absence of other musculoskeletal pain disorders (e.g., rheumatoid arthritis). The sample was composed of women recruited from an adult rheumatology practice ($n = 60$) and a pool of community volunteers with widespread pain ($n = 29$). The latter subgroup were all

verified by a rheumatology nurse practitioner to meet criteria for primary fibromyalgia syndrome. Demographic data can be found in Table 1, and a full description of study sampling procedures and patient demographic characteristics can be found in Affleck et al. (2001).

Daily Variables and Measures

Data from both end-of-day summaries of daily events (using paper-and-pencil diaries) and within-day "momentary" interviews of pain, affect, and fatigue (using hand-held computers) were collected from each participant for 30 consecutive days. As a modest incentive to increase adherence to the data collection protocol, participants were paid $0.50 for each completed electronic interview and $2.50 for each completed diary.

Electronic interviews. Participants carried palm-top computers programmed as an electronic interviewer (ELI). This device was a programmable battery-powered Psion Organizer II (Psion, Concord, MA) weighing 8.8 oz. The Psion Organizer has amply demonstrated its feasibility and reliability as a data collection instrument in several other daily self-monitoring studies (e.g., Affleck et al., 2000; Shiffman et al., 1994).

Some procedures for the ELI protocol parallel electronic diary studies of cigarette smokers (e.g.,Shiffman et al., 1994). The data entry procedure for each ELI request proceeded from the termination, with a keystroke, of an audible beep to the choice to answer the interview either then, 5 minutes later, or 15 minutes later. The auditory signal lasted 60 seconds; if the participant did not respond within this time, she was beeped 5 minutes later, and again another 5 minutes later. Failure to answer this sequence of three requests for data produced a missing entry for that time period.

Interview questions were presented one at a time in a fixed order on a liquid crystal display. Participants replied to each question by scrolling across fixed response options and then pressing an "enter" button to save the response and its time stamp on a data storage device, which could not easily be erased. The response option appearing first on the screen with each new question was randomized to minimize response set. Data were uploaded into a laptop computer, where they were entered automatically in a spreadsheet file for data analysis.

Table 1
Descriptive Statistics

Variables	*M* (*SD*) or %
Demographic	
Age	44.4 (8.79)
Ethnicity (Caucasian)	88.8
Duration of fibromyalgia symptoms	106 months (88 months)
Outcome	
Positive affect	6.45 (2.82)
Negative affect	4.53 (4.86)
Fatigue	5.89 (3.34)
Predictor	
Pain	34.67 (19.33)
Negative event ratings (per day)	3.091 (5.01)
Positive event ratings (per day)	15.19 (15.01)
Sleep quality	2.66 (1.53)
Sleep duration	6.38 (1.49)
Symptoms of depression	0.6421 (0.58)
Sleep debt (*n*)	
0–1 nights < 6 hr sleep	16
2–3 nights < 6 hours of sleep	41
4–5 nights < 6 hours of sleep	15
5 or more nights < 6 hours of sleep	16

Note. Values in the table are based on the grand means and standard deviations.

Affect, pain, and fatigue assessments were scheduled three times each day: at times randomly selected by ELI within response windows opened between 9:45 a.m. and 11:15 a.m. (morning interview); between 2:45 p.m. and 4:15 p.m. (afternoon interview); and between 6:45 p.m. and 9:15 p.m. (evening interview). Participants rated their current pain intensity on scales ranging from 0 to 6 (anchored verbally at 0 = *none,* 2 = *mild,* 4 = *moderate,* and 6 = *severe*) in each of 14 areas of the body: neck, shoulders, chest, buttocks, upper and lower back, left and right upper leg, left and right lower leg, left and right lower arm, and left and right upper arm, reflecting the widespread pain criteria required for the diagnosis of primary fibromyalgia syndrome. Current pain for that interview was scored as the sum across body regions. Fatigue was measured by the two adjectives *tired* and *drowsy* (r = .61). NA and PA items were chosen to maximize overlap with previous work linking sleep to affective disturbance (Hamilton, Catley, Karlson, 2007). PA was assessed by two adjectives, *happy* and *cheerful*, and NA was assessed by four adjectives, *sad, blue, nervous*, and *anxious*. Each of these adjectives was rated on a scale ranging from 0 to 6 (anchored verbally at 0 = *none*, 2 = *slightly*, 4 = *moderately*, and 6 = *extremely*) and then summed for a measure of current fatigue, PA, and NA, respectively. These indices of affect were internally consistent (PA, r = .82, and NA, α = .82). Of a total of 8,010 interviews requested of the 89 participants, only 135 (1.7%) were missing.

Sleep. Each morning, participants answered questions about the previous night's sleep. Participants used the computer's internal clock feature to wake them with an audible alarm each morning. Within 30 min of awakening, the ELI signaled them to answer a sleep interview. Sleep quality was assessed using the question "How refreshed or rested do you feel after last night's sleep?" Participants responded on a rating scale ranging from 0 to 6, with higher scores reflecting more restful sleep. Duration was assessed using the question "Estimate how many hours you were actually asleep last night." Sleep duration was recorded in 1-hr increments. The nonlinear effect of sleep duration was calculated by squaring the centered term for sleep duration.

Sleep debt was evaluated by coding the raw sleep data as follows: Sleep durations of 6 hr or greater were coded as 0, and the first instance of sleep duration of less than 6 hr was coded as 1. The following night's sleep duration was coded as a 2 if it fell below 6 hr; the next night was coded as a 3 if it fell below 6 hr, and so on, until the person reported obtaining at least 6 hr of sleep. In this way, we sought to capture periods of cumulative sleep debt embedded in an individual's normal sleep pattern. We used 6 hr as a threshold for adequate sleep to be consistent with data showing that sleep durations of 4–6 hr across seven consecutive nights produces progressively worsening mood (Dinges et al., 1997).

Daily diaries. Before bedtime, participants completed a paper-and-pencil modified version of the Inventory of Small Life Events (Zautra, Guarnaccia, & Dohrenwend, 1986). The checklist consists of 100 events classified as concerning (a) spouse/partner, (b) children, (c) other family members, (d) friends/acquaintances, (e)

work/coworkers, (f) household or finances, and (g) recreation/leisure activities. Each occurring event was rated for its degree of desirability or undesirability on a 7-point scale. Internal consistency and test–retest reliability estimates are not appropriate for daily events checklists. To permit more precise comparisons between ELI reports and daily events, participants also indicated whether the event occurred in the morning (before noon), in the afternoon (between noon and 5 p.m.), or in the evening (after 5 p.m.).

Depressive symptoms. The seven-item depression subscale of the Brief Symptom Inventory (Derogatis & Melisaratos, 1983) is a reliable index of current symptoms of depression (observed $\alpha = .81$). Participants completed this measure shortly before daily diary keeping.

Data Analytic Strategy

Study hypotheses were tested using multilevel modeling, implemented in SAS (Statistical Analysis System) PROC MIXED (Kreft & de Leeuw, 1998). Multilevel modeling facilitates the analysis of data that have a hierarchical structure. In this case, three daily observations were nested within 30 days and 89 participants. There were three principal sources of variance: variability between persons, variability between days (within persons), and variability between assessments (within days and persons). Level 1 variables were those that were measured three times a day for 30 days (i.e., pain and daily events). It should be noted that although events were recorded once per day, participants also indicated whether the event occurred during one of three time windows: morning, afternoon (between noon and 5 p.m.), or evening. Level 2 variables were those that were measured once per day for 30 days (i.e., sleep) and contained both within-person and between-persons variance. Variables that were measured once, such as symptoms of depression, contained only between-persons variance and were modeled as Level 3 variables.

Multilevel analyses were conducted according to the following data-analytic strategy. First, an autoregressive covariance AR (1) matrix was used to control for serial dependency in repeated measures (Affleck, Tennen, Urrows, & Higgins, 1994). The AR (1) matrix, when used appropriately, ensures that each of the dependent variables represents a change in relation to previous scores (Affleck, Zautra, Tennen, & Armeli, 1999). Second, time of observations was included as a Level 1 variable to control for diurnal variation in affect, and day of observation was included as a Level 2 variable to control for time-related artifacts in data collection (Zautra et al., 2001). Third, the slope of PA and NA on negative events was treated as a random effect. Treating slopes as random effects enables us to generalize the findings to the population of within-person relations that these samples are intended to represent (Affleck et al., 2001, 1999).

Variable centering was consistent with the level of analysis. Level 3 variables were centered around the grand mean (Aiken & West, 1991). The Level 1 and Level 2 predictor variables (pain, stressful events, and sleep variables) were centered around each individual's mean. Person-centered Level 1 and Level 2 variables provide a conceptual advantage in that relationships between person-centered predictors and outcomes cleanly reflect the within-person ebb and flow of emotion in response to events (Hamilton, Zautra, & Reich, 2005). Furthermore, inclusion of each person's deviation score along with each person's mean (a Level 3 predictor of the same variable) cleanly differentiates within-person change from individual differences (Kreft & de Leeuw, 1998). In the case of sleep, person-centered sleep duration should be interpreted as change in sleep duration, above or below each person's average sleep duration.

We also tested several Level 3 covariates: age, the duration of FM symptoms, and depressive symptoms. Age and years with FM failed to predict PA, NA, or fatigue and did not alter the relationship of predictors to the outcomes, thus they were omitted from the analysis. Although depressive symptoms did not alter the relationship of any of the sleep predictors to PA, NA, or fatigue, this variable was retained because it predicted both PA and NA and fatigue.

Results

Pain and Events on Daily Mood

Negative events, positive events, and pain were used to predict within-day variance in affective states and fatigue at assessment i on day j for person k. β_{0jk} represents the conditional mean outcome score (PA, NA, or fatigue), β_{1jk} to β_{4jk} represent the slopes of the criterion variables on this set of biopsychosocial predictors, and e_{ijk} represents Level 1 random error.

$$\text{Level 1: (Affect and Fatigue)}_{ijk} = \beta_{0jk} + \beta_{1jk}\,(\text{Negative Events})_{ijk} + \beta_{2jk}\,(\text{Positive Events})_{ijk} + \beta_{3jk}\,(\text{Pain})_{ijk} + \beta_{4jk}(\text{Time of Day})_{ijk} + e_{ijk}$$

Results presented in Table 2 show that higher NA and lower PA accompanied increased daily pain and negative events. It should be noted that the effect of pain on NA and PA is consistent with data reported by Zautra et al. (2001). Positive events were associated with lower NA, higher PA, and lower fatigue. There was evidence of diurnal variation in all study variables. In particular, both NA and PA decreased over the course of the day, whereas fatigue increased. Fatigue appeared to decrease concomitantly with positive events and increase with pain, but did not vary as a function of negative events.

Sleep and affect. Next, we tested the hypothesis that acute changes in sleep duration and quality would predict the intercepts of affect and fatigue. In addition, we created a lagged day-level event variable by summing the number of negative events reported on the previous day $d - 1$.

$$\text{Level 2: } \beta_{0jk} = \beta_{00k} + \beta_{01k}\,(\text{Sleep Duration})_{jk} + \beta_{02k}\,(\text{Sleep Quality})_{jk} + \beta_{03k}\,(\text{Sleep Debt})_{jk} + \beta_{04k}\,(\text{Sleep Duration})^2_{jk} + \beta_{05k}\,(\text{Lagged Negative Events})_{jk} + \beta_{06k}(\text{Study Day})_{jk} + u_{0jk}$$

High-quality sleep the previous night predicted a day with lower NA and fatigue and higher PA. Changes in sleep duration were unrelated to affect. However, an increase in sleep duration was associated with decreased fatigue, and the first-order relationship

Table 2
Affect and Fatigue Regressed on Negative Events, Pain, and Sleep

	Negative Affect		Positive Affect		Fatigue	
	B	*t*(7670)	B	*t*(7670)	B	*t*(7660)
Intercept	2.87	6.54	7.29	27.85		
Level 1 (observation-level) variables						
Time of day	−0.15	−4.00****	−0.14	−5.48****	0.88	30.58****
Δ negative events	0.27	10.34****	−0.11	−7.69****	−0.01	*ns*
Δ positive events	−0.04	−4.76****	0.07	13.64****	−0.03	−6.22****
Δ pain	0.05	9.79****	−0.03	−10.06****	−0.06	19.18****
Level 2 (day-level) variables						
Study day	−0.00	*ns*	0.005	*ns*		
Δ total negative events yesterday	0.03	2.92**	−0.008	−2.45*	0.002	*ns*
Acute change in sleep						
Δ sleep quality	−0.16	−3.58***	0.15	5.69****	−0.19	−7.63****
Δ sleep duration	0.02	*ns*	−0.05	*ns*	−0.16	−5.63****
Δ sleep duration^2	−0.00	*ns*	−0.04	*ns*	0.03	2.91**
Δ sleep debt	0.12	2.69****	−0.02	−1.67†	−0.01	*ns*
Level 3 variables						
Mean negative events	0.96	3.14**	−0.32	−1.77†	0.05	*ns*
Mean positive events	0.03	*ns*	0.13	3.06**	0.09	*ns*
Mean pain	0.03	*ns*	−0.00	*ns*	0.04	3.06**
Symptoms of depression	2.85	5.61****	−1.18	−3.87***	1.41	3.38**
Characteristic sleep						
Mean sleep quality	0.05	*ns*	0.50	2.92**	−0.51	−2.16*
Mean sleep duration	−0.19	−2.05*	0.21	1.24	−0.10	*ns*
Negative event recovery[1]						
Δ Sleep Quality × Δ Total Negative Events Yesterday	−0.01	*ns*	0.00	*ns*	−0.00	*ns*
Δ Sleep Duration × Δ Total Negative Events Yesterday	−0.03	−2.26*	0.02	3.08**	0.01	*ns*

Note. Event recovery operationalized as total stress yesterday × sleep last night → mood today.
*$p < .05$. **$p < .01$. ***$p < .001$. ****$p < .0001$.

between sleep and fatigue was qualified by a nonlinear relationship between sleep duration and fatigue such that more moderate sleep durations were associated with less fatigue than those at the low or the high end of the distribution. Although the previous night's sleep duration did not relate to affect, mounting sleep debt (i.e., successive nights of sleep less than 6 hr) was related to increased negative affect. As shown in Figure 1, there appears to be a "dose–response" relationship between successive nights of inadequate sleep and the within-person change in NA. We chose to plot this relationship to demonstrate that the effects of sleep debt accrue across fairly short time intervals.

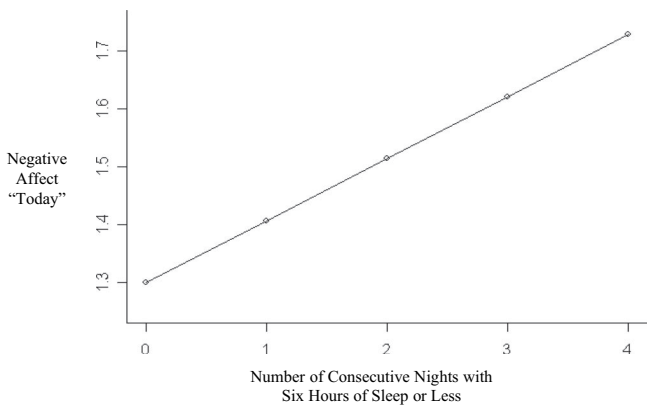

Figure 1. Affective consequences of sleep debt accrued across three successive days.

Dispositional effects of sleep. Next, we tested whether Level 3 variables predicted individual differences in the intercepts of affect and fatigue. In these equations, each person's intercept (β_0) was predicted by an intercept γ_{01}, each person's average sleep duration (Average Sleep-D), average sleep quality (Average Sleep-Q), Average Positive Events (Average Pos), Average Negative Events (Average Neg), Average Pain, symptoms of depression (SxDep), and an error term (u_0).

$$\text{Level 3: } \beta_{00k} = \gamma_{001} + \gamma_{002}\,(\text{Average Sleep D})_k + \gamma_{003}\,(\text{Average Sleep Q})_k + \gamma_{004}\,(\text{Average Pos})_k + \gamma_{005}\,(\text{Average Neg})_k + \gamma_{006}\,(\text{Average Pain})_k + \gamma_{007}\,(\text{SxDep})_k + u_{00k}$$

After controlling for all Level 1 and Level 2 variables, there were Level 3 differences for each person's average number of negative events, positive events, symptoms of depression, and pain (see Table 2). More important, between-person differences in affect were attributable to sleep duration. Women who slept longer on average had lower daily levels of NA, and individual differences in sleep quality were associated with both PA and fatigue. These data

are further evidence that the effects of sleep disruption accrue over time and should be construed as increasing allostatic load.

Negative event reactivity and recovery. Next, we created four interaction terms predicting the slopes of affect and fatigue on negative events. To test the hypothesis that sleep disruption increased reactivity to negative events, we created two interaction terms, Sleep Duration × Negative Events and Sleep Quality × Negative Events. To test the hypothesis that sleep disruption prevented recovery from yesterday's negative events, we created two interaction terms, Sleep Duration × Yesterday's Negative Events and Sleep Quality × Yesterday's Negative Events. There was a small but significant relationship between negative events and sleep duration ($r = -.03$, $p < .01$) and no relationship between sleep quality and negative events, allowing for a straightforward interpretation of interaction effects.

Level 1: (Affect and Fatigue)

$$
\begin{aligned}
&= \beta_{00k} + \beta_{01k}\,(\text{Sleep Duration})_{jk} \\
&+ \beta_{02k}\,(\text{Sleep Quality})_{jk} + u_{0jk} \\
&+ \beta_{10k}\,(\text{Negative Events})_{ijk} \\
&+ \beta_{20k}\,(\text{Lagged Negative Events})_{ijk} \\
&+ \beta_{11k}\,(\text{Sleep Duration} \times \text{Negative Events})_{jk} \\
&+ \beta_{12k}\,(\text{Sleep Quality} \times \text{Negative Events})_{jk} \\
&+ \beta_{21k}\,(\text{Sleep Duration} \times \text{Lagged Negative Events})_{jk} + \beta_{22k}\,(\text{Sleep Quality} \times \text{Lagged Negative Events})_{jk} + (\text{Negative Events})_{ijk}\,u_{1jk} \\
&+ (\text{Lagged Negative Events})_{ijk}\,u_{2jk} + e_{ijk}
\end{aligned}
$$

There were no sleep-related differences in the concurrent relationship between negative events and affect. Thus, sleep did not appear to predict reactivity (these interaction terms were omitted from Table 2, but not from the statistical model). However, as shown in Figures 2 and 3, sleep played a role in affective recovery. Significant interactions between sleep duration and the lagged effect of negative events were plotted using the utility provided by Preacher, Curran, and Bauer (2006). Days with a high number of negative events followed by a shortened, or even an average, night's sleep were followed by lower PA (low sleep: $\beta = -0.0398$ [0.0095], $t(7413) = -4.1678$, $p = .001$; average sleep: $\beta = -0.0067$ [0.0067], $t(7413) = 2.4567$, $p = .014$) and greater NA (low sleep: $\beta = 0.0637$ [0.0159], $t(7413) = 4.0127$, $p = .001$; average sleep: $\beta = 0.0334$ [0.0114], $t(7413) = 2.9152$, $p = .004$) the next day. In contrast, when participants slept longer than average, yesterday's negative events had no relationship to affect the next day (negative affect: $\beta = 0.0031$ [0.0162], $t(7413) = .1894$; positive affect: $\beta = 0.0068$ [0.0094], $t(7413) = .7231$, $p = .469$). Consistent with allostatic load theory, these results indicate that inadequate sleep prevents the restoration of affect following a day with a high number of negative events.

Although our model focused on the outcomes related to sleep problems, it is also likely that daily events, mood, and pain could disrupt sleep. Thus, we reran our analyses with sleep duration and sleep quality as the outcome measures, predicted by the day's total events, average PA and NA, and controlling for pain and depression. Average daily pain predicted lower sleep quality ($B = -0.009$, $p = .002$). However, daily events and mood did not predict sleep that night.

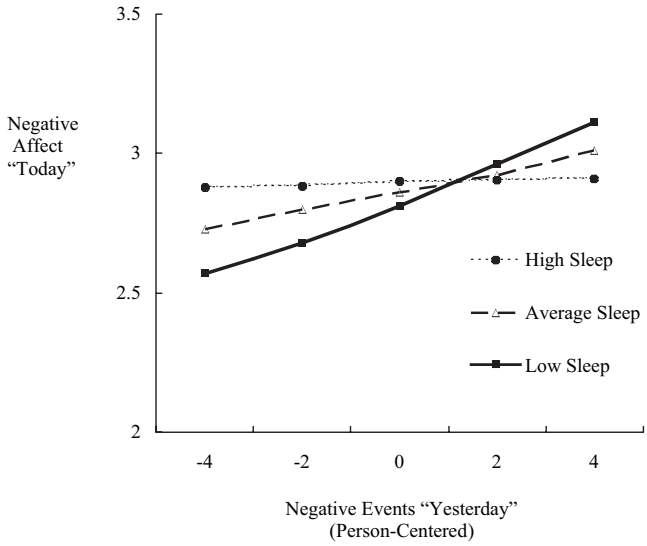

Figure 2. Negative affect and recovery from "yesterday's" negative events.

Affective complexity. To evaluate whether sleep exerts its effects via reduced affective complexity, we tested a parsimonious model in which NA was predicted by person-centered deviations from average PA, sleep duration, sleep quality, and sleep debt (Level 1) as well as each person's average of these variables (Level 3). Consistent with similar diary data (e.g., Zautra, Fasman, et al., 2005), PA was related to NA ($B = -0.63$, $SE = .03$, $p < .00001$). However, there was no evidence that sleep duration, sleep quality, or sleep debt moderated this relationship. Thus, these data do not support a model in which sleep disruption affects the dynamic relationship between PA and NA.

Discussion

The purpose of this study was to examine the upstream role of sleep on affect among women with FM. Our findings suggest that sleep is prospectively related to the next day's affect and fatigue, even after controlling for that day's events and pain. Affective consequences follow a single night of poor-quality sleep. However, accumulated sleep debt appears to be more important than a single night with little sleep. Sleep did not predict individual differences in event reactivity. However, sleep did predict recovery from days with a high number of negative events. This latter effect strongly supports the hypothesis that sleep is a component of allostatic load.

The construct of allostasis suggests that long-term stress-related pathology may be mitigated or exacerbated by psychological, biological, developmental, and behavioral processes (McEwen, 1998). Thus, reactivity may be less important than the speed of recovery. Our results suggest a scenario in which the affective response to negative daily encounters lingers into the next day

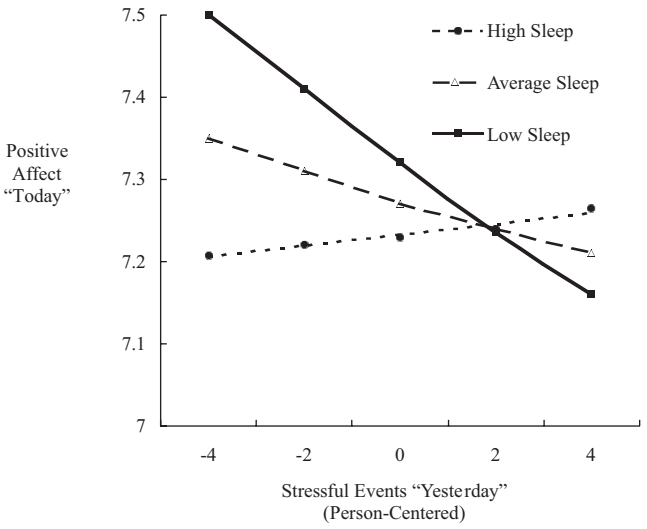

Figure 3. Positive affect and recovery from "yesterday's" negative events. *Note:* This figure *does not* represent the regression line reported in the analysis. Instead, for descriptive purposes we plotted the within-person trajectory of negative affect in 31 women with a total of 36 episodes of sleep debt accrued across at least 3 nights.

unless the slate is wiped clean by a longer than average night's sleep. It is interesting that sleep quality did not enter into this equation, perhaps because poor sleep quality is intractable for women with FM (Moldofsky et al., 1975) and less responsive to increased environmental demands.

The effects of sleep on affect and fatigue suggest that the consequences of inadequate sleep accrue over time. Fluctuation in sleep quality appears to be felt immediately and in all assessed domains of functioning. Dispositional levels of sleep quality were correlated with individual differences in PA and fatigue. In contrast, the effects of sleep duration on affect appear to accrue over time and only in the NA domain. These results provide ecologically valid evidence consistent with experimental findings showing that successive nights of partial sleep deprivation lead to progressively worsened affect (Dinges et al., 1997).

Changes in sleep duration and quality had a direct effect on fatigue the next day. However, more sleep was not necessarily better. Even after controlling for sleep quality, the significant quadratic term indicated that greater fatigue followed nights with an unusually long or short sleep duration and that moderate sleep duration was associated with the least fatigue. This finding may be of critical importance to people with FM. Because sleep quality is so poor in this population, people with FM may attempt to overcompensate for poor sleep by sleeping longer, or at least staying in bed for longer periods of time. The results reported here indicate that this might be a losing strategy unless one needs to compensate for a particularly stressful day.

The results of this study are inconsistent with other research showing sleep-related differences in pain reactivity and negative event reactivity (Hamilton, Catley, Karlson, et al., 2007; Zohar, Tzischinsky, Epstein, & Lavie, 2005). However, these differences may be attributable to methodological differences between studies rather than a true failure to replicate. The strength of these previous studies was that they both limited retrospective recall of stressful encounters to 1 hr or less and measured sleep in 1-min increments. In contrast, the current study relied on end-of-day reports about stressful events that happened at three time points during the day and measured sleep in hour increments. Thus, it is possible that our measurement strategies were not sensitive enough to capture differences in sleep-related event reactivity.

Although we limited the scope of this study to examining the effects of sleep on reactivity and daily affect, our results should be interpreted within the context of previously published data. Women in this sample reported more pain following nights of disrupted sleep and also had trouble focusing on other, more rewarding activities (Affleck et al., 1996). Furthermore, women in this sample reported that following a night of poor sleep, they reduced effort in pursuing social and fitness goals (Affleck et al., 1998). Integrated with current findings, these results suggest that sleep duration and quality play central roles in day-to-day functioning for women with FM, predicting mood, cognition, and also pain. Furthermore, sleep may have an indirect effect on social relationships and participation in health-promoting activities.

One of the strengths of this study is that we were able to temporally order the occurrence of study variables. However, because we have not experimentally manipulated sleep, we cannot positively assert that the changes in sleep caused changes in affect. One other possibility is that sleep problems and disturbed affect are epiphenomena related to a third variable such as rumination. Although we cannot rule this out, we did control for depressive symptoms, which would be highly correlated with rumination (Ingram, Miranda, & Segal, 1998).

Theoretical and Clinical Implications

The results of this body of work suggest a synthesis of current theories about the etiology and maintenance of FM symptoms with Moldofsky et al.'s (1975) work on sleep and FM. Specifically, our results suggest that poor sleep quality may contribute to positive affective deficits observed in women with FM (e.g., Zautra, Fasman, et al., 2005; Zautra et al., 1999). This is important because positive affect appears to moderate pain and stress in women with FM (Zautra, Johnson, & Davis, 2005). However, it was not the case that sleep problems were related to affective complexity. It is possible that because the effects of sleep on PA and NA appear to accrue at different rates across time, sleep does not lead to a coupling of affect in the same way as stressors such as negative events and pain. Finally, the relationship of sleep to negative affect, negative event recovery, and pain (Affleck et al., 1996) also suggests that sleep may lie upstream of the central processing problems theorized to drive the onset and maintenance of FM (Bennett, 2005).

Although most patients with FM complain of sleep problems, to our knowledge only two studies have focused on changing sleep patterns of patients with FM, both with encouraging results. Cognitive–behavioral therapy for insomnia and sleep hygiene therapy were both used to treat insomnia problems in patients with FM (Edinger, Wohlgemuth, Krystal, & Rick, 2005). Both therapies produced changes in sleep, mood, and mental health, and sleep hygiene therapy produced reductions in pain. Another study that changed sleep quality produced more striking results (Gold, Dipalo, Gold, & Broderick, 2004). FM patients with comorbid sleep-disordered breathing were treated with 30 days of continuous

positive airway pressure. These patients reported a 38% improvement in pain and a 46% improvement in fatigue (Gold et al., 2004). The results of this study, along with recent treatment outcome data on sleep interventions, suggest that sleep should be a primary intervention target for people with FM.

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