Fossey, M.E., Bailes, S., Baltzan, M., Schondorf, R., Alapin, I., Fichten, C.S., & Libman, E. (2002, June). Sleep disruption and psychological characteristics in Chronic Fatigue Syndrome. Presentation at the Annual Meeting of the Association of Professional Sleep Societies (APSS), Seattle, Washington. Abstracted in Sleep, 25 (Abstract Supplement 2002), A479-A480, # 681.L.

Research supported by Australian Research Council

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SLEEP VARIABLES, SLEEP QUALITY, AND DAYTIME FUNCTIONING DEFICITS

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Introduction: Sleep latency (LAT), wake time after sleep onset (WASO) and sleep efficiency (SE=Time in Bed/Total Sleep Time) are three sleep variables that are important in evaluating sleep performance. It is not clear, however, which variable among the three is most correlated with subjectively related poor sleep and associated daytime complaints. In this study we obtained information on the self-reported mental and physical health of a randomly selected population in the metropolitan Memphis (TN) area. The subjects completed two weeks of sleep diaries, which affords us a thorough assessment of their sleep patterns. This investigation will focus on the relationship between the three sleep variables and sleep quality as well as daytime sleepiness, and daytime fatigue.

Methods: We used random-digit dialing to recruit at least 50 men and 50 women in each decade from 20 to 80+. Volunteers were paid between $15 and $200 (older adults were paid more) for completing 14 days of sleep diaries and questionnaires regarding daily functioning. Sleep quality was measured from 1 (poor sleep) to 5 (excellent sleep). Measures of daily functioning included the Epworth Sleepiness Scale (ESS), the Stanford Sleepiness Scale (SSS), and the Fatigue Severity Scale (FSS).

Results: We have data on 771 participants, and these individuals had a quality rating of 3.4. Of the three sleep variables SE was the better predictor of sleep quality and daytime functioning deficits. The correlations between SE, WASO and LAT with sleep quality were .49, -.43, and -.32 respectively. As expected, higher SE and lower WASO and LAT were associated with higher quality of sleep. Stepwise multiple regression was conducted using the three sleep measures as independent variables. SE was included in the first model and explained 24% of the variance in sleep quality. WASO added an additional .05% in explained variance, and LAT was excluded from the analysis. Correlations between the sleep variables and daytime sleepiness and fatigue are reported in table 1. SE had slightly higher correlations with FSS scores as compared with LAT and WASO, and WASO had the highest correlation with ESS.

Table 1

Correlations	FSS	ESS	SSS
SE	-.235*	-.082*	.204*
LAT	.225*	.011	.181*
WASO	.212*	.113*	.181*

*= correlation is significant at the .05 level.

Conclusions: As expected, all three variables were significantly associated with sleep quality, and SE had the highest association. LAT and WASO only added an additional 5% in explained variance after accounting for SE. Correlations between daytime sleepiness and fatigue scores were slightly higher for SE as compared with LAT and WASO. Although the correlation differences were somewhat minimal it appears that SE is the better predictor of sleep quality, daytime fatigue and sleepiness. SE is an aggregate measure of sleep performance and does not inform the clinician of specific sleep concerns. However, it might be more informative when evaluating overall perceived sleep quality and certain daytime complaints.

Research supported by National Institute on Aging grants AG12136 and AG14738.

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SLEEP DISRUPTION AND PSYCHOLOGICAL CHARACTERISTICS IN CHRONIC FATIGUE SYNDROME

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Introduction: Chronic fatigue syndrome (CFS) has been a contentious diagnosis for many years. Without definitive laboratory tests, no specific etiology, and no effective treatment, it has long been characterized a functional disorder with a very substantial psychological component[1].An alternative to treating CFS itself is to treat its symptoms. Pilot data from our laboratory as well as indications in the literature[2] suggested that treatable medically based sleep disorders might characterize an important subgroup of people with CFS. If this is confirmed, then a subgroup of CFS patients may benefit from treatment. To shed light on this issue the goal of this preliminary study was to compare CFS patients and normal controls on sleep and daytime functioning as well as psychological adjustment.

Methods: Ten persons with CFS (mean age 47) were recruited primarily through CFS support groups. 10 controls (mean age 41) were recruited from the community. They were compared on nighttime sleep quality and daytime functioning (polysomnography and questionnaires) as well as measures of psychological adjustment.

Results: On polysomnographic measures, individuals with CFS had significantly greater sleep disruption (based on respiratory disturbance and periodic limb movements) than controls On self-report measures, significantly more CFS participants report insomnia than controls. This includes longer sleep onset latency and nocturnal wake times as well as more daytime sleepiness, fatigue and difficulty concentrating On psychological adjustment measures, individuals with CFS do not differ from controls on Anxiety (Speilberger State-Trait Anxiety Inventory: STAI), Depression (Beck Depression Inventory: BDI), or Neuroticism (Eysenck Personality Questionnaire:

EPQ).

Table 1

Sleep Variables

PSG	CFS	Control	t-test
Sleep onset latency (min.)	31.10	15.70	1.42
Nocturnal wake time (min.)	27.38	14.33	1.40
PLM arousals	2.04	0.31	2.73**
RDI	11.00	9.33	2.98**
Questionnaire (Sleep)			X^2
Insomnia: Yes	90%	20%	9.90**
Sleep onset insomnia	80%	30%	5.05*
Middle of the night insomnia	80%	20%	7.20**
Terminal insomnia	50%	0%	6.67**
Nonrestorative sleep	100%	20%	13.33***
Questionnaire (Daytime)			**t-test**
Refreshed in morning (1-10)	1.3	7	5.06**
Sleep quality (1-10)	2.2	8.1	0.14
Sleep satisfaction (1-10)	1.5	8.4	0.00
Fatigue (1-10)	9.2	2.4	3.95**
Sleepiness (1-10)	6.5	2.3	1.74
Concentration difficulties	8.1	2.7	0.89

* p<.05 **p<.01 *** p<.001 *Note.* (1-10): 1 = lo, 10 = hi

Table 2

Mean Scores On Psychological Variables

Measure	CFS	Control	t-test
Anxiety (STAI)	40.89	31.10	0.99
Depression (BDI)	15.00	6.50	0.60
Neuroticism (EPQ)	5.40	2.80	0.36

Note. All tests non significant.

Conclusions: Our findings do not support the wisespread belief that there is a high concordance between CFS and psychological disorder[3]. Instead, they highlight the significant amount of sleep disruption based on physiologically based sleep disorder and the very high rate of insomnia complaints.The findings raise intriguing questions about cause and effect for CFS patients: To what extent does primary sleep disorder have an etiological significance? Is CFS primarily an overlooked sleep disorder [3]? Even if not implicated in etiology, will treatment of sleep apnea and periodic limb movement disorder (RLS/PLMD) have beneficial effects? What role does reported insomnia play in either etiology or maintenance of CFS? What are the implications of insomnia treatment for relief of the symptoms? An investigation is currently ongoing in our laboratory to investigate these questions.

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Research supported by Canadian Institutes of Health Research (CIHR) Medical Research Council of Canada (MRC)

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IS HYPOTHYROIDISM A POTENTIAL RISK FACTOR FOR DEVELOPMENT OF INSOMNIA?

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Introduction: As part of an ongoing series of studies on primary insomnia, we have noted that an unexpected number of subjects have reported being diagnosed with hypothyroidism. While hyperthyroidism has long been thought to be a predisposing and/or maintaining factor for insomnia, there is no literature to suggest that this is also the case for hypothyroidism. In the present report, we provide descriptive data on the prevalence of hypothyroidism in a sample of 90 individuals.

Methods: Subjects for this analysis were evaluated as part of a recruitment procedure for two on going studies of insomnia. Potential participants were directed to call an information number (4-SLEEPY) where they were provided with instructions regarding study eligibility. Those who believed that they were eligible completed a comprehensive screening questionnaire. This instrument was either administered by phone or was completed online at our website (www.sleeplessinrochester.com).Over the last six months, we obtained self-report information from 90 subjects on a wide range of topics including quantitative and qualitative measures of sleep disturbance, medical and psychiatric illness and history, medication and substance use. Approximately 70 of the 90 screenings were performed by phone; the remaining were submitted electronically.

Results: Of the 90 subjects screened to date, 7 (8%) presented with a history of overt hypothyroidism. None reported being hyperthyroid. This 8% rate appears substantially higher than the epidemiologically assessed 0.4% estimated population prevalence (1). Moreover, this rate of occurrence more than doubled the second most common medical condition reported in our sample - fibromyalgia. This disorder was reported by 3 subjects (3.3%).

Conclusions: Give these descriptive data, it appears that hypothyroidism may represent a risk factor for development of insomnia. That is, acute hypothyroidism may precipitate sleep initiation and/or maintenance problems. It is unlikely, however, that hypothyroidism is responsible for chronic insomnia as all of the subjects in the present report were euthyroid. One way that insomnia may occur in association with acute hypothyroidism is as a systemic response. This possibility follows from evidence derived from several sleep deprivation studies which show that thyroid stimulating hormone (TSH) levels rise in response to both partial and full-night sleep loss (2). A second possibility is that over the course of the thyroid disease, the initial decrease in thyroid function results in transient increases T3/T4 which in turn are associated with the first-onset episode of insomnia. In either case, the persistence of insomnia beyond the initial phase of thyroid illness may occur (as with primary insomnia) for reasons more related to the engagement of maladaptive coping strategies and/or thru conditioned arousal. In order to assess if hypothyroidism is a