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Effects of reduced time in bed on daytime sleepiness and recovery sleep in fibromyalgia and rheumatoid arthritis

Timothy Roehrs [a,c,*], Christina Diederichs [a], Mazy Gillis [a], Amanda J Burger [b], Rebecca A Stout [b], Mark A Lumley [b], Thomas Roth [a,c]

[a] Henry Ford Hospital Sleep Disorders and Research Center, United States
[b] Department of Psychology, Wayne State University, United States
[c] Department of Psychiatry and Behavioral Neurosciences, Wayne State University, School of Medicine, Detroit, MI, United States

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ABSTRACT

Objectives: : Fibromyalgia (FM) and rheumatoid arthritis (RA) are associated with sleep disturbance and daytime sleepiness. We sought to determine whether sleep homeostatic mechanisms are blunted in FM by assessing the effects of reduced time in bed (4 h) on next day sleepiness and recovery sleep.
Methods: : Fifty women (18 with FM, 16 with RA, and 16 HC) had a baseline 8 h time-in-bed (TIB) and Multiple Sleep Latency Test (MSLT) the following day, and 3–7 days later bedtime was reduced (4 h) followed by MSLT and an 8 h TIB recovery night.
Results: : Following reduced bedtime the MSLT was reduced relative to baseline in the FM group by an amount (4.3 +/− 4.8 min) similar to that of the RA (3.1 +/− 5.2 min) and HC (4.8 +/− 3.1 min) groups. Relative to the baseline on the recovery night the FM group showed increased sleep efficiency (83.7 +/− 7.8 to 88.1 +/− 9.2%) relative to the RA (83.9 +/− 8.6 to 80.9 +/− 13.3%) and HC (90.1 +/− 5.0 to 87.4 +/− 7.6%) groups due primarily to reduced wake after sleep onset. The groups did not differ in recovery night sleep stages with the exception that the FM group showed REM rebound (21.6 +/− 6.5 to 25.2 +/− 6.0%), which was not found in the RA (20.4 +/− 7.4 to 17.8 +/− 6.5%) or HC (16.6 +/− 6.6 to 17.5 +/− 6.0%) groups.
Conclusions: : Compared to RA and HC, people with FM responded to reduced bedtime with a comparable increase in sleepiness and greater recovery sleep efficiency, suggesting that homeostatic sleep mechanisms are functional in FM. People with FM uniquely showed REM rebound on recovery from reduced bedtime suggesting underlying REM pressure.

Introduction

Fibromyalgia (FM) and rheumatoid arthritis (RA) are chronic pain disorders which are associated with complaints of sleep disturbance õincluding non-refreshing sleep, as well as daytime sleepiness and fatigue. Polysomnographic (PSG) studies have documented disturbed sleep in people with FM and RA [1]. Given the similarity of symptoms and PSG findings, but differences between relative central and peripheral nervous system and immune system involvement in these two disorders, a recent study directly compared both patient groups [2]. Polysomnographic total sleep time was similarly reduced in FM and RA patients relative to age-matched controls, primarily due to increased wake time after sleep onset (WASO). However, despite having comparable nocturnal sleep and reporting comparable levels of daytime sleepiness and fatigue as people with RA, those with FM had an elevated level of daytime arousal as shown on an objective assessment of sleepiness/alertness, the Multiple Sleep Latency Test (MSLT). People with FM showed greater alertness on the MSLT relative to both RA and healthy control groups (average daily sleep latencies of 14.4, 10.3, and 8.6 min respectively) [2].

Elevated MSLT latencies, despite shorter total sleep times, have been reported in people with primary insomnia [3]. Given that among healthy controls, reduced nocturnal sleep duration is associated with greater sleepiness (shorter sleep latencies) on the MSLT [4], it was hypothesized that homeostatic sleep mechanisms were weakened in people with insomnia [5]. To test homeostatic mechanisms in insomnia, the effects of total sleep deprivation on the MSLT and recovery nocturnal sleep were compared to age-matched healthy controls [5]. Although elevated at baseline relative to controls, the average daily sleep latency on the MSLT after total deprivation was reduced in those with insomnia to similar levels as controls. The total sleep time of people with insomnia was less than that of controls at baseline (6.1 vs. 7.6 h), and it increased to that of the controls (7.5 versus 7.8 h) on the recovery night, suggesting normally responsive homeostatic sleep mechanisms. Thus, it was concluded that the elevated MSLT latencies

* Corresponding author at: Sleep Disorders & Research Center, Henry Ford Hospital, 2799 W Grand Blvd, Detroit, MI 48202, United States. Tel.: +1 313 916 5177; fax: +1 313 916 5167.
E-mail address: troehrs1@hfhs.org (T. Roehrs).

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in people with primary insomnia are not a consequence of impaired homeostatic sleep processes, but rather are reflective of a state of "hyperarousal". The "hyperarousal" associated with insomnia is reflected as activation in a number of physiological indices in addition to the MSLT [3].

To assess homeostatic sleep mechanisms in people with FM compared to people with RA and healthy controls, we reduced bedtime to 4 h and assessed next-day sleepiness/alertness on the MSLT and the recovery sleep of the following night. Normal homeostatic sensitivity in response to reduced bedtime would be reflected in similar reductions in MSLT sleep latencies and similar sleep efficiencies (SE) on the recovery night as found in controls. The basal sleep, daytime sleepiness, mood and pain of these two patient groups and healthy controls were reported previously [2].

Methods

Participants

Participants were 50 women who were recruited from the community by local newspaper advertisements and physician referrals: 18 with FM, 16 with RA, and 16 healthy controls (HC) that were age-matched to the FM and RA participants. See Table 1 for participant demographic information. Participants in the two pain groups were required to: a) have FM or RA as their primary pain condition; b) report a customary bedtime of midnight or earlier; and c) report current pain severity of at least 4 on a scale of 1–10. We excluded anyone who: a) met criteria for other pain conditions co-morbid with FM or RA; b) had a primary sleep disorder, based on a screening 8 h PSG (i.e., apnea/hypopnea or periodic leg movement indices >10); c) was taking medications that interfere with sleep/wake function (i.e. opiates, hypnotics, antidepressants, and anti-seizure medications); or d) had current psychiatric disorders as screened by the DSM-IV Axis I Disorders (SCID-I). Healthy control participants had a customary bedtime of midnight or earlier and had no history of any pain condition or sleep-related complaint or medical and psychiatric disorders as screened in the patient groups. All participants signed an IRB-approved informed consent and were paid for their participation.

Procedures

Participants made a screening visit to the Henry Ford Hospital Sleep Disorders and Research Center in Detroit and provided informed consent and completed screening procedures as previously described [2]. Briefly, participants met with a physician for a history and physical to confirm their normal health or pain diagnosis and provided blood and urine samples to screen for the presence of CNS-acting drugs and of diseases that may mimic FM or RA. They underwent a structured interview [DSM-IV Axis 1 Disorders (SCID-1)] to rule out current clinical depression. Participants completed two consecutive 8-h nights of PSG (night 1 was a laboratory adaptation and sleep disorders screening, and night 2 was baseline) and Multiple Sleep Latency Test (MSLT) that followed night 2 (the baseline MSLT). After the first two nights and day designed to describe baseline pain, sleep, fatigue and sleepiness in FM, RA, and controls [2], participants returned within 3–7 days for the reduced bedtime protocol.

On the first night bedtime was reduced to 4 h by delaying lights out until 3 am and fixing time of arising to 7 am. The reduced bedtime night was followed by a day of Multiple Sleep Latency Test (MSLT) with testing at 0915, 1115, 1315, 1515, and 1715 h as on the baseline day. For the following recovery night, bedtime was set to 8 h (11 pm–7 am). For all sleep nights, participants arrived 2 h prior to their bedtime to complete check-in procedures and undergo electrode placement for the PSG [6]. On the 4 h reduced bedtime night participants were monitored after their arrival to ensure continued wakefulness until their 3 am bedtime. Overnight PSG recordings on each night consisted of continuous monitoring of two channels of EEG (C3-A2 and O2-A1), left and right EOG, and submental EMG according to the standards of Rechtschaffen and Kales [6].

Each test of the MSLT on the day following the baseline and reduced bedtime nights was conducted according to the standard protocol [7]. Participants were placed in bed in quiet, darkened rooms and instructed to close their eyes, relax, and fall asleep. Each test was concluded after 20 min of continuous wake or three consecutive 30 s epochs of any sleep stage. Latency to sleep onset was scored as min to the first epoch of sleep or 20 min if sleep did not occur. We averaged the latencies for the 5 tests to generate a daily mean latency value.

Statistical analysis

To assess homeostatic sleep mechanisms, General Linear Model (GLM) analyses were computed for the MSLT and PSG variables. The between-subjects factor was grouped (FM, RA, and HC) and the within-subject repeated variable was the baseline versus bedtime reduction MSLT days and the baseline versus recovery PSG variables. PSG variables for the reduction night were compared between groups using a between-groups ANOVA. Post-hoc tests were conducted where appropriate comparing the groups. An alpha level of $p < .05$ was used for determining significance. Data in the tables and text are presented as means (+/− sd), while the graphs present means (+/− SEM).

Results

Fig. 1 presents the average daily sleep latency on the MSLT for the baseline and the bedtime reduction days. The Group by Condition GLM analysis revealed a significant Group effect ($F = 12.32$, $p = .001$) and Condition effect ($F = 4.64$, $p = .037$), but no

Table 1
Participant demographics by group.

Demographics	FM (n = 18)	RA (n = 16)	HC (n = 16)
Age years mean (+sd)	48 (9)	52 (7)	47 (6)
Ethnicity n (%)			
Caucasian	15 (83)	8 (50)	6 (38)
African-American	2 (12)	4 (25)	9 (56)
Hispanic	0 (0)	2 (12.5)	0 (0)
Undisclosed	1 (5)	2 (12.5)	1 (6)

FM = fibromyalgia, RA = rheumatoid arthritis and HC = healthy controls.

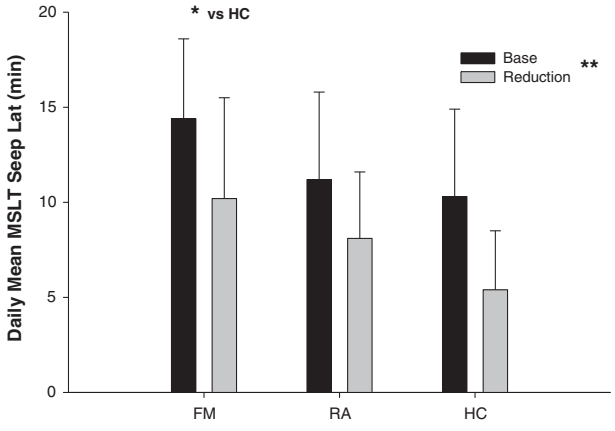

Fig. 1. Multiple Sleep Latency Test scores in the groups (mean of five tests) at baseline and after bedtime reduction. FM = fibromyalgia, RA = rheumatoid arthritis and HC = healthy controls. Data = mean (+/− SEM) Group effect ($F = 12.32$, $p = .001$) and Condition effect ($F = 4.64$, **$p = .037$) and no interaction. FM MSLT was higher than HC (*$p = .05$).

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