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Original Article

Sleep disturbances in fibromyalgia syndrome: the role of clinical and polysomnographic variables explaining poor sleep quality in patients

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ABSTRACT

Background: Sleep complaints are one of the most frequent and relevant symptoms that characterize fibromyalgia syndrome (FMS). However, objective sleep disturbances have not been consistently described across FMS studies. It is therefore commonly accepted that FMS patients experience sleep misperception, even though no studies have investigated the contribution of polysomnographic parameters to determine subjective sleep quality in FMS. We aimed to compare sleep variables (polysomnographic parameters and subjective sleep quality) between FMS patients and healthy controls. Furthermore, we also aimed to define the predictors of subjective sleep quality in FMS.
Methods: We performed in-home polysomnography to 99 women (53 FMS patients and 36 healthy controls). We also collected subjective ratings of sleep quality, daytime sleepiness, pain, depression, and anxiety.
Results: Multivariate analysis showed that groups differed in polysomnographic parameters ($p = 0.015$) – after accounting for age, body mass index, and antidepressant consumption. Specifically, FMS patients exhibited lower sleep efficiency, greater percentage of stage N1 and wakefulness, and more frequent awakenings than controls (p-values < 0.05). Patients also complained about poorer subjective sleep quality ($p < 0.001$). Percentage of time awake (as obtained by polysomnography), depression levels, and antidepressant consumption predicted self-reported sleep quality in FMS patients (adjusted $R^2 = 0.33$, $p < 0.001$).
Conclusions: One night of in-home polysomnography supports the hypothesis that women with FMS show polysomnographic alterations compared to age-matched controls. In addition, the time spent awake is the best predictor of subjective sleep quality, although greater levels of depression and antidepressant consumption might result in exaggerated complaints. These findings contribute to our understanding of FMS symptoms and its management.

1. Introduction

Fibromyalgia syndrome (FMS) is a nonarticular rheumatic condition characterized by chronic widespread pain and several nonspecific symptoms, including other body and joint pains, emotional disorders, cognitive dysfunction, neuropathies, fatigue, daytime sleepiness, and sleep disturbances [1,2]. Depending on the diagnostic criteria and population studied, the prevalence of FMS ranges from 1.1% to 6.4%, being more common among perimenopausal and postmenopausal women [3–5]. FMS has a severe negative impact on the quality of life [6], and, therefore, FMS patients very frequently use health-care systems [7]. However, pharmacological therapies have limited efficacy, and the access to nonpharmacological therapies (which are often more effective than the former) is not broadly available [8]. The fact that there is no universally effective treatment yet might be largely because the etiology and pathophysiology of FMS are not fully understood, and the optimal treatment of FMS is not clear [9]. Therefore, an in-depth analysis of the main symptoms and their interactions is crucial [10].

Pain is the cardinal feature of FMS and the main reason that leads patients to look for medical care [11]. Sleep disturbances are especially relevant as well, as they are reported to be highly debilitating [12,13]. FMS patients consistently complain of poor sleep quality, exhibiting insomnia symptoms, and feelings of unrefreshing sleep, daytime tiredness, and sleepiness [14,15]. However, some authors (eg, Ref. [16]) still maintain that those sleep complaints would reflect some kind of sleep misperception in FMS. Although extensive research has focused on sleep in FMS patients since the mid-1970s,

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Table 1
Overview regarding the characterization of polysomnographic alterations in patients with fibromyalgia syndrome relative to healthy controls.

		Country	*N* FMS/HC	Sample gender	Setting	*N* nights recorded
Decreased total sleep time	Besteiro et al., 2011 [23]	Spain	32/20	♀	Sleep lab	1
	Roehrs et al., 2013 [24]	USA	18/16	♀	Sleep lab	2
Lower sleep efficiency	Drewes et al., 1994 [25]	Denmark	14/12	♀	In home	2
	Landis et al., 2001 [26]	USA	25/21	♀	Sleep lab	3
	Landis et al., 2004 [27]	USA	33/37	♀	Sleep lab	3
	Riva et al., 2010 [28]	Sweden	29/29	♀	Sleep lab	1
	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
	Sergi et al., 1999 [30]	Italy	17/17	♀/♂	Sleep lab	2
Prolonged sleep-onset latency	Landis et alet al., 2004 [27]	USA	33/37	♀	Sleep lab	3
Increase of light sleep to the detriment of deep sleep	Drewes et al., 1994 [25]	Denmark	14/12	♀	In home	2
	Côté and Moldofsky, 1997 [31]	Canada	10/9	♀	Sleep lab	2
	Sergi et al., 1999 [30]	Italy	17/17	♀/♂	Sleep lab	2
	Roizenblatt et al., 2001 [32]	Brazil	40/43	♀	Sleep lab	2
	Landis et al., 2004 [27]	USA	33/37	♀	Sleep lab	3
	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
	Besteiro et al., 2011 [23]	Spain	32/20	♀	Sleep lab	1
	Shaver et al., 1997 [33]	USA	11/11	♀	Sleep lab	2
Longer wake after sleep-onset time	Besteiro et al., 2011 [23]	Spain	32/20	♀	Sleep lab	1
	Drewes et al., 1994 [25]	Denmark	14/12	♀	In home	2
	Roehrs et al., 2013 [24]	USA	18/16	♀	Sleep lab	2
Greater *N* of awakenings	Besteiro et al., 2011 [23]	Spain	32/20	♀	Sleep lab	1
Greater *N* of stage changes	Landis et al., 2001 [26]	USA	25/21	♀	Sleep lab	3
	Burns et al., 2008 [34]	USA	15/15	♀	Sleep lab	3
Greater *N* of arousals	Sergi et al., 1999 [30]	Italy	17/17	♀/♂	Sleep lab	2
	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
Greater *N* of respiratory events[a]	Sergi et al., 1999 [30]	Italy	17/17	♀/♂	Sleep lab	2
Greater *N* of cardiac events[b]	Chervin et al., 2009 [35]	USA	15/15	♀	Sleep lab	3
Greater *N* of oxygen desaturations	Lario et al., 1996 [36]	Spain	28/15	♀	Sleep lab	2
	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
Greater *N* of limb movements	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
	Besteiro et al., 2011 [23]	Spain	32/20	♀	Sleep lab	1
Greater presence of α-EEG sleep	Branco et al.,1994 [37][c]	Portugal	10/14	♀/♂	Sleep lab	2
	Drewes et al., 1994 [25][d]	Denmark	14/12	♀	In home	2
	Roizenblatt et al., 2001 [32][e]	Brazil	40/43	♀	Sleep lab	2
	Rosenfeld et al., 2014 [38][f]	USA	133/252	♀/♂	Sleep lab	1
Presence of CAP	Rizzi et al., 2004 [29]	Italy	45/38	♀/♂	Sleep lab	2
Decreased sleep spindles	Landis et al., 2004 [39]	USA	37/30	♀	Sleep lab	3

Abbreviations: ♀, Female sample; ♂, Male sample; CAP, Cyclic alternating pattern; EEG, Electroencephalography; FMS, Fibromyalgia syndrome; HC, Healthy controls; N, Number.
[a] Apnea–hypopnea index, flow-limited breaths, periodic breathing.
[b] Heart rate variability (HRT), ratio-based HRT, complexity of HRT.
[c] Alpha–delta ratio (greater in patients).
[d] Mean power in alpha band during non-REM sleep (greater in patients).
[e] Percentage of participants with the low presence of alpha activity (greater percentage in controls).
[f] Delta event–alpha event ratio (lower in patients).

little is known about the contribution of polysomnographic (PSG) parameters determining subjective sleep quality in FMS. In their pioneering PSG studies, Moldofsky and colleagues [17,18] were the first to describe specific α-electroencephalographic (EEG) sleep patterns in *fibrositis*[1] patients. Subsequent studies suggested that α-EEG sleep would explain unrefreshing sleep in FMS (for a review, see Refs. [20,21]). Unfortunately, much of the following research – investigating PSG parameters in FMS patients compared to controls – has been inconclusive [22], obtaining only some consistency regarding (a) an increase of light sleep to the detriment of deep sleep, (b) a lower sleep efficiency (SE), and (c) signs of sleep fragmentation (eg, number of awakenings) (see Table 1). Contrarily, several studies have reported minimal differences [24,34,35,38] or no abnormalities at all [40] between FMS patients and controls.

One plausible explanation for the heterogeneity and incongruence among PSG results could arise from several confounding variables that modulate sleep outcomes (eg, sample selection criteria, sample size, methodological aspects of the sleep assessment – including different definitions for PSG parameters – and/or other clinical variables). Another explanation might come from the intrinsic heterogeneity that characterizes the FMS population. As the clinical profiles of these patients are quite variable, FMS would not constitute a single clinical entity, sleep disturbances being a key symptom to differentiate among patients' typologies [41,42]. Furthermore, sleep is a factor that has bidirectional relationships with pain [43], and it magnifies adverse pain-related outcomes in FMS [44,45]. Thus, the existence of sleep disturbances in FMS is a complex problem that remains contested in the literature, even when the recognition and treatment of sleep complaints in FMS is a priority for physicians.

Here, to disentangle the discussion about whether specific PSG parameters characterize sleep disturbances in FMS, we aimed to compare sleep variables (PSG parameters and subjective sleep quality) between a large sample of women with FMS and healthy controls, matched for sociodemographic variables and assessed in real environments. In our design, we consider participants' sociodemographic and clinical features as they might influence PSG parameters [46,47]. We expected that PSG parameters would distinguish FMS patients from controls. Using the categories of PSG parameters proposed by Shaver and colleagues [33], we specifically

[1] For centuries, muscle pains have been known as "rheumatism" and "muscular rheumatism." The term "fibrositis" was first used in 1904, and it was changed to "fibromyalgia" only after 1976 [19].