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Effects of cognitive-behavioral therapy for insomnia on polysomnographic parameters in fibromyalgia patients[1]

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ABSTRACT. This study aimed to evaluate the efficacy of cognitive-behavioral therapy for insomnia (CBT-I) on polysomnographic parameters in patients with fibromyalgia (FM). Twenty-six women with FM participated in the study and were randomly assigned to a CBT-I (n = 13) group or sleep hygiene (SH) condition (n = 13). The evaluation consisted in two interview sessions and domiciliary polysomnography study before and after treatment. The results show that time-in-bed and wake percentage diminish after CBT-I. Improvements were also observed in sleep efficiency, which was close to normal levels. The percentage of NREM stage 1 sleep decreased and NREM stages 3 sleep and 4 increased. Similarly, light sleep (stages 1 and 2) diminished and deep sleep increased (stages 3 and 4) after CBT-I. No improvements were observed in any of these parameters in the individuals undergoing SH therapy. This randomized controlled trial provides new evidence that the use of CBT-I in FM patients can significantly improve objective sleep parameters.

KEYWORDS. Fibromyalgia. Insomnia. Cognitive-behavioral therapy. Domiciliary polisomnography. Randomized controlled trial.

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RESUMEN. El objetivo de este estudio fue evaluar la eficacia de la terapia cognitivo-conductual para el insomnio (TCC-I) en los parámetros polisomnográficos de pacientes con fibromialgia (FM). Veintiséis mujeres con FM participaron en el estudio y fueron asignadas al azar a un grupo de TCC-I (n = 13) o a una condición de higiene del sueño (HS) (n = 13). La evaluación consistió en dos sesiones de entrevista y un estudio polisomnográfico domiciliario antes y después del tratamiento. Los resultados mostraron que el tiempo en cama y el porcentaje de vigilia disminuyeron después de la TCC-I. Se observaron mejorías en la eficiencia del sueño, acercándose a niveles normales. El porcentaje de etapa 1 del sueño NREM disminuyó y se observó un aumento en las etapas 3 y 4 del sueño NREM. Del mismo modo, el sueño ligero (etapas 1 y 2) disminuyó y el sueño profundo aumentó (etapas 3 y 4) después de la TCC-I. Los sujetos que participaron en la terapia de HS no mostraron ninguna mejoría en ninguno de estos parámetros. Este ensayo controlado aleatorizado proporciona nueva evidencia de que el uso de la TCC-I en pacientes con FM puede mejorar significativamente los parámetros objetivos del sueño.

PALABRAS CLAVE. Fibromialgia. Insomnio. Terapia cognitivo-conductual. Polisomnografía domiciliaria. Ensayo controlado aleatorizado.

Fibromyalgia (FM) is a disorder characterized by widespread musculoskeletal chronic pain and multiple tender points (11 of 18 tender points) (Wolfe *et al.,* 1990). The symptoms of FM are very heterogeneous. Besides pain, up to 96-99% of patients with FM describe fatigue and sleep dysfunction (Lineberger, Means, and Edinger, 2007). They also complain of anxiety, depression, cognitive dysfunction, stiffness, cold sensitivity, irritable bowel syndrome and headaches (Gormsen, Rosenberg, Bach, and Jensen, 2010; Miró *et al.,* in press; Miró, Martínez, Sánchez, Prados, and Medina, 2011; Pérez-Pareja, Sesé, González-Ordi, and Palmer, 2010), with significant negative repercussions on the patient's quality of life (Lledó-Boyer *et al.,* 2010; Sánchez, Martínez, Miró, and Medina, 2011).

The etiology of FM is unknown. Thus, it is currently difficult to have an in-depth understanding of the role of, and relationships between, pain and other symptoms that may accompany this syndrome, and effective treatment is therefore lacking (Häuser, Thieme, and Turk, 2010). A number of hypotheses have been proposed regarding the pathophysiology of FM, including central nervous system dysfunction affecting pain sensitivity, viral infections, immunological causes, neuroendocrine dysfunction, neuromuscular, metabolic or immune system issues, and it has even been suggested that FM is associated with a history of trauma or other psychological disorders (Bradley, McKendree-Smith, Alarcón, and Cianfrini, 2002; Broderick, Junghaenel, and Schwartz, 2005; Gur and Oktayoglu, 2008).

Some authors suggest that sleep disturbances may have an important role in the maintenance of pain and other symptoms of FM (for a review see Moldofsky, 2001, 2002, 2008, 2010). Moreover, Nicassio, Moxham, Schuman, and Gevirtz (2002) analyzed the influence of pain, depression and sleep disorders on fatigue in FM using questionnaires and self-records, and observed multiple relationships between pain, sleep and fatigue,

beyond the prevailing notion that pain is responsible for the other symptoms. Recently, Hamilton *et al.* (2008) reported that sleep duration and sleep quality are prospectively related to affect and fatigue. In addition, inadequate sleep has a cumulative effect on negative mood. In this line, recent clinical and experimental research shows that sleep disturbances have a reciprocal influence on musculoskeletal pain and fatigue (Moldofsky, 2008, 2010). In fact, the American College of Rheumatology has developed diagnostic criteria for FM in which unrefreshing sleep is included as one of the most important diagnostic variables (Wolfe *et al.,* 2010).

Although the presence of abnormal nocturnal sleep in FM has been reported and recognized, its significance with respect to the pathophysiology of the syndrome is debated. Also, sleep recordings are rarely used for evaluation in these patients and sleep disturbance is often considered a consequence of pain (Spitzer and Broadman, 2010).

Most research studies that have used subjective measures of sleep (mainly self-reports) mention the poor subjective quality of sleep in FM patients. Thus, for example, 99% of FM patients in the study by Theadom, Cropley, and Humphrey (2007) reported poor sleep quality measured using the Pittsburgh Sleep Quality Index (PSQI). Also, in this study sleep quality was significantly predictive of pain, fatigue, and social functioning in patients with FM. Osorio, Gallinaro, Lorenzi-Filho, and Lage (2006), also using the PSQI, observed that patients with FM achieved higher scores than healthy controls in all the PSQI components except *Use of sleep medications*.

Different alterations have been identified by polysomnography (PSG) in patients with FM, although not all research results are consistent. Specifically, it has been described that these patients display shorter total sleep time, greater sleep latency, more awakenings, less sleep efficiency, higher percentages of non-rapid eye movement (NREM) stage 1 sleep, greater fragmentation of sleep, lower percentages of REM (rapid eye movement) sleep and shorter NREM sleep stages 3 and 4 compared with healthy controls (Besteiro *et al.,* 2011; Dauvilliers and Carlander, 2007; Moldofsky, 2001, 2008; Roizenblatt, Moldofsky, Benedito-Silva, and Tufik, 2001). A recent study used actigraphy, showing that female FM patients with objective sleep deficits (less than 6 hours of sleep) presented significantly lower sleep efficiency, significantly longer sleep onset latency and significantly shorter nighttime sleep times than women without sleep deficits (Stuifbergen, Phillips, Carter, Morrison, and Todd, 2010).

In terms of the microstructure of sleep in FM, observed alterations include alpha-delta intrusions, as well as regular K-alpha intrusions, decreased sleep spindles, larger number of oxygen desaturations per hour of sleep and twice as many arousals per hour of sleep than controls, with an alternating cyclic pattern associated with severity of pain and low sleep efficiency (Lineberger *et al.*, 2007; Rizzi *et al.*, 2004). Moldofsky in 1975 was the first to suggest, through polysomnography, that the presence of alpha intrusions in deep delta sleep could be related with the set of symptoms known as FM. Other studies have also reported these alpha intrusions in slow-wave or NREM sleep, as well as different changes indicative of sleep fragmentation (Lineberger *et al.*, 2007; Moldofsky, 2001; Roizenblatt *et al.*, 2001). The alpha sleep pattern in FM has been associated with longer duration of pain symptoms, a perception of generally unrestful sleep and the

presence of pain when getting up in the morning (Lineberger *et al.*, 2007; Moldofsky, 2008).

As regards treatment, a recent review of the latest Clinical Practice Guidelines on the treatment of FM of the American Pain Society (APS) (Burckhardt *et al.*, 2005), the European League Against Rheumatism (EULAR) (Carville *et al.*, 2008) and the Association of the Scientific Medical Societies in Germany (AWMF) (2008) (Häuser *et al.,* 2010), recommend that FM be treated using a multidisciplinary approach combining aerobic exercise, cognitive-behavioral therapy (CBT), amitriptyline and multicomponent treatments (Häuser *et al.*, 2010). However, although it is accepted that sleep alteration is one key symptom of FM, the treatment of sleep alterations is not covered in current clinical guidelines on this syndrome. In most cases, such guidelines only include sleep hygiene (SH) instructions and pharmacological therapies to treat such alterations, but other much more effective cognitive-behavioral therapy for insomnia techniques (CBT-I) are not applied (Edinger, Wohlgemuth, Krystal, and Rice, 2005; Miró, Sánchez, and Buela-Casal, 2003; Pigeon, 2010).

In existing literature, two pilot studies have shown that CBT-I may improve sleep (Edinger *et al.*, 2005; Miró, Lupiañez *et al.,* 2011). In this first study, Edinger *et al.* (2005) compared sleep and other symptom improvements in FM patients who received CBT-I, sleep hygiene (SH) or only usual care: 57% of the CBT-I group reported significantly improved sleep quality and mood, compared with 20% of the SH group and 3.5% of the medication therapy group. However, Edinger *et al.* (2005) used different subjective scales and actigraphy, which are less reliable than polysomnography (PSG). Recently, Miró, Lupiañez *et al.* (2011) compared CBT-I with SH and observed greater improvements not only in sleep quality but also in attention function and daily functioning in the CBT-I group. However, these studies did not use PSG records to evaluate changes in sleep quality.

In summary, sleep alterations are one of the most prevalent symptoms in FM. Several studies have suggested that an improvement in sleep quality could be associated with a positive change in pain, fatigue and daily functioning. Therefore, determining whether CBT-I can improve not only subjective sleep quality but also objective sleep parameters is crucial to establish the clinical utility of this intervention. Thus, the aim of this study was to evaluate the efficacy of CBT-I on polysomnographic parameters in patients with FM compared to a control group that received SH.

Method

Participants

Twenty-six women with FM (M = 46.79 years of age, SD = 5.15) participated in the study and were assigned to a CBT-I group (n = 13; M = 44.83, SD = 5.30) or a sleep hygiene (SH) condition (n = 13; M = 48.75, SD = 4.37). Simple randomization (1:1) was implemented by a computerized number generator designed by an investigator with no clinical involvement in the trial. The clinical sample was selected from the Rheumatology Service and Pain Unit of the *Hospital Universitario Virgen de las Nieves* in Granada

(Spain). The mean duration of the illness was 5.02 years (*SD* = 4.28), although the mean onset of symptoms was greater (*M* = 12.96 years; *SD* = 8.33). The women who were fulfilling the inclusion criteria to participate in the study (see Table 1) were referred from the hospital at the Clinical Psychology Unit of the Faculty of Psychology (University of Granada), where three psychologist therapists conducted both assessment and treatment (CBT-I and SH) to patients with FM. All participants were informed about the characteristics of the study and an informed consent was obtained. The study received ethical approval from the University of Granada Ethics Committee.

TABLE 1. Inclusion and exclusion criteria established for participation in the study.

	Inclusion criteria
1.	Age between 25 and 60 years old.
2.	Met the diagnostic criteria for FM as defined by the American College of Rheumatology (ACR) (Wolfe *et al.,* 1990).
3.	Have chronic insomnia according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, American Psychiatric Association, 2000).
	Exclusion criteria
1.	Currently pregnant.
2.	Medical history of significant head injury or neurological disorder.
3.	Concomitant major medical conditions (e.g., inflammatory rheumatic diseases, endocrine disturbances).
4.	Major depressive disorder with severe symptoms or suicide ideation, or other major Axis I diagnoses of the DSM-IV-TR (American Psychiatric Association, 2000).
5.	Severe hypnotic dependence.
6.	Having symptoms of sleep-disruptive comorbidities with insomnia.
7.	Having an apnea-hypopnea index or periodic limb movement (PLM) related arousal index of 15 or more per hour on a polisomnography (PSG) recording.
8.	To be receiving another psychological or physical therapy at the time of the study.

Measures. Polysomnography (PSG)

A domiciliary PSG recording (with a SomnoScreen PSG-Tele, SomnoMedics) was used to collect information from key sleep parameters in patients with FM. The PSG recordings included electroencephalography in the frontal, central, parietal, and occipital regions (F_Z/A1, C_Z/A1, P_Z/A1, O_Z/A1), bilateral electrooculography, bilateral submental and anterior tibial electromyography and respiratory variables (chest belt, respiratory thermistance and oximetry). Sleep stages were scored visually according to the criteria of Rechtschaffen and Kales (1968) using 30 seconds' epochs. Table 2 contains a brief description of the sleep variables analysis.

TABLE 2. Sleep variables analysis in the PSG.

Time in bed (TIB) (hours)	Period of time between bedtime and awakening in the morning.
Total sleep time (TST) (hours)	Equal to total sleep period less movement and awake time.
Wake percentage	Percentage of time awake scored from bedtime to the final wake-up.
% REM	Total time spent in REM sleep as a percentage of TST.
Stage 1, 2	Total time spent in stage 1, stage 2, stage 3 and stage 4 sleep as a
3 and 4 percentages	percentage of TST.
Light sleep	(stages 1+2).
Deep sleep	(stages 3+4).
Sleep efficiency	The proportion of sleep in the period potentially filled by sleep: ratio of TST to TIB as a percentage.
NREM sleep latency	The time period measured from bedtime to the beginning of sleep.
REM latency	The period of time from sleep onset to the first appearance of REM sleep.
REM density	Average rate of the whole REM phase with mini REM epochs (3 sec.).
Number of awakenings > 3 minute (index)	Number of wake periods longer than 3 minute during TIB (index: per hour of sleep).
Wake after sleep onset	Time spent awake after sleep onset had occurred.
Arousals index	The average number of arousals per hour of sleep.

Procedure

The evaluation and therapeutic treatment (CBT-I and SH) of sleep disorders in patients with FM was carried out at the Clinical Psychology Unit of the Faculty of Psychology. The whole evaluation consisted of two sessions of individual interviews focusing on the origin and evolution of the problem and domiciliary PSG. Three female CBT experts with experience in FM provided the therapy guided by a treatment manual designed for the study. Each therapist applied both treatments (CBT-I and SH). Therapists delivered CBT-I and SH treatment in 6 weekly groups sessions. Each session included 5-6 participants and lasted around 90 minutes. The CBT-I program was designed according the works of Edinger *et al*. (2005), and met the recommendations of the American Academy of Sleep Medicine (Morgenthaler *et al.,* 2006). Subjects who participated in SH therapy just received sleep hygiene instructions and were offered CBT-I after their post-treatment assessment. The contents of the SH therapy can be seen in Table 3. Also, all patients continued with their usual medical treatment for FM. All participants were on stable doses of medication during the trial (see Table 4). On the consumption of medicaments, 4 patients in the CBT-I group and 2 patients in the SH group consumed occasionally benzodiazepines (less than once a week). Moreover, most patients in both groups consumed regularly non-benzodiazepine anxiolytics (those patients with severe hypnotics dependence were excluded from the study), and antidepressants. In relation to the latter category of medicaments, although they can affect sleep, we must specify that patients had taken these medicaments for months before the psychological interventions, so we think that the possible effect on sleep was controlled.

TABLE 3. Contents of the CBT-I treatment and SH therapy.

	CBT-I sessions	*SH sessions*
Session 1	Information about the relationship between sleep and FM, basic notions about sleep (*e.g.,* sleep stages, sleep functions, effects of sleep deprivation on wake functioning, explanation of insomnia) and SH education.	Participants were given the same information about sleep as the CBT-I group.
Session 2	Sleep restriction therapy combined with stimulus control instructions.	Sleep hygiene rules related to environmental factors.
Session 3	Relaxation training (a combination of passive relaxation and imagery training).	Lifestyle factors that influence sleep (consumption of stimulants and other substances).
Session 4 and 5	Cognitive therapy for the dysfunctional beliefs related to insomnia.	Information about diet and physical exercise, respectively.
Session 6	Maintaining achievements and preventing relapses.	Similiar as the CBT-I group.

Study desing and statistical analysis

This was a controlled randomized trial or "experimental design with an independent variable (IV) and random groups" in which the IV was the type of treatment to be received by the subjects (CBT-I and SH). Statistical analysis was performed using SPSS 15.0 for Windows. Non-parametric statistical tests were used because they are recommended when the sample size is less than 15 (Bryman and Cramer, 1990). To compare the groups on demographic and clinical variables at baseline the Mann-Whitney's *U* test for interval data and the Pearson chi-square (χ^2) test for nominal data were computed. In order to examine the therapeutic changes between-group in PSG parameters the Mann-Whitney's *U* test was used. Finally, the therapeutic changes intra-group in PSG parameters were analyzed via the Wilcoxon test.

Results

Table 4 shows the demographic and clinical characteristics of the FM sample. The results of the non-parametric tests performed (Mann-Whitney's *U* and the Pearson chi-square test, showed that the two groups (CBT-I *vs*. SH) did not differ significantly in terms of age, marital status and work, education, and clinical variables such as years since diagnosis of FM, insomnia or type of insomnia problem and drug intake (all $p > .05$).

TABLE 4. Demographic and clinical characteristics of the FM sample completing the domiciliary PSG study.

Variable	*Total sample (n=26)*	*CBT-I group (n=13)*	*SH group (n=13)*	*p value*
Age, mean (SD)	46.79 (5.15)	44.83 (5.30)	48.75 (4.37)	.07
Education (%)				.08
Basic education	31.8	18.2	45.5	
High school	27.3	45.5	9.1	
Professional instruction	22.7	9.1	36.4	
University studies	18.2	27.3	9.1	
Marital status (%)				.386
Married	92.3	92.3	92.3	
Single	3.8	0	7.7	
Divorced or widowed	3.8	7.7	0	
Work status (%)				.094
Currently employed	50.0	69.2	30.8	
Unemployed	23.1	7.7	38.5	
Disabled	26.9	23.1	30.8	
Duration of FM (years), mean (SD)	5.02 (4.28)	4.67 (3.66)	5.34 (4.91)	.913
Duration of sleep problem (years), mean (SD)	11.25 (9.08)	11.41 (9.24)	9.85 (8.23)	.682
Nature of sleep problem				
Onset (%)	69.3	69.3	69.5	.884
Maintenance (%)	84.6	84.7	84.6	.836
Early awakening (%)	76.9	69.3	84.6	.661
Drug intake (%)				
Antidepressants	50.0	45.5	53.8	.682
Anxiolytics	63.6	61.5	62.5	.916
Anti-inflammatory	63.6	69.2	66.7	.772
Analgesics	72.7	69.2	70.8	.851

Table 5 shows PSG variables before and after CBT-I or SH therapy. As can be seen, the results of the Mann-Whitney's *U* test indicate that prior to treatment there were no statistically significant differences in any of the PSG variables between the two groups (CBT-I and SH) (*U* values between 75.00 and 84.00, $p > .05$). Secondly, a Wilcoxon test was carried out to determine whether there were any differences in the PSG variables analyzed pre-post treatment in each treatment group (CBT-I and the SH). The results for the active treatment group receiving CBT-I showed a decrease in time-in-bed [$z = -2.62$, $p < .01$] and wake percentage [$z = -2.41$, $p < .05$] after treatment. Thus, sleep efficiency [$z = -2.41$, $p < .05$] improved, almost reaching normal levels. As regards sleep architecture, the results revealed a decrease in the percentage of non-rapid eye movement (NREM) stage 1 sleep [$z = -2.90$, $p < .01$] and an increase in the percentage of NREM stage 3 sleep [$z = -2.20$, $p < .05$] and NREM stage 4 sleep [$z = -2.19$, $p < .05$]. No differences were observed between pre-post treatment in the percentage of REM sleep [$z = -.17$, $p = .86$] and NREM stage 2 sleep [$z = -1.15$, $p = .24$]. In addition, the results showed light sleep (NREM stages 1 and 2) decreased [$z = -2.20$, $p < .05$] and deep sleep (NREM stages 3 and 4) increased [$z = -2.55$, $p < .01$] after CBT-I. No differences were observed in sleep duration (hours) [$z = -1.37$, $p = .12$], NREM sleep latency [$z = -1.49$, $p = .136$], REM latency

[z = -1.17, p= .23], % REM density [z = -.31, p= .75], wake after sleep onset [z = -.31, p= .75], number of awakenings greater than 3 minutes [z = -1.67, p = .09] and arousal index [z = -.80, p = .42]. Subjects participating in SH group therapy showed no significant improvements (z values between -1.50 and -.15, p > .05). Finally, we checked for significant differences in PSG sleep parameters post-treatment between the CBT-I group *vs.* HS group. The Mann-Whitney's U test revealed statistically significant differences in three of the PSG variables analyzed, namely % NREM stage 1 sleep [U = 44.50, p < .05], % stage 4 sleep [U = 48.00, p < .05] and deep sleep (NREM stages 3 and 4) [U = 44.50, p <.05]. As can be seen in the mean scores (table 5), in the CBT-I group, a lower percentage of NREM stage 1, and a higher percentage of NREM stage 4 and deep sleep (stage 3 and 4) were observed, compared with the group that received only HS.

TABLE 5. Polysomnographic measures of the group assigned a cognitive-behavioral therapy for insomnia (CBT-I) and the group sleep hygiene (SH).

	CBT-I group			*SH group*			*CBT-I vs. HS*	*CBT-I vs. HS*
	Mean (Standard deviation)			*Mean (Standard deviation)*			*Pre-treatment*	*Post-treatment*
PSG variables	*Pre-treatment*	*Post-treatment*	*Z*	*Pre-treatment*	*Post-treatment*	*Z*	*U*	*U*
Total sleep time (hours)	7:03 (1:04)	6:53 (2:19)	-1.37	7:31 (0:54)	6:57 (0:55)	-1.21	84.00	77.00
Time in bed (hours)	8:54 (0:40)	8:21 (0:53)	-2.62**	8:31 (0:53)	7:45 (1:10)	-1.50	68.00	68.00
Wake percentage	15.51 (9.38)	12.51 (9.47)	-2.41*	11.56 (6.18)	10.06 (3.67)	-.52	64.00	77.00
% REM	23.88 (6.22)	23.83 (5.66)	-.17	22.02 (6.30)	25.43 (9.72)	-1.50	75.00	76.00
% Stage 1	6.89 (4.20)	4.55 (2.23)	-2.90**	6.50 (3.02)	7.00 (2.87)	-.80	80.00	44.50*
% Stage 2	54.05 (9.44)	50.74 (9.28)	-1.15	52.71 (7.50)	52.51 (7.78)	-.80	77.00	70.50
% Stage 3	10.20 (3.93)	13.17 (4.75)	-2.20*	11.24 (4.40)	11.36 (5.91)	-.21	77.00	72.00
% Stage 4	4.81 (3.67)	7.43 (5.56)	-2.19*	4.51 (4.55)	3.62 (3.82)	-.96	78.00	48.00*
Light sleep	60.95 (9.88)	55.26 (9.64)	-2.20*	62.20(7.78)	59.53 (8.24)	-.94	72.00	57.50
Deep sleep	15.03 (5.68)	20.58 (8.41)	-2.55**	15.76 (6.53)	14.97 (7.36)	-.31	81.00	44.50*
Sleep efficiency	84.48 (9.39)	87.48 (9.47)	-2.41*	88.43 (6.18)	89.29 (3.67)	-.52	64.00	77.00
NREM sleep latency	0:27 (0:24)	0:24 (0:45)	-1.49	0:19 (0:16)	0:15 (0:13)	-.80	58.00	74.00
REM latency	1:53 (0:55)	1:35 (0:56)	-1.17	2:05 (1:15)	1.55 (1.34)	-.15	64.00	83.00
% REM density	13.00 (8.57)	13.00 (6.32)	-.31	9.46 (9.78)	9.85 (6.40)	-.26	64.50	56.55
Nº of awakenings > 3 min.	3.33 (2.09)	2.15 (2.37)	-1.67	2.15 (1.62)	1.77 (1.92)	-.73	61.50	77.50
Wake after sleep onset	0:40 (0:18)	0:36 (0:23)	-.31	0.31 (0.22)	0.31 (0.20)	-.94	79.00	76.00
Arousals	10.55 (4.34)	13.18 (15.35)	-.80	13.86 (9.28)	13.56 (13.82)	-.94	74.00	80.00

* p < .05; ** p < .01

Discussion

This clinical trial provides new evidence that the use of CBT-I in women with FM can improve objective sleep disturbance parameters. The few studies identified in the literature that have used PSG to evaluate sleep in patients with FM have reported relevant findings, including shorter total sleep time, greater sleep fragmentation, greater sleep latency, less sleep efficiency, an increase in NREM stage 1 sleep and a reduction of the quantity of NREM stage 3 and 4 sleep compared with healthy controls (Besteiro *et al*., 2011; Moldofsky, 2001, 2008; Roizenblatt *et al.*, 2001). Moldofsky's studies in the seventies showed that patients with FM did not reach NREM sleep stages 3 and 4, *i.e.* the deepest and most restful phases of sleep. Research comparing sleep problems in patients with FM with those observed in other chronic pain diseases, including rheumatoid arthritis, have found that FM patients reported more insomnia, less contentment with sleep and more lack of deep and restful sleep in comparison to rheumatoid arthritis patients (Belt, Kronholm, and Kauppi, 2009).

In the present study, patients with FM showed at pre-treatment sleep onset insomnia, maintenance and early awakening (see Table 4). However, upon completion of treatment, the CBT-I group evidenced improvements in sleep efficiency to almost normal levels and a decrease in wake percentage and time-in-bed. Significant changes were also observed in sleep architecture; specifically, a decrease in the percentage of NREM stage 1 sleep and an increase in the percentage of NREM stages 3 and 4 sleep. Similarly, light sleep (NREM stages 1 and 2) decreased, accompanied by increased deep sleep (NREM stages 3 and 4). Despite the small size of the sample, the results showed objective evidence of a change in sleep features. Therefore, although no changes were observed in the total sleep time of patients with FM, significant differences were observed in the percentages of deepest sleep, which increased after intervention, and a decrease the percentage of wake that provided the most restful sleep. Although both sleep quality and sleep quantity parameters are relevant, the association with different health indicators is stronger in the case of the former (Miró, Cano Lozano, and Buela Casal, 2005; Pilcher and Ott, 1998). Deep sleep (stages 3 and 4) has been related with corporal and neurological restoration, and these stages are closely related to the correct immune system functioning (Buela-Casal and Miró, 2001). The improvement in these phases observed in this study could have a great positive impact on other symptoms of FM and on the actual severity of the syndrome. However, further research is necessary in order to determine how these changes in objective sleep parameters are related to the improvement of other FM symptoms. Moreover, time-in-bed decreases and sleep efficiency increases, indicating an improvement in SH. These clinical parameters are normally used as evidence of improvements in sleep in insomnia treatment studies (Morin and Espie, 2003).

In this study, the patients in the SH therapy group showed no improvements in any parameter. These findings do not coincide with those reported elsewhere in the literature. For example, in a recent study Miró, Lupiañez *et al.* (2011) reported that 55% of the subjects in their SH group displayd significant clinical changes in sleep quality compared with 85% in the CBT-I group. Edinger *et al.* (2005) also reported that the participants receiving SH therapy had reduced their nocturnal wake time by nearly 20% at the end of the study, compared with a 50% decrease in patients receiving CBT-I therapy. Patients receiving CBT-I also showed a higher rate of sleep improvement (57%) compared with patients in SH therapy (17%). A possible explanation for these results may be the evaluation instruments used. In our study, the pre- and post-treatment changes observed in sleep variables after the application of CBT-I and SH therapy were evaluated using polysomnographic measurements, whereas in the aforementioned studies measurements were taken using actigraphy and subjective measurements that provided different and complementary information to polysomnography. Many studies in the literature argue that therapy groups based on education (*e.g.*, sleep hygiene) produce improvements, albeit more modest than those obtained with more structured therapy groups. SH may slightly improve the subjective sensation of sleeping better but it is not a sufficiently powerful treatment to change objective sleep parameters (for review about use of sleep hygiene in the treatment of insomnia see Stepanski and Wyatt, 2003).

Moreover, some studies have also examined CBT-I with chronic pain patients and obtained positive results, but basically using questionnaires. Previous studies have suggested that improving sleep quality (rapid sleep onset, absence of early awakening and restorative sleep) in chronic widespread pain subjects could decrease pain (Davies *et al.*, 2008), as well as impact on daily life functioning and depression. In osteoarthritis patients, Vitiello, Rybarczyk, Von Korff, and Stepanski (2009) observed that patients receiving CBT-I reported significantly decreased sleep latency and wake after sleep onset and increased sleep efficiency after treatment, compared with before treatment. They also reported significantly reduced pain. One-year follow-up found maintenance of improved sleep (in sleep latency and wake after sleep onset and increased sleep efficiency and total sleep time) and reduced pain in the CBT-I group. Finally, a recent study conducted for Jungquist *et al.* (2010) shows that CBT-I can significantly improve sleep and daily functioning in patients suffering from chronic neck or back pain. After eight weeks, participants reported significant improvements in sleep quality and also a reduction in the extent to which pain interfered in daily activities. Specifically, subjects receiving CBT-I compared with controls (who did not receive directed form of therapy was provided for pain, depression, or sleep disturbance) exhibited significant decreases in sleep latency (time to fall asleep), wake after sleep onset, number of awakenings and significant improvements in sleep efficiency (Jungquist *et al.*, 2010).

However, although our study shows that CBT-I improves sleep quality in FM patients, evidenced by increased deep sleep and efficiency and decreased light sleep and awakenings, little research has been carried out into the therapeutic potential of CBT-I in these patients. Moreover, sleep problems in FM are treated, indirectly, with analgesic medication (opioids, tricyclic antidepressants or anticonvulsants) and sedative hypnotics that promote sleep through analgesic and soporific effects (Smith and Haythornthwaite, 2004). These effects may increase sleep quantity but do not usually reduce sleep complaints. Hence, since current medication is unable to improve sleep quality, pain intensity or quality of life, it seems obvious that CBT-I is a promising treatment option for inclusion in current FM treatments.

It is important to recognize various additional limitations in the interpretation of the results obtained in this study. Firstly, the size of the sample. However, in spite of the small size of the sample, results showed changes in PSG sleep variables. Another limitation is the frequent use of different medication. Most patients in our study took antidepressants, analgesic or anxiolytic medication during treatment. However, and although this may be considered a limitation of the study, it is important to remember that the medication was maintained constant throughout the entire trial. Although there was a random allocation of patients to treatment groups, another limitation of the study is the lack of uniform distribution of employment status in groups (although the differences are not statistically significant). Finally only women were included in this study since prevalence is lower in men.

To summarize, CBT-I may be a promising sleep therapy for FM patients. However, further research is necessary in the future to replicate these results with larger samples

of FM patients, as well as in patients recruited in other contexts, such as patients' associations. Another future line of research could focus on establishing the relative efficacy of CBT-I compared with common psychological and medical treatment and determining what the study of sleep can contribute to current psychological treatment, in order to improve the symptoms and the quality of life of these patients. Finally, future studies must be carried out using PSG to analyze how treatment can improve different variables, including pain, daily functioning and cognitive function, and their relationships with the improvements observed in sleep parameters.

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