

Effect of Acute Physical Exercise on Patients with Chronic Primary Insomnia

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Study Objectives: The aim was to assess and to compare the acute effects of three different modalities of physical exercise on sleep pattern of patients with chronic primary insomnia.

Methods: Forty-eight insomnia patients, 38 female (mean age 44.4 ± 8 y) were randomly assigned to 4 groups: control (CTR, n = 12), moderate-intensity aerobic exercise (MAE, n = 12), high-intensity aerobic exercise (HAE, n = 12), and moderate-intensity resistance exercise (MRE, n = 12). The patients were assessed on sleep pattern (by polysomnogram and daily sleep log) and anxiety (STAI) before and after the acute exercise.

Results: The polysomnogram data showed reduction in the sleep onset latency (SOL) (55%) and in the total wake time (TWT) (30%); increase in total sleep time (TST) (18%), and

in the sleep efficiency (SE) (13%) in the MAE group. The daily sleep log data showed increase in the TST (26%) and reduction in the SOL (39%). In addition, reduction (15%) in anxiety was also observed after moderate-intensity aerobic exercise.

Conclusions: Acute moderate-intensity aerobic exercise appears to reduce pre-sleep anxiety and improve sleep in patients with chronic primary insomnia.

Keywords: Insomniacs, physical activity, total sleep time, anxiety, polysomnography.

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Chronic primary insomnia is a sleep disorder characterized by long-term difficulties with initiating and maintaining sleep, waking up too early, or nonrestorative sleep. It is often associated with occupational and psychosocial impairments such as daytime fatigue, mood disturbances, cognitive deficits, and poor quality of life.¹ In addition, heightened anxiety about sleep, which is often the result of a psychophysiological arousal associated with conditioned sleep difficulties has been reported.² The prevalence of chronic insomnia in the general population is between 10% and 15%,³ is common in adulthood, and predominantly affects women.

Pharmacotherapy is the most often prescribed remedy for insomnia. However, sleep-inducing substances often cause side-effects and may not be recommended for long-term treatment.⁴ Consequently, non-pharmacological therapies have been suggested in the literature, most frequently cognitive and behavioral therapies.^{5,6} According to Morin et al.,⁷ those therapies are responsible for reduction of symptoms in 70% to 80% of treated patients.

Nevertheless, the choice of a non-pharmacologic therapy might result in a high maintenance cost, since it requires close follow-up. Physical exercise has been proposed in the literature as a non-drug treatment alternative with low cost and easy access.⁸ This recommendation is based on evidence including: the beneficial effects of exercise on the sleep quality of good sleepers^{9,10} the epidemiological findings that physically active individuals have fewer complaints of insomnia than sedentary individuals¹¹; and the assumption that low levels of physical ac-

BRIEF SUMMARY

Current Knowledge/Study Rationale: Physical exercise has been proposed in the literature as a non-drug treatment alternative with low cost and easy access, however, no evidence of the best type, intensity or duration of exercise. The purpose of this study was to evaluate three different modes of acute exercise on the sleep of patients with chronic primary insomnia.

Study Impact: This is the first study to find significant effect on sleep of insomniacs after acute aerobic exercise. These data open the field for further research to assess the chronic effects of exercise, and later to compare with conventional therapies.

tivity and physical exercise are associated with the prevalence, incidence, and persistence of insomnia.¹² In spite of that, only one experimental study has been carried out to assess the effect of physical exercise on the sleep of adult insomnia patients. In that study, the authors detected a trend towards an increase in the total sleep time and a reduction in the sleep latency and wake time after sleep onset of volunteers who participated in a 4-week trial.¹³ Additional data have been published in older adults with mean age of 61 years, with sleep complaints, treated with moderate-intensity aerobic exercise for 12 months. The authors showed a significant improvement in sleep onset latency and in the sensation of refreshed sleep using sleep logs.¹⁴

It has been reported that there is an increase in anxiety scores prior to sleep in primary insomnia patients.¹⁵ The literature also suggests that moderate- and high-intensity physical exercises,^{16,17} as well as moderate-intensity resistance exercises^{18,19}

might reduce anxiety for up to 5 h.¹⁹ However, there is no evidence for an association between reduction in anxiety with improvement in the sleep quality of insomniacs.

Thus, the aim of this study was to assess and to compare the acute effect of 3 types of physical exercise—moderate-intensity aerobic exercise, high-intensity aerobic exercise, and moderate-intensity resistance exercise—on pre-sleep anxiety state and sleep quality of patients with chronic primary insomnia.

METHODS

The study was previously approved by the ethics committee of Federal University of Sao Paulo. Inclusion criteria were: (a) age between 30 and 55 years old; (b) clinical diagnosis of primary insomnia according to the DSM-IV¹ and the ICSD²; (c) history of insomnia complaints > 6 months; (d) at least one complaint of daytime impairment resulting from insomnia (regarding mood, cognition, or perception of fatigue). Exclusion criteria were: (a) evidence of association of insomnia with medical conditions or side-effects of medications; (b) use of medication or psychotherapeutic components for insomnia or other psychiatric disorder; (c) depression (Beck depression Inventory score > 20)²¹ or other psychiatric disorders; (d) shift work; (e) abnormalities in the cardiology evaluation, resting ECG, or exercise stress test; (f) blood test results contraindicating physical exercise; (g) practice of regular (≥ 1 time/week) physical exercise.

A total of 243 volunteers participated in the initial selection. Of this number, 23 were excluded for high score (> 20) on the depression inventory, 22 for other major psychiatric disorders (such as severe anxiety disorders), 30 for regular physical exercise, 14 for shiftwork, 76 for pharmacologic treatment, 23 for lack of insomnia symptoms at the time of the evaluation, and 8 for abnormalities in blood tests, electrocardiogram, or exercise test. The remaining 48 participants (38 female) were enrolled to participate in the study. Written informed consent was obtained from all volunteers. They were randomly allocated to the groups of moderate-intensity aerobic exercise (MAE, $n = 12$), high-intensity aerobic exercise (HAE, $n = 12$), moderate-intensity resistance exercise (MRE, $n = 12$) and control (CTL, $n = 12$).

Design and Procedures

This study involved 2 evaluations: the first at entry (baseline) and the second after exercise (post-exercise). Full polysomnography for adaptation to sleep laboratory was performed, followed by a baseline recording night. Patients arrived at the sleep laboratory at 21:00, completed the STAI (state anxiety subscale), and then underwent baseline polysomnographic recording. They filled out the sleep log on subsequent morning. In the post-exercise evaluation, subjects arrived at exercise laboratory at 17:30 and started the exercise session at 18:00. After the exercise session, patients went to sleep laboratory at 21:00 for their third polysomnographic recording, when they again answered the STAI (state anxiety subscale) questionnaire. As in the baseline evaluation, they filled out the sleep log the subsequent morning.

The volunteers in the control group did not attend the exercise program but underwent the same evaluations as did the exercise group.

MEASURES

Polysomnography

The assisted polysomnography was carried out 3 times, the first serving as an adaptation night, the second serving as a baseline night, and the third proceeded by the one-session exercise intervention. Electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), air flow (oral and nasal), nasal pressure transducer, respiratory effort (thoracic and abdominal), tracheal microphone, and oxygen saturation parameters were recorded. Sleep variables analyzed were: total sleep time (TST), sleep efficiency (SE; ratio between total sleep time and total time of recording multiplied by 100), sleep onset latency (SOL), REM latency (LREM), total wake time (TWA), wake time after sleep onset (WASO), arousals, apnea hypopnea index (AHI), periodic leg movements (PLM), and percentage of sleep stages. The analysis of the events in the polysomnography was carried out by 2 investigators who used international criteria²²⁻²⁴ and were blind to the grouping of the volunteers. The equipment used was the digital system EMBLA (EMBLA S7000, Embla Systems Inc., CO, USA).

Daily Sleep Log

The sleep log was filled out twice—at the baseline evaluation and again at the post-exercise evaluation. In this sleep log, the following variables were analyzed: SOL, TST, and SE. The volunteers were instructed to fill it out in the morning upon waking.

STAI Questionnaire-State Scale

Prior sleep state anxiety was assessed with the State-Trait Anxiety Inventory–STAI,²⁵ the Portuguese version of which was validated by Gorenstein and Andrade.²⁶ This scale encompasses 20 items and provides a one-dimensional measurement of anxiety. The volunteers were instructed to answer it 30 minutes before going to sleep at baseline and at the post-exercise evaluation.

Menstrual Cycle

The menstrual cycle of the women of reproductive age was controlled for by means of a description of the date of their menstrual period regarding the 2 months preceding the onset of the study. The baseline and post-exercise evaluations were carried out during their proliferative phase (1st to 10th day of the post-menstrual period), which was recommended by Cox et al.¹⁷ for the evaluation of anxiety in women.

Exercise Protocols

Moderate-intensity Aerobic Exercise and High-intensity Aerobic Exercise

The volunteers in the MAE and HAE groups performed an incremental exercise on a treadmill (Life Fitness 9500HR, USA). The initial running speed was 4 km/h (for 3-min warm-up) with increments of 0.5 km/h every minute up to voluntary exhaustion. The ventilatory variables—minute ventilation (VE), oxygen consumption (VO₂), and carbon dioxide produc-

Table 1—Sample descriptive analysis

Characteristics	CTL (n = 12)	MAE (n = 12)	HAE (n = 12)	MRE (n = 12)	Total (n = 48)
Gender (M/F)	3/9	2/10	3/9	2/10	10/38
Age (years)	45.2 ± 8	47.7 ± 7	42.2 ± 9	42.4 ± 9	44.4 ± 8
BMI (kg/m ²)	26.4 ± 6	26.1 ± 3	24.4 ± 4	23.4 ± 3	25.0 ± 4
AHI	8.6 ± 9	9.6 ± 8	7.0 ± 5	5.1 ± 6	7.6 ± 7
PLM	3.6 ± 8	1.0 ± 2	1.0 ± 2	1.4 ± 3	2.4 ± 5
Duration of insomnia (years)	9.5 ± 11	10.9 ± 10	6.4 ± 8	9.6 ± 7	9.1 ± 9
STAI trait (score)	44 ± 9	43 ± 7	40 ± 7	38 ± 8	41 ± 8
Beck Depression Inventory (score)	12 ± 5	10 ± 4	10 ± 6	9 ± 4	10 ± 5

MRE, moderate-intensity resistance exercise; MAE, moderate-intensity aerobic exercise; HAE, high-intensity aerobic exercise; CTL, control; BMI, body mass index; AHI, apnea hypopnea index; PLM, periodic leg movements. One-way ANOVA, no significant results, $p > 0.05$; data are expressed as mean ± SD.

tion (VCO_2)—were collected breath by breath by a gas analyzer (Quark PFT4, Rome, Italy). To analyze the data, we used means of 20-sec intervals and considered the highest VO_2 obtained in the last interval of the test as the peak oxygen consumption (VO_{2peak}).

The intensity of exercise in the MAE was based on the first ventilatory threshold (VT_1), which is considered moderate intensity,^{27,28} and the training session of this group on a treadmill lasted 50 continuous minutes. The intensity of training in the HAE group was based on the second ventilatory threshold (VT_2), considered high-intensity,^{28,29} and the exercise session consisted of 3 periods of 10 min of exercise on a treadmill alternating with 10 min of rest.

Moderate-intensity Resistance Exercise

To obtain overload training, we used the recommendations for the one repetition maximum (1 RM) test proposed by Kraemer and Fry³⁰ to evaluate MRE volunteers. The program incorporated the following exercises: shoulder press, chest press, vertical traction, leg press, leg curl, leg extension, abdominal crunch, and lower back (Technogym, Italy). Three sets of 10 repetitions at 50% of 1 RM were performed, interspersed with 90-s recovery intervals. The exercise session time was approximately 50 minutes.

Statistical Analysis

The program STATISTICA (Statsoft, Inc, version 6.0) was used for data analysis. All variables presented normal distribution ($p > 0.05$) according to the normal probabilistic graph and the Kolmogorov-Smirnov test. One-way analysis of variance (ANOVA) was used to determine the homogeneity of the descriptive variables of the groups studied at baseline. We used a repeated-measure ANOVA (baseline versus post-exercise) to compare the subjective reports (the variables extracted from the sleep log and from the STAI questionnaire) and objective reports (polysomnogram) of the 4 groups; significant effects were followed by the Duncan post hoc test. Pearson correlation coefficient was used to assess correlation between pre-sleep anxiety reduction and sleep improvements. Results were expressed in delta% ($D\% = \text{post-exercise value} - \text{baseline value} / \text{baseline value}$). A calculation of effect size (post-exercise mean - base-

line mean/ SD of the baseline mean = effect size) was used for the sleep variables that were significantly different after the exercise session.⁹ Data are expressed as mean ± SD. Significance levels were set at $p < 0.05$.

RESULTS

Participants

The demographic characteristics are presented in **Table 1**. No differences were found between groups at baseline.

Physiological Parameters

The volunteers in the MAE presented VO_{2peak} of 27.8 ± 7 mL/kg/min, HR_{max} of 161 ± 18 bpm, speed of 5.2 ± 1 km/h in VT_1 , and HR of 110 ± 12 bpm in VT_1 . In the HAE group, the VO_{2peak} was 29.0 ± 7 mL/kg/min, the HR_{max} 168 ± 15 bpm, speed in VT_2 was 6.9 ± 1 km/h, and HR in VT_2 was 146 ± 19 bpm. The MRE group used the following training loads: 55.4 ± 21 lb on the chest press, 25.8 ± 10 lb on the shoulder press, 85.8 ± 29 lb on the vertical traction, 128.3 ± 56 lb on the leg press, 68.7 ± 25 lb on the leg curl, 83.3 ± 26 lb on the leg extension, 43.7 ± 15 lb on the abdominal crunch, and 57.0 ± 22 lb on the lower back.

Polysomnography

No significant differences were detected across groups for any baseline variables. The repeated-measures ANOVA showed a reduction in the SOL (55%) and in the TWT (30%), as well as an increase in TST (18%) and SE (13%) in the MAE in the post-exercise evaluation (**Table 2**). The calculation of effect size showed an effect of -0.67 for the SOL, 0.53 for the SE, 0.90 for the TST, and -0.49 for the TWT. The percentage of sleep stages did not change after the training sessions (**Table 3**).

Sleep Log

We observed a significant reduction in the SOL (39%) and an increase in the TST (26%) in the MAE after exercise session (**Table 4**). The calculation of *effect size* showed an effect of -0.70 in the SOL and 1.0 in the TST.

Table 2—Polysomnographic variables

Variables	CTL (n = 12)	MAE (n = 12)	HAE (n = 12)	MRE (n = 12)
SOL (min)				
Baseline	19.7 ± 21	37.6 ± 31	22.4 ± 19	27.7 ± 29
Post-exercise	13.8 ± 14	16.8 ± 16*	18.7 ± 23	24.1 ± 29
REM latency (min)				
Baseline	86.6 ± 38	116.3 ± 90	101.2 ± 92	106.6 ± 38
Post-exercise	107.8 ± 48	118.9 ± 66	105.7 ± 41	112.0 ± 91
SE (%)				
Baseline	78.7 ± 13	71.8 ± 17	78.6 ± 15	82.6 ± 8
Post-exercise	81.9 ± 12	80.9 ± 13*	80.1 ± 13	83.1 ± 10
TST (h)				
Baseline	5.4 ± 1	4.9 ± 1	5.5 ± 1	5.7 ± 1
Post-exercise	5.7 ± 1	5.8 ± 1*	5.4 ± 1	5.8 ± 1
WASO (min)				
Baseline	69.7 ± 36	80.0 ± 64	67.5 ± 58	45.5 ± 20
Post-exercise	60.7 ± 41	65.3 ± 48	63.3 ± 36	48.0 ± 34
TWT (min)				
Baseline	89.4 ± 54	117.7 ± 72	89.9 ± 66	73.2 ± 37
Post-exercise	74.6 ± 51	82.0 ± 56*	81.9 ± 54	72.2 ± 47
Arousals (events/hour)				
Baseline	13.2 ± 10	12.5 ± 6	12.5 ± 5	10.8 ± 7
Post-exercise	14.8 ± 12	12.8 ± 5	12.9 ± 6	8.4 ± 4
AHI (events/hour)				
Baseline	8.6 ± 9	9.6 ± 8	7.0 ± 5	5.1 ± 6
Post-exercise	8.5 ± 7	8.3 ± 7	6.7 ± 6	4.0 ± 5
PLM (events/hour)				
Baseline	3.6 ± 8	1.0 ± 2	1.0 ± 2	1.4 ± 3
Post-exercise	1.5 ± 2	0.5 ± 1	1.3 ± 4	1.8 ± 5

MRE, moderate-intensity resistance exercise; MAE, moderate-intensity aerobic exercise; HAE, high-intensity aerobic exercise; CTL, control; TST, total sleep time; SE, sleep efficiency; SOL, sleep onset latency; TWT, total wake time; WASO, wake time after sleep onset; AHI, apnea hypopnea index; PLM, periodic leg movements.

Repeated-measures ANOVA, significant results, $p < 0.05$; data are expressed as mean ± SD; *baseline ≠ post-exercise.

STAI Questionnaire

A significant reduction in pre-sleep anxiety state after the training session was found in the MAE group (15%) (**Table 4**).

STAI Questionnaire versus Sleep Improvements

No significant correlation was observed between: D% STAI-state × D% TST (sleep Log) = ($r = -0.39$); D% STAI-state × D% SOL (sleep Log) = ($r = 0.28$); D% STAI-state × D% SOL (PSG) = ($r = -0.27$); D% STAI-state × D% TWT (PSG) = ($r = -0.42$); D% STAI-state × D% TST (PSG) = ($r = 0.29$); D% STAI-state × D% SE (PSG) = ($r = 0.39$); $p > 0.05$, all.

DISCUSSION

To our knowledge, this is the first study investigating the acute effect of physical exercise on the sleep pattern of patients with chronic primary insomnia. Physical exercises might re-

Table 3—Sleep stages by polysomnography

Variables	CTL (n = 12)	MAE (n = 12)	HAE (n = 12)	MRE (n = 12)
NREM Sleep				
Stage 1 (%)				
Baseline	3.4 ± 2	4.8 ± 6	5.7 ± 5	3.7 ± 2
Post-exercise	3.3 ± 2	4.4 ± 3	5.9 ± 3	3.8 ± 2
Stage 2 (%)				
Baseline	55.3 ± 10	54.4 ± 9	55.3 ± 9	58.0 ± 5
Post-exercise	56.9 ± 6	56.6 ± 8	59.1 ± 12	59.4 ± 10
Stages 3 and 4 (%)				
Baseline	23.1 ± 7	23.5 ± 6	18.8 ± 7	20.4 ± 5
Post-exercise	19.2 ± 7	22.0 ± 12	15.5 ± 8	19.4 ± 6
REM Sleep (%)				
Baseline	18.1 ± 5	17.3 ± 8	20.2 ± 8	17.9 ± 6
Post-exercise	20.5 ± 6	18.7 ± 5	19.4 ± 7	17.5 ± 6

MRE, moderate-intensity resistance exercise; MAE, moderate-intensity aerobic exercise; HAE, high-intensity aerobic exercise; CTL, control. Repeated-measures ANOVA, significant results, $p < 0.05$; data are expressed as mean ± SD; *baseline ≠ post-exercise.

Table 4—Anxiety and sleep log variables

Variables	CTL (n = 12)	MAE (n = 12)	HAE (n = 12)	MRE (n = 12)
STAI State (score)				
Baseline	40.4 ± 13	42.7 ± 7	38.5 ± 11	37.0 ± 7
Post-exercise	38.0 ± 8	36.0 ± 5*	34.0 ± 7	31.4 ± 7
Sleep Log				
SOL (min)				
Baseline	50.0 ± 55	80.8 ± 45	56.2 ± 44	77.5 ± 42
Post-exercise	54.6 ± 54	49.0 ± 32*	33.4 ± 20	53.3 ± 30
SE (%)				
Baseline	63.1 ± 21	64.3 ± 19	65.9 ± 20	66.6 ± 18
Post-exercise	57.9 ± 27	72.4 ± 13	58.1 ± 28	73.0 ± 16
TST (h)				
Baseline	4.2 ± 1	3.9 ± 1	4.3 ± 1	4.5 ± 1
Post-exercise	4.1 ± 2	4.9 ± 1*	3.8 ± 2	4.8 ± 1

MRE, moderate-intensity resistance exercise; MAE, moderate-intensity aerobic exercise; HAE, high-intensity aerobic exercise; CTL, control; TST, total sleep time; SE, sleep efficiency; SOL, sleep onset latency. Repeated-measures ANOVA, significant results, $p < 0.05$; data are expressed as mean ± SD; *baseline ≠ post-exercise.

duce the anxiety state and therefore improve sleep quality.⁸ Primary insomnia patients do not fulfill anxiety disorders criteria, but they may present high levels of pre-sleep anxiety. Indeed, by analyzing the effect of different exercise modalities, we observed that moderate-intensity aerobic exercise significantly reduced anxiety state and improved sleep variables. Decreases in anxiety state after the aerobic exercises performed at moderate intensity have also been observed in healthy, physically active individuals who did not complain of insomnia.^{16,17,31} Morgan²⁰ suggested mechanisms to explain some of these effects: (a) the distraction effect and (b) an increase in monoamine levels (norepinephrine and serotonin). These cognitive and chemical changes due to exercise acute effect may also affect dif-

faculty falling asleep and maintaining sleep among insomniacs. In fact, significant improvements have also been observed in good sleepers after moderate-intensity aerobic exercise. Nevertheless, the changes observed are significant, yet modest, compared to those observed in the MAE group. In a meta-analysis, Youngstedt et al.⁹ described an average increase in TST of 10 minutes in normal volunteers after exercise; while in our population of insomnia patients, the mean increase observed was approximately 1 hour. In good sleepers, physical exercise does not seem to significantly change SOL,⁹ while the reduction in this parameter was 55% in the MAE insomnia group. The same authors suggested that the reduction in the SOL is associated with the time of day that exercise is performed. In this sense, the authors noted that aerobic exercises performed between 4 and 8 hours before bedtime might reduce the SOL. Perhaps the reduction in the SOL found in the present study might be explained by the time when the exercises were performed (18:00), which is 4 to 5 hours before bedtime.

Only one previous study evaluated the effect of physical exercise on the improvement of the sleep of adult patients with insomnia.¹³ In this study, the authors associated the moderate-intensity aerobic exercise with sleep hygiene therapy. After four weeks of intervention, they observed a nonsignificant trend towards an increase in TST and a reduction in SOL and TWT of insomniacs, evaluated by sleep log and actigraphy. Similar results were found in the present study for the sleep variables evaluated by the sleep log and the polysomnogram after a single intervention of moderate-intensity aerobic exercise. In the present study, polysomnography demonstrated an increase in TST (18%) and SE (13%) and a reduction in SOL (55%) and TWT (30%); sleep logs showed an increase in TST (26%) and reduction in SOL (39%). Moreover, the effect size observed for the sleep variables TST and SOL, evaluated by the polysomnogram (0.90 and -0.67) and by the sleep log (1.0 and -0.70) was more remarkable than that observed in the study of Guilleminault et al.¹³ for the same variables evaluated by actigraphy (0.39 and -0.36). A possible explanation for those differences might be the volume and intensity of exercise, since in the study of Guilleminault et al., those parameters were not controlled. Another study performed in older adults with sleep complaints found significant subjective improvement in SOL associated with more refreshed sleep, again with moderate-intensity aerobic exercise.¹⁴

Post-exercise sleep variables were not different between the MAE group and the control group (CTL). This lack of difference might be partially explained by the worse but not significant sleep parameters observed in MAE group at baseline, as well as by the limited number of subjects per group.

One possible explanation for the absence of a significant effect of exercise on anxiety of the subjects might be the high intensity of exercise, as observed in the HAE group. The literature describes positive results of moderate and heavy aerobic exercises on healthy, physically active individuals who apparently have no complaints of insomnia.¹⁶ Results regarding the practice of high-intensity exercise, however, are somewhat controversial. Some authors found a reduction in anxiety after high-intensity exercises performed at 70%, 80%, and 85% of the VO_{2peak} .³² Other authors, however, did not detect any significant alterations after the performance of exercise at similar

intensities.³³ In a study with high-active compared to low-active men, Dishman et al.³⁴ suggested that the anxiolytic effect of high-intensity exercise was exclusive to the high-active population. In this sense, the absence of effects in the HAE group might be related to the level of physical fitness of the subjects, since being sedentary was one of the inclusion criteria for this study. However, caution is needed when speculating on the cause-effect relationship or the absence of an association between an effect and any causes, since no previous studies have taken place to evaluate this factor in this population.

Few studies have evaluated the effects of high-intensity physical exercise on sleep, and those that did presented conflicting results. Shapiro et al.³¹ observed an increase in stages 3 and 4 of NREM sleep and a reduction of REM sleep in physically active good sleepers after graded exercise. Another study, carried out with female good sleepers following a 12-week physical fitness training program at 70% of VO_{2max} observed an increase in stage 2 and a decrease in stages 3 and 4 sleep.³⁵ In the present study, high-intensity aerobic exercise did not show any significant effect on the percentage of sleep stages or on the quality of sleep of the HAE group.

Similarly, the practice of resistance exercise (MRE group) did not yield any significant influence on anxiety state or on the sleep variables of subjects. However, the absence of an effect on the anxiety state in this group does not seem to be associated with the intensity at which the exercise was performed (50% of 1 RM), since the reduction in anxiety state after the practice of resistance exercises at moderate intensity is well established in the literature.³⁶

The absence of improvement in the quality of sleep of young, physically active, good sleepers has also been described in the literature.¹⁰ In contrast, significant effects were observed in depressed elderly after the practice of this kind of exercise.³⁷ According to those results, the improvement in the quality of sleep of that population seems to be more closely mediated by mood improvement than by anxiety state reduction.

In this study we did not observe significant differences among groups, but the acute moderate-intensity aerobic exercise appear to reduce pre-sleep anxiety state and improve sleep in patients with chronic primary insomnia. Regarding the other modalities, high-intensity aerobic exercise and moderate-intensity resistance exercise did not seem to change pre-sleep anxiety or the sleep of insomnia subjects. This is a preliminary study and was performed to select the best type of exercise to acutely improve sleep.

In conclusion there is a potential role for MAE applied in a regular basis to chronic insomnia patients. Whether or not the acute effect remains and the sleep improvement is persistent is a subject for future studies. Our results reinforce that MAE may help reducing anxiety state prior to sleep and sleep quality even in non-insomnia distressed subjects.

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