

Physical exercise performed before bedtime improves the sleep pattern of healthy young good sleepers

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Abstract

To investigate the influence of different intensities and durations of exercise before bedtime on the sleep pattern and core body temperature of individuals considered good sleepers, we selected 17 healthy males and all underwent 5 nonconsecutive days of study. Measurements of polysomnographic parameters and core body temperature were taken at baseline and after each experimental protocol, performed at night. We found increased sleep efficiency ($p = .016$) among all protocols compared with baseline data and increase in REM sleep latency ($p = .047$) between two experiments; there was decrease in the percentage of stage 1 sleep ($p = .046$) and wake after sleep onset ($p = .003$). Core body temperature did not change significantly during the nights following exercise. Exercise performed before sleep does not impair sleep quality; rather, its practice improves sleep in good sleepers who are nonathletes, and may be considered to improve sleep pattern.

Descriptors: Exercise, Core body temperature, Polysomnography

The idea that daily exercise leads to a good night of sleep has been accepted for many years. Physical exercise has been associated with better sleep quality (Ancoli-Israel, 2001) and is accepted as a nonpharmacological intervention for sleep disorders by the American Sleep Disorders Association (Thorpy, 1991). However, the limitations of experimental procedures may affect the analysis of exercise's effect on sleep pattern and efficiency. The reproducibility of objective sleep recordings obtained in the laboratory is limited by several factors compared with subjective sleep recordings, including a relatively short evaluation period (only 1 or 2 days) and a focus on the acute effects of short duration exercise (≤ 60 min) (Youngstedt et al., 2003).

Moreover, previous studies have shown contradictory results regarding the effects of exercise on sleep (Driver & Taylor, 2000; Youngstedt, 2005; Youngstedt, O'Connor, & Dishman, 1997). In part, these contradictions can be attributed to methodological limitations such as a lack or inadequacy of control groups, small

sample sizes, and a focus only on good sleepers or young athletes (Driver & Taylor, 2000; Youngstedt, 2005). Other factors that may account for these differences include differences in the time of day and location where the exercise was performed, resulting in different exposures to daylight, as well as the type, intensity, and duration of exercise and the physical fitness level of the subjects evaluated, preventing appropriate comparisons (Youngstedt, 2005; Youngstedt et al., 2003).

The effect of exercise on sleep appears to be less pronounced in people with higher fitness levels. A study conducted with 16 highly trained cyclists who performed an acute session of 3 h of physical exercise (65%–75% heart rate reserve (HRR)) ending 30 min before bedtime found no change in sleep pattern (Youngstedt, Kripke, & Elliott, 1999). However, most studies have reported positive effects of exercise on sleep, such as increased total sleep time (TST), increased duration of slow-wave sleep (SWS), increased latency to rapid eye movements (REM), and decreased time in REM sleep (Kubitz, Landers, Petruzzello, & Han, 1996; Youngstedt, 2005; Youngstedt et al., 1997).

One important factor impacting sleep pattern, based on sleep hygiene recommendations, is the schedule of exercise. Some studies have indicated that physical exercise in the morning can improve sleep, while the opposite effect can be observed for physical exercise in the evening (Driver & Taylor, 2000; Kubitz et al., 1996; Youngstedt et al., 1997). However, these results remain inconclusive because there is little evidence to suggest that an acute exercise session (O'Connor, Breus, & Youngstedt, 1998) or strenuous exercise (Youngstedt, 2005) close to bedtime adversely impacts sleep.

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Despite evidence suggesting that changes in sleep pattern are caused by physical exercise, it is unclear whether these changes are directly associated with physical exercise or conditions that negatively affect sleep, such as organic and/or neurobehavioral disorders, or if they are predisposing factors for promoting improvements in sleep pattern (Driver & Taylor, 2000).

Therefore, the purpose of this study was to assess the influence of different intensities and durations of physical exercise before bedtime on sleep pattern and body temperature in individuals considered to be good sleepers.

Methods

This study was approved by the Ethics Committee of the Universidade Federal de São Paulo (CEP 1252/07). All participants read and signed the consent form before performing the experimental procedures.

Participants

We selected 18 healthy men (mean age of 27.2 ± 3.6 years) who were classified as good sleepers. All subjects met the following inclusion criteria: (a) have not engaged in more than 60 min per week of regular, moderate to vigorous physical exercise in the last 6 months; (b) do not have physical/organic limitations or any medical condition that would prevent physical exercise; (c) do not have a history of neurological or cognitive disorders; (d) have not had a clinical diagnosis (sleep apnea) or medical/psychiatric condition (chronic pain, depression) responsible for sleep complaints; (e) do not habitually use and have not used illegal drugs, psychotropic drugs, hypnotics, stimulants, or analgesics in the last month; (f) are nonsmokers and consume less than 3 doses of alcohol and less than 300 mg of coffee per day; (g) have a body mass index (BMI) $< 30 \text{ kg/m}^2$; (h) completed primary education or a higher education level and are able to read and understand the consent form; and (i) have not had an irregular sleep routine, worked night shifts, or taken transmeridional trips in the month preceding the study. Participants were recruited via advertisements, flyers, telephone survey, and personal contact by the team responsible for the study. One participant was excluded from the study because of irregular sleep during the experimental protocols.

Experimental design. All participants attended the Centro de Estudos em Psicobiologia e Exercício (CEPE) for 5 nonconsecutive days during the investigation. The interval between experiments was at least 1 week. During the study period, the motor activity of the participants was monitored by wrist actigraphy. To detect sleep disorders or changes in sleep pattern, we applied the Pittsburgh Sleep Quality Index (PSQI) survey for sample selection and the Epworth Sleepiness Scale (ESS) questionnaire. Participants received instructions regarding the proper intake of the thermistor pill, completion of the sleep diary, and actigraphy use. Additionally, we assessed body composition, measured physiological/hormonal factors, and performed a cardiorespiratory fitness test. During the basal period, the testing exercise was conducted to determine the functional capacity and ventilatory thresholds 1 (VT1) and 2 (VT2) for each subject. For this baseline test session, participants underwent progressive running tests on a treadmill (Life Fitness HR 9700), with an initial 4.0 km/h speed and increasing increments of 1.0 km/h per minute until maximum voluntary exhaustion was reached. The purpose of this procedure was to determine each subject's peak oxygen consumption ($\text{VO}_{2\text{peak}}$), defined as the mean

of the two highest O_2 consumption levels observed in the last minute of the cardiac stress test. Ventilatory variables were measured for each breath using a gas analyzer (COSMED, Quark model PFT4, Rome, Italy). Ventilatory parameters, including VT1 and VT2, were determined by pre-established criteria (Wasserman & Koike, 1992). After intensity thresholds were determined for each participant, the intensities of effort were obtained for the subsequent experimental protocols: VT1 30 min, equivalent to 30 min of continuous exercise at VT1 intensity; VT1 60 min, equivalent to 60 min of continuous exercise at VT1 intensity; Delta50 30 min, equivalent to 30 min of continuous exercise at an intensity 50% above VT1 (Demarie et al., 2001); and Delta50 60 min, equivalent to 60 min of continuous activity at an intensity 50% above VT1. After the baseline period, the other exercise protocols were randomized for each participant and administered on four different nights. All exercise tests were conducted between 8:00 and 8:30 pm. After each experimental protocol, the participants had a period of personal hygiene and ate dinner. The subjects were then referred to the Sleep Laboratory for instrumentation, and nocturnal polysomnography was performed thereafter. Bedtime was set between 10:30 and 11:00 pm, and waking time was set between 6:00 and 7:00 am. After waking up and removing the polysomnographic (PSG) equipment, the subjects completed the sleep diary and were allowed to have breakfast and leave the laboratory.

Measures

Objective measures of sleep

Actigraphy. Volunteers were instructed to use the SleepWatch actigraph (Ambulatory Monitoring, Ardsley, NY) on the nondominant wrist continuously during the night before each protocol to verify the presence or absence of sleep restrictions, which were determined by the ActionW software. All subjects were instructed to maintain their normal sleep-wake routine and usual lifestyle until all protocols were completed.

Polysomnography. The recording system consisted of four electroencephalography derivations (electroencephalogram (EEG)—C3-A2, C4-A1, O2-A1, and O1-A2), submental and tibial electromyography (EMG), bilateral electrooculogram (EOG), and electrocardiography (ECG). Breathing was monitored with nasal cannula to measure airflow by a pressure transducer. Oral flow was determined by a thermal sensor, and thoracic and abdominal movements were monitored. To noninvasively measure oxygen saturation (SaO_2), pulse oximetry was conducted by an infrared sensor attached to one distal phalange. We placed a sensor on the sternum to facilitate recording in the supine position. The polysomnographic equipment was set up by a trained technician according to the criteria suggested by Rechtschaffen and Kales (1968) using the Embla 7000 equipment. Sleep parameters were recorded in 30-s intervals during wakefulness and stages 1, 2, 3, 4, and REM sleep. In addition, TST, sleep onset latency, REM sleep latency, and sleep efficiency percentages were measured.

Self-report measures

PSQI. This index was used to assess sleep quality and sleep disorders in the month prior to the experimental procedures. We sought to assess seven components: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The scores of the seven components were summed to produce a total score (range = 0–21). This index was used only in the preselection stage (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989).

Table 1. General Characteristics of Participants (n = 17)

Variables	Mean	SD
Age (years)	27.2	±3.6
Weight (kg)	75.9	±11.8
Height (cm)	174.0	±0.1
BMI (kg/m ²)	25.0	±3.3
Body fat (%)	17.6	±8.5
Lean body mass (%)	82.4	±8.5
Functional capacity		
VO _{2peak} (mL.kg ⁻¹ .min ⁻¹)	46.4	±7.2
VT1 (mL.kg ⁻¹ .min ⁻¹)	28.2	±5.0
VT2 (mL.kg ⁻¹ .min ⁻¹)	37.1	±4.5
PSQI score	4.6	±2.3

Notes. BMI = body mass index; VO_{2peak} = maximal oxygen uptake; VT1 = ventilatory threshold 1; VT2 = ventilatory threshold 2; PSQI = Pittsburgh Sleep Quality Index.

ESS. This survey consisted of eight items assessing the participant’s likelihood of falling asleep in different situations that encouraged sleep (Johns, 1991). The ESS was applied for all periods preceding the PSG.

Sleep diary. This form aimed to obtain information about bedtime and waking hours, estimated time needed to fall asleep, number and duration of awakenings during the night, and subjective emotional state upon waking. The form was completed by all subjects when they woke up in the morning on the same days that the PSG recordings were performed.

Measurements of secondary interest

Body composition. Anthropometric assessments were conducted for all participants to characterize the study sample. It was assessed using the Bod Pod, which is composed of an electronic scale, a plethysmograph, a calibration cylinder, and a computer with the required software. This system measures total body densitometry by dividing body mass by body volume (Dempster & Aitkens, 1995).

Central measurement of body temperature. Core body temperature was monitored at baseline and on days of the experimental protocols by a thermistor pill with a CorTemp HT150002 core temperature sensor coupled with a CorTemp HT150001/150016 (Palmetto, FL) data recorder. This monitoring was recorded continuously throughout the night while the subjects slept in the laboratory.

Statistical analysis. We used SPSS software version 16.0 (SPSS Inc., Chicago, IL) to perform the data analyses. To determine the effect of physical exercise type (basal, VT1 30, VT1 60, Delta50 30, and Delta50 60) on sleep parameters and temperature, we used the Friedman analysis of variance followed by the Wilcoxon test for paired data. The level of significance for all tests was set at $p < .05$.

Results

The characteristics of all participants are presented in Table 1. The sleep parameters measured by actigraphy and ESS confirmed that no subjects experienced sleep deprivation/restriction on the nights before the experimental procedures.

Sleep parameters recorded by PSG are shown in Table 2. The results show that the physical exercise protocols resulted in significant differences in the following: decreased time wake after sleep onset, increased REM sleep latency, increased sleep efficiency percentage, and decreased percentage of stage 1 sleep ($p < .05$).

Core body temperature data are shown in Figure 1. There was no significant difference between basal conditions after the different exercise durations and intensities. We observed a decrease in core body temperature after the completion of physical exercise and lights out. The minimum and maximum temperatures, although not significantly different between the baseline and exercise conditions, varied with respect to means ($\pm SD$) and times as follows: basal (Tmax = 37.34 ± 0.35°C at 8:00 pm; Tmin = 36.53 ± 0.50°C at 3:30 am), VT1 30 min (Tmax = 37.38 ± 0.46°C at 8:00 pm; Tmin = 36.45 ± 0.52°C at 6:00 am), VT1 60 min (Tmax = 37.62°C ± 0.69 at 9:30 pm; Tmin = 36.56 ± 0.27°C at 4:00 am), Delta50 30 min (Tmax = 37.45 ± 0.57°C at 9:00 pm; Tmin = 36.53 ± 0.50°C at 3:30 am), and Delta50 60 min (Tmax = 37.79 ± 0.58°C at 9:30 pm; Tmin = 36.49 ± 0.99°C at 5:30 am).

Discussion

This study shows that physical exercise before bedtime did not impair the sleep pattern of the subjects evaluated. Increased sleep efficiency, longer REM sleep latency, less time wake after sleep onset, and a lower percentage of stage 1 were observed as an effect of the different exercise protocols compared with baseline. The other sleep parameters and core temperature did not change significantly after exercise. The data also show that physical exercise of moderate intensity (VT1) and lasting 30 min resulted in the greatest changes in sleep pattern.

Table 2. Polysomnographic Parameters at Baseline and for the Four Exercise Protocols Evaluated

Parameter	Baseline	VT1 30 min	VT1 60 min	Delta50 30 min	Delta50 60 min	p value
Total sleep time (min)	339.1 ± 54.9	363.2 ± 36.4	351.9 ± 49.6	359.2 ± 35.4	357.4 ± 45.1	0.283
Wake after sleep onset (min)	62.5 ± 36.5	26.0 ± 16.1 ^a	36.5 ± 26.8 ^a	29.0 ± 21.8 ^a	35.4 ± 25.8 ^a	0.003
Sleep onset latency (min)	10.0 ± 9.7	10.6 ± 10.7	8.9 ± 10.6	16.4 ± 20.6	14.0 ± 28.4	0.212
REM latency (min)	106.1 ± 44.8	78.1 ± 48.0 ^b	104.4 ± 44.5	96.9 ± 38.5	106.7 ± 45.6	0.047
Sleep efficiency (%)	82.2 ± 9.2	90.8 ± 5.0 ^a	88.4 ± 9.4 ^a	89.1 ± 6.3 ^a	88.2 ± 8.5 ^a	0.016
Stage 1 (%)	4.2 ± 2.0	2.6 ± 1.5 ^a	3.2 ± 1.8	2.8 ± 1.7 ^a	2.9 ± 1.2 ^a	0.046
Stage 2 (%)	57.8 ± 7.1	57.4 ± 6.4	61.0 ± 10.5	57.3 ± 9.1	60.5 ± 8.3	0.254
SWS (%)	18.6 ± 5.8	18.0 ± 5.2	17.1 ± 7.8	18.3 ± 7.4	18.0 ± 7.0	0.99
REM (%)	19.4 ± 4.9	22.3 ± 7.7	19.4 ± 8.3	21.4 ± 4.4	18.6 ± 6.4	0.187

Notes. Values are reported as mean ± standard deviation. SWS = slow-wave sleep.

^a $p \leq .05$ vs. baseline.

^b $p = .01$ vs. Delta50 60 min.

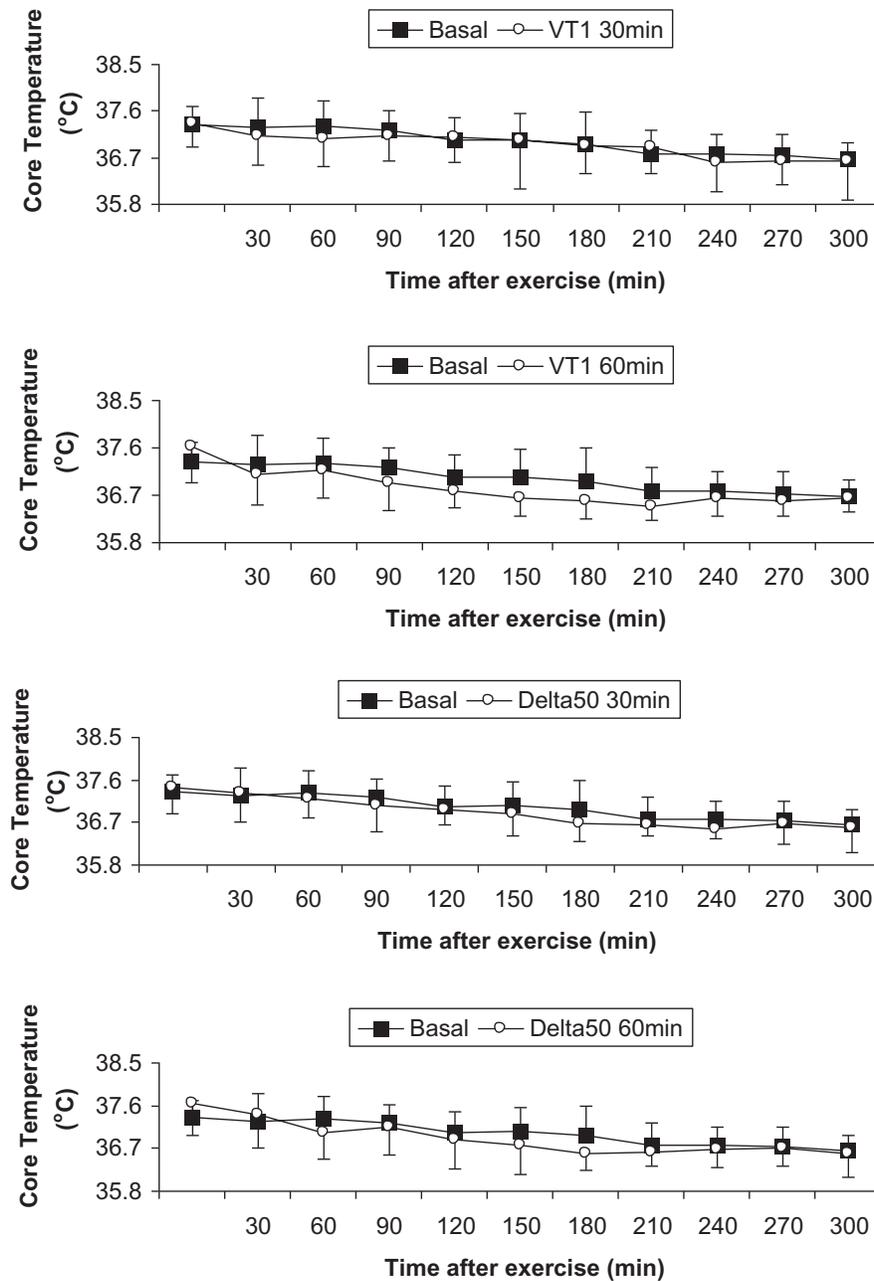


Figure 1. Data represent the mean \pm SD of temperature for every 30 min of recording of the 17 subjects assessed at basal conditions and after 4 different exercise intensities.

These findings contrast with sleep hygiene recommendations (Zarcone, 1994) and studies suggesting that physical exercise before bedtime should be avoided because it inhibits and disturbs sleep (Porter & Horne, 1981; Urponen, Vuori, Hasan, & Partinen, 1988). In addition, this study contradicts the experimental data of Youngstedt et al. (1997), which showed no significant improvement in sleep with physical exercise.

The participants in this study had not been engaged in systematic physical exercise for at least 6 months. They were young, healthy adults and thus achieved good to excellent functional capacity values observed by VO_{2peak} according to the classification published by the American College of Sports Medicine (Armstrong et al., 2006).

Although the most frequently observed effect of physical exercise is an increase in SWS (Hague, Gilbert, Burgess, Ferguson, & Dawson, 2003; Horne & Staff, 1983; Taylor, Rogers, & Driver, 1997), the present study showed no statistical differences in SWS or TST, regardless of the intensity and duration of exercise. Nevertheless, when we compare the intensities of exercise, that is, VT1, it was observed that the exercise of shorter duration (30 min) showed greater amount of TST (363.2 ± 36.4 min) than longer exercise duration performed at 60 min (351.9 ± 49.6 min; $p > .05$). This finding applies equally to the Delta 50 intensity. However, this trend is inconsistent with previous meta-analysis of Youngstedt et al. (1997), which found that the effects of physical exercise on sleep duration appear to be dose-dependent, that is, the greater the

duration of exercise, the greater the sleep benefit. For physical exercise lasting less than 1 h, the effects on sleep duration are insignificant (approximately 2 min); these effects increase with the increases at duration of physical exercise (approximately 11 min for 1–2 h of physical exercise and 15 min when lasting more than 2 h). Therefore, long-term physical exercise seems to promote the greatest benefit for sleep time.

Furthermore, endurance exercise results in a greater magnitude of SWS compared with mixed aerobic and anaerobic power, weight lifting, and a sedentary control group (Trinder, Paxton, Montgomery, & Fraser, 1985). Very intense long-term exercise can lead to an inadequate recovery period, preventing a reduction in body temperature, and may result in night-time insomnia (Driver & Taylor, 2000).

Physical exercise does not need to be intense to positively affect sleep (Youngstedt et al., 1997). Low intensity physical exercise has been associated with a reduction in time wake after sleep onset (approximately 16 min), while high intensity exercise has been correlated with an increase in time wake (approximately 4 min) (Youngstedt, 2005). Some studies have shown increased SWS after exercising to 50%–80% of VO_{2max} , which is considered a moderate intensity (Bunnell, Bevier, & Horvath, 1983; Horne & Moore, 1985; Horne & Staff, 1983; Paxton, Trinder, & Montgomery, 1983). In the study by Bunnell et al. (1983), physical exercise of moderate intensity ranged from 50%–70% of VO_{2max} in physically active individuals and lasted approximately 2 h and 30 min. However, some studies have reported no impairment in sleep after strenuous exercise for both sedentary and active individuals (O'Connor et al., 1998; Paxton et al., 1983; Youngstedt et al., 1999).

According to a meta-analysis by Driver and Taylor (2000), controversy remains regarding the influence of exercise intensity on sleep pattern, but physical exercise has been generally associated with increased TST and SWS, increased REM sleep latency, and a reduced percentage of REM sleep. Our findings on sleep efficiency corroborate evidence from previous studies (Driver & Taylor, 2000; Hague et al., 2003). Adrenergic influence on the duration of REM sleep was demonstrated in trained cyclists, suggesting a catecholamine imbalance in the pre-exercise phase (Netzer, Kristo, Steinle, Lehmann, & Strohl, 2001). Therefore, inhibition of REM sleep or delay in its onset would occur due to this imbalance between cholinergic and adrenergic substances and not specifically because of physical factors promoting the improvement or extension of non-REM sleep. The intensity, duration, and type of physical exercise have important influences on metabolic, cardiovascular, hemodynamic, endocrine, and thermoregulatory changes.

It is important to note that the participants in this study were good sleepers, which was confirmed by clinical evaluation by the PSQI and night PSG. Studies with subjects reporting poor sleep quality and sleep problems (Esteves, de Mello, Pradella-Hallinan, & Tufik, 2009; Passos et al., 2010; Passos, Tufik, Santana, Poyares, & Mello, 2007; Tanaka et al., 2001) as well as elderly participants (King, Oman, Brassington, Bliwise, & Haskell, 1997) have demonstrated significant effects on sleep pattern resulting from exercise. Youngstedt (2005) states that including only good sleepers in the experiment may not result in any significant changes because there is little room for improvement in the sleep pattern of the subjects, referred to as ceiling and floor effects. The effectiveness of the stimulus in promoting sleep should be proportional to the degree of sleep disturbance, that is, subjects with more severe sleep problems should have the greatest potential for improvement. Data

from our laboratory confirm this hypothesis because no statistical differences were found in studies with good sleepers subjected to aerobic or resistance exercise (Cavagnoli et al., 2010; Rossi et al., 2010).

In the present study, we found increases in core body temperature with the longer duration protocols (60 min). A mean elevation of 0.28°C was found in the VTI that returned to baseline levels 120 min later, and the Delta50 protocol promoted a mean elevation of 0.45°C, which returned to baseline within 30 min after completion of the effort. Neither of these protocols interfered with sleep induction. Core body temperature in the remaining protocols remained very close to baseline. We initially hypothesized that peripheral body temperature would increase proportionally to the intensity of exercise and would be important for the delay in sleep latency and TST reduction, but it did not occur. Core body temperature rapidly declined in the minutes immediately following exercise and did not affect these parameters. Therefore, experimental conditions that produce a temporary decoupling between temperature and the rhythm of sleep propensity suggest that these rhythms are independent and not causal (Lack & Lushington, 1996).

The main hypotheses about the effects of exercise on sleep were originally influenced by the theories of energy conservation and tissue regeneration or body restoration. Only since the 1980s have the effects of physical exercise been compared with the hypothesis of thermoregulation (Atkinson & Davenne, 2007). This thermoregulatory hypothesis is based on evidence linking the preoptic area of the anterior hypothalamus to sleep regulation and temperature downregulation (McGinty & Szymusiak, 1990).

The theories of energy conservation and body restoration are based on the homeostatic mechanisms regulating sleep, and both theories claim that TST and SWS quality increase with an increase in energy expenditure. The body restoration theory stems from the idea that high catabolic activity during wakefulness favors anabolic activity during sleep. This hypothesis is supported by the release of growth hormone in the SWS muscle, possibly stimulating anabolic pathways (Driver & Taylor, 2000). Thus, exercise would facilitate sleep by reducing the body's energy reserves, which in turn increases the need for sleep, especially SWS (Montgomery, Trinder, & Paxton, 1982).

The thermoregulatory hypothesis has been supported by highly relevant studies that reported that hyperthermia induced by exercise leads to an increase in the percentage of SWS, which is thought to be the best and deepest form of sleep (Horne & Moore, 1985; Horne & Staff, 1983). However, the overemphasis on the amount of SWS as an indicator of sleep quality has been widely criticized. This hypothesis is tenuous because REM sleep can be considered the deepest sleep form in terms of awakenings, and there is little evidence linking SWS with better results than REM sleep (Rechtschaffen, Bergmann, Gilliland, & Bauer, 1999). A study by O'Connor et al. (1998) showed that the temperature rise mediated by exercise performed 90 min before sleep did not change sleep measurements in moderately active individuals deprived of sleep.

Some studies have shown evidence suggesting a rapid decline in core temperature in the 20 min after low intensity ($\leq 50\% VO_{2peak}$) and short (≤ 1 h) exercise (Kelso, Herbert, Gwazdauskas, Goss, & Hess, 1984), returning to baseline levels within 30 min. In contrast, short duration exercise and moderate duration high intensity exercise ($\geq 60\% VO_{2peak}$) can result in elevated core body temperature for 1 to 3 h (Brooks, Hittelman, Faulkner, & Beyer, 1971). However, the body temperature increase after moderate intensity exercise ($60\% VO_{2peak}$) performed 90 min before sleep (O'Connor

et al., 1998) or vigorous intensity exercise (65%–75% of HRR) performed 30 min before bedtime (Youngstedt et al., 1999) did not significantly influence sleep variables.

Our data suggest that physical exercise before bedtime positively affects the sleep pattern of healthy, nonathlete, young adult males without sleep complaints. These changes include increased sleep efficiency percentage, increased REM sleep latency, less time awake after sleep onset, and decreased percentage of stage 1 sleep. Similarly, the body temperature increases, and metabolic changes induced by different intensities and durations of exercise did not negatively alter sleep parameters. It is important to emphasize the key role of physical exercise on sleep regulation as

an important nonpharmacological resource; however, more studies are needed to further investigate the effects of physical exercise on sleep as in other population groups (e.g., athletes, women, children) as well as in different chronotypes and other individual characteristics that not were contemplated in this study.

There are several limitations in this study that should be noted and considered in future studies. The sample size was relatively small, but the data showed statistical significance. In addition, the sample consisted of healthy, young male adults, nonathletes, without sleep complaints and, therefore, these findings cannot be generalized to the population as a whole.

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