

Clinical Study

Improvements in Chronic Primary Insomnia after Exercise Training Are Correlated with Changes in Metabolic and Hormonal Profile

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Objective. The objective of this study was to correlate metabolic and hormonal parameters before and after 8, 16, and 24 weeks (wk) of moderate aerobic training in individuals with chronic primary insomnia. **Method.** Four male and sixteen female volunteers (adults, sedentary, and healthy) performed exercise training for 24 weeks. Blood and Pittsburgh Sleep Quality Index (PSQI) was obtained at baseline, 8, 16, and 24 wk of training. **Results.** PSQI scores decreased after 8, 16, and 24 wk of training regarding baseline values. Indeed, total sleep time (TST) increased after 16 and 24 wk of exercise training regarding baseline values. The correlations were analyzed using the delta (Δ) values ($\Delta 1 = 8$ wk less baseline; $\Delta 2 = 16$ wk less baseline; $\Delta 3 = 24$ wk less baseline). We have observed a negative correlation for $\Delta 1$ between TST and cortisol, a positive correlation for $\Delta 3$ between TST and growth hormone, a negative correlation for $\Delta 1$ between TST and VLDL, a negative correlation for $\Delta 1$ between TST and triacylglycerols, and a negative correlation for $\Delta 1$ and $\Delta 2$ between TST and thyroid-stimulating hormone. **Conclusion.** The exercise training improved the sleep quality of patients with chronic primary insomnia.

1. Introduction

In modern society, sleep disorders are increasing every day around the world. Recently, a study with 4957 American adults determined that 40% of the participants had at least one sleep disorder [1]. In São Paulo, Brazil, a study observed that 33% of the society presented insomnia characteristics, and a higher incidence of insomnia was detected in women (40%) [2].

Chronic primary insomnia is a sleep disorder that is characterized by long-term difficulties with initiating and maintaining sleep, waking up too early, nonrestorative sleep, and daytime impairment, including fatigue, poor mood,

impaired concentration, and poor quality of life [3–6]. Budhiraja et al. 2011 [7] have shown that medical disorders such as hypertension and diabetes are associated with a higher prevalence of insomnia. The prevalence of insomnia increased as the number of medical disorders increased, suggesting an additive adverse effect of these medical conditions on insomnia [7]. Other studies have concluded that few hours of sleep are associated with overweight, obesity, metabolic alterations (insulin resistance, dyslipidemia, and increased activity of the HPA axis), and inflammatory diseases [8–11].

Physical exercise is used as a nonpharmacological treatment in many different diseases, including sleep disorders [12–15]. Recently, our group demonstrated that acute and

chronic aerobic exercise improves subjective and objective sleep in patients with chronic primary insomnia [14, 15]. However, the effects of chronic exercise on the metabolic and hormonal profile in patients with chronic primary insomnia are unknown. Therefore, the main propose of this study was to verify the association of metabolic and hormonal parameters before and after 8, 16, and 24 weeks (wk) of moderate aerobic exercise training in patients with chronic primary insomnia. Our hypothesis is that exercise training can promote improvements in sleep and as a consequence promote alterations in metabolic parameters evaluated.

2. Methods

Two hundred and sixty-seven people contacted the researchers by telephone or email with an interest in participating of the study. Of this total, 229 individuals were excluded for not meeting the inclusion criteria. Thirty-eight volunteers (29 women) passed the initial screening. However, before beginning the exercise training protocol, 3 men and 5 women withdrew from the study during the baseline period.

The exercise protocol began with a sample size of 30 volunteers. During the course of the study, 10 volunteers withdrew. Of these volunteers, 9 (30%) withdrew because of problems with the schedule of the program, which could not be changed during the study. As a result, the final sample size was 20. These 20 volunteers exhibited good adherence to the study protocol (>90%) in terms of attending the classes and spending greater than 75% of the exercise time within the prescribed workout intensity. In the present study, 80% of the volunteers were women with a mean age of 45.5 ± 8.39 years and a mean duration of insomnia of 10.71 ± 8.83 years, and all volunteers finished the study with more than 75% compliance.

The study was approved by the University Human Research Ethics Committee and conformed to the principles outlined in the Declaration of Helsinki. The volunteers were recruited through advertisements in newspapers, magazines, and radio shows.

Interested prospective volunteers contacted the laboratory and were initially screened through a phone interview. The initial inclusion criteria included being between 30 and 55 years old, having insomnia complaints for longer than 6 months, and having at least one complaint of daytime impairment resulting from insomnia (regarding mood, cognition, or perception of fatigue). The initial criteria for exclusion were the use of medication or psychotherapeutic drugs for insomnia or other psychiatric disorders, working the night shift, and exercising at least 1 day per week. Prospective volunteers who passed the phone interview were invited to the laboratory for further study and for orientation and screening. During the visit, prospects signed a written informed consent waiver approved by the Ethics Committee. Volunteers were excluded if their score on the Beck Depression Inventory exceeded 20 [16].

Further medical screening included establishing a clinical diagnosis of primary insomnia according to the DSM-IV [3] and ICSD [4] that was not associated with medical conditions or side effects from medications, and patients were excluded

if they had blood test results contraindicating participation in exercise training. In addition, the subjects were required to have normal ECG recordings at rest and on a graded treadmill while undergoing a VO_2 max test to volitional exhaustion, which collected expired air breath by breath and analyzed it using a gas analyzer (Quark PFT4; Rome, Italy). The VO_2 test also established each volunteer's ventilatory threshold (VT1) [17, 18], which was used for the prescription of moderate intensity exercise.

2.1. Exercise Protocol. The volunteers performed an incremental exercise on a treadmill (Life Fitness 9500 HR; USA). The initial running speed was 4 km/h (for a 3-minute warm-up), with increments of 0.5 km/h every minute up to voluntary exhaustion. The ventilatory variables, minute ventilation (VE), oxygen consumption (VO_2), and carbon dioxide production (VCO_2) were measured. To analyze the data, we used the means from 20-second intervals and considered the highest VO_2 obtained in the last interval of the test to be the peak oxygen consumption (VO_{2peak}). The intensity of exercise was based on the first ventilatory threshold (VT₁) [17, 18], which was considered moderate intensity, and the training session for the treadmill lasted 50 continuous minutes, which was performed in the morning or late afternoon.

2.2. Design and Procedures

2.2.1. Questionnaire and Blood Samples. The patients were evaluated at baseline and after 8, 16, and 24 wk of training exercise. They were instructed to arrive in the laboratory at 9 AM, when venous blood was drawn after the Pittsburgh Sleep Quality Index questionnaire was completed [19].

2.3. Measurements

2.3.1. Pittsburgh Sleep Quality Index (PSQI). The PSQI questionnaire was administered before training and 24 h after the last exercise session. The PSQI is a validated instrument that measures some domains. We used sleep latency (SL) (items 2 and 5a), total sleep time (TST) (item 4), habitual sleep efficiency SE (ratio of item 4 and items 1, 3), and total score of quality of life over the past month. The total score can range from 0 to 21, and a total score of 5 or greater indicates poor sleep quality [19].

2.3.2. Blood Collection and Biochemical Tests. Blood samples were collected (20 mL) in sterile tubes containing heparin from an antecubital vein at the beginning of the study (baseline) and after 8, 16, and 24 wk of exercise training. The blood samples were collected 24 h after the last exercise session after fasting for 12 h. Blood samples were centrifuged at $650 \times g$ for 15 minutes. Plasma samples were stored at $-80^\circ C$ and analyzed within 1 week. From each sample, the concentrations of lactate, total cholesterol, high-density lipoprotein (HDL), and triacylglycerols (TG) in the plasma were assessed using commercial kits (Labtest; São Paulo, Brazil). Low-density lipoprotein (LDL) and very low-density

TABLE 1: Anthropometric and training characteristics in patients with chronic primary insomnia after 8, 16, and 24 wk of exercise training.

Variables	Baseline	After 8 wk	After 16 wk	After 24 wk	<i>P</i>
Age	45.5 ± 8.39	—	—	—	—
BMI (Kg/m ²)	24.80 ± 4.70	24.88 ± 4.46	24.68 ± 4.39	24.53 ± 4.56	NS
% Fat	31.31 ± 9.91	32.29 ± 8.00	32.3 ± 8.07	32.77 ± 8.55	NS
VO _{2peak} (mL/kg/min)	27.82 ± 5.17	29.17 ± 5.23	30.32 ± 5.38	31.14 ± 6.73	NS
VO _{2peak} (L/min)	1747.00 ± 461.20	1825.00 ± 436.50	1901.00 ± 469.10	1927.00 ± 436.30	NS
Speed VT ₁ (km/h)	5.32 ± 0.79	5.925 ± 0.5684	6.125 ± 0.5821	6.36 ± 0.62	0.001

One-way ANOVA, significant results with $P \leq 0.05$; data are expressed as the mean \pm SD. VO_{2peak}: peak oxygen consumption; VT₁: first ventilatory threshold.

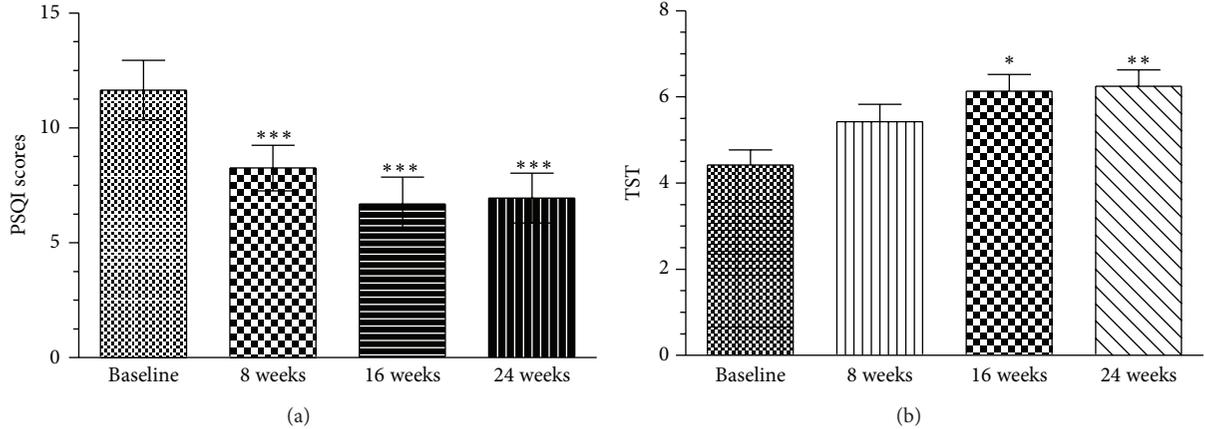


FIGURE 1: PSQI scores after 8, 16 and 24 wk of training when compared with the baseline values (** $P < 0.001$). The total sleep time (TST) after 8 wk, 16 wk ($*P < 0.05$) and 24 wk (** $P < 0.01$) of exercise training when compared with the baseline values. Repeated-measures ANOVA, significant results, $P < 0.05$; data are expressed as mean \pm SD.

lipoprotein (VLDL) were calculated according to Friedewald et al. [20]. Fasting insulin concentrations were determined using commercially available ELISA kits from Millipore (Billerica, MA), and glucose concentrations were determined using an enzymatic method (Labtest). The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the determined values of glucose and insulin. Plasma pituitary hormone (TSH, GH, and ACTH), adrenal hormone (cortisol), thyroid hormone triiodothyronine (T3 and T3 free), thyroxin (T4 and T4 free), and sexual hormone (FSH, Prolactin, progesterone, estradiol, testosterone free, and prolactin) levels were measured using RIA with commercial kits (Orion Diagnostica).

2.4. Statistical Analyses. Values are represented as the mean \pm standard deviation (SD). Nonparametric distribution dependent variables were transformed into *Z* scores to normalize the data. The differences in the plasma and sleep parameters at the measured time points (before and after 8, 16, and 24 wk of exercise training) were evaluated using a variance analysis for repeated measures (ANOVA), followed by the Duncan test. The Pearson correlation was used and the data were analyzed using delta values (Δ) ($\Delta 1 = 8$ wk less baseline; $\Delta 2 = 16$ wk less baseline; $\Delta 3 = 24$ wk less baseline). The level of significance was established as $P < 0.05$ (the software SSPS 19 was used for all analyses).

3. Results

The individual physiological and anthropometric characteristics of the volunteers before the exercise regimen and after 8, 16, and 24 wk of exercise training are presented in Table 1. In addition, Table 1 shows that 8, 16, and 24 wk of moderate aerobic training were effective at improving aerobic capacity, which was demonstrated by an increase in speed ($P < 0.0001$). VO₂ values compared with values before training showed no significant difference.

We observed decreased PSQI scores after 8, 16, and 24 wk of training when compared with the baseline values ($P < 0.001$). The total sleep time (TST) increased after 16 wk ($P < 0.05$) and 24 wk ($P < 0.01$) of exercise training when compared with the baseline values. Sleep efficiency and latency showed no differences between the measured time points (see Figure 1).

In addition, as shown in Table 2, we observed a negative correlation for the value of $\Delta 1$ between TST and cortisol ($r = -0.504$, $P < 0.05$), a positive correlation for the value of $\Delta 3$ between TST and growth hormone ($r = 0.597$, $P < 0.01$), a negative correlation for the value of $\Delta 1$ between TST and VLDL ($r = -0.613$, $P < 0.01$), a negative correlation for the value of $\Delta 1$ between TST and triacylglycerols ($r = -0.530$, $P < 0.05$), and a negative correlations for the values of $\Delta 1$ and 2 between TST and thyroid-stimulating hormone ($\Delta 1 : r = -0.685$, $P < 0.001$ and $\Delta 2 : r = -0.612$, $P < 0.01$).

TABLE 2: Correlation between the variables PSQI-TST and cortisol, GH, VLDL-C, TAG, and TSH of patients with chronic primary insomnia.

	Delta 1	Delta 2	Delta 3
Cortisol	$R = -0.504^*$	$R = 0.084$	$R = -0.110$
GH	$R = 0.063$	$R = 0.328$	$R = 0.597^{**}$
VLDL-C	$R = -0.613^{**}$	$R = -0.241$	$R = -0.161$
Triacylglycerols	$R = -0.530^*$	$R = -0.262$	$R = -0.171$
TSH	$R = -0.685^\#$	$R = -0.612^{**}$	$R = -0.390$

* $P < 0.05$; ** $P < 0.01$; # $P < 0.001$.

4. Discussion

The results indicate that aerobic exercise training improved sleep patterns in patients with chronic primary insomnia. We have observed decreased PSQI scores after 8, 16, and 24 wk of training when compared with the baseline values. However, there were no changes in hormonal parameters compared to pretraining period, although we have found a correlation between changes in subjective sleep parameters and some metabolic parameters.

The hypothesis of this study is that exercise is a cheap and simple activity that is capable of treating variables jointly promoting an improvement in sleep [12]. Our group [14, 15] and Reid et al. [21] have already demonstrated the efficiency of moderate aerobic exercise in the treatment of patients with chronic primary insomnia. The results of our study showed that a program of 24 wk of moderate aerobic exercise is effective in improving the self-assessment of sleep quality in patients with chronic primary insomnia. We also have observed a significant increase in TST at sixteenth and twenty-fourth weeks of training.

In a recent study, our group evaluated the effect of aerobic exercise on the treatment of chronic insomnia. The conclusion was that exercise training is effective at decreasing sleep complaints and insomnia [14]. Aerobic exercise has been more extensively studied, and its effects are similar to those observed after hypnotic medication use. There is additional documented evidence on the antidepressant and anxiolytic effects of exercise [22].

In addition to the central adaptations, physical training promotes peripheral adjustments that are related to metabolic alterations. The major data were found after 8 wk of training, despite our exercise protocol being adjusted by the first ventilatory threshold conforms recommended by ACSM [23]; however, this specific population may require training-strategy modifications to ensure consistent modifications of metabolism.

Vgontzas et al. [10] hypothesized that chronic primary insomnia is associated with an increase in ACTH and cortisol plasma levels [24]. Other studies have shown that nocturnal cortisol levels were significantly increased in patients with chronic primary insomnia [24, 25]. In addition, chronic primary insomnia patients presented a significant correlation between sleep parameters and the first 4 hours of nocturnal cortisol secretion. This result indicates that changes in the HPA axis in insomnia can reflect a pathophysiological

mechanism for chronic insomnia in which both functions of the HPA axis are disturbed. The increase in HPA axis activity promotes the fragmentation of sleep; however, sleep fragmentation increases cortisol levels, which exacerbates the cycle [24]. Our results demonstrate a negative correlation between the TST and free cortisol levels. These results may be due to the benefits induced by the exercise training protocol in chronic primary insomnia patients.

Sleep disturbance has a stimulatory effect on the HPA axis and a suppressive effect on the GH axis [26]. van Cauter et al. [27], based on measurements of plasma GH levels, have challenged the concept that GH secretion is dependent on sleep and not modulated by circadian rhythmicity. The amount of GH secreted in slow wave-associated pulses was correlated with the amount of slow waves occurring during the pulse, even when sleep-onset pulses were not considered [27]. In the present study, we have found a positive correlation between GH and TST after 24 wk training compared to baseline. Exercise is a robust physiological stimulator of the pituitary secretion of growth hormone (GH), and within approximately 15 min after the onset of exercise, plasma GH begins to increase. Perhaps a program of physical exercise promotes a decrease in the activity of the HPA axis, leading to an improvement in sleep and consequently an increase in production of GH.

The thyroid hormones thyroxine (T4) and triiodothyronine (T3) exert physiologic effects in all tissues affecting oxygen consumption and protein, carbohydrate, lipid, hormone, growth factor, and vitamin metabolism [28]. The release of T4 and T3 by the thyroid is controlled by the activity of the hypothalamic thyrotropin-releasing hormone (TRH) and pituitary thyroid-stimulating hormone (TSH) cascade. Sleep loss can also affect the function of the human hypothalamus-pituitary-thyroid axis. The effects of sleep deprivation in humans are associated with increased TSH, T4, and T3, and human sleep is believed to have an acute inhibitory effect on overnight TSH secretion [29]. In the present study, a negative correlation was observed between TST and TSH after 8 and 16 wk of physical exercise. The thyroid gland is involved in changes in body temperature related to the stimulus of exercise, which can promote improvements in sleep, especially in individuals with a sleep disorder. Moreover, changes in energy expenditure can promote changes in lipid metabolism, contributing to an improved metabolic profile in these individuals and a consequent improvement in sleep [30].

Few works in the literature have evaluated whether there is an association between sleep duration and dyslipidemia [31, 32]. Sabanayagam and Shankar [31] in a cross-sectional study of 16652 participants in the 2008 National Health Interview Survey have found that sleep duration ≤ 5 h was positively associated with hypercholesterolaemia in women after adjusting for potential confounders and mediators including physical activity, psychological distress, body mass index, diabetes mellitus, and hypertension [31]. In the present study, we have found a negative correlation between TST and triglycerides/VLDL after 8 wk of exercise protocol. Our group has shown that a protocol of aerobic exercise can promote changes in lipid profile mainly due to an increase in energy

expenditure and consequently increased lipolysis caused by an aerobic training protocol [33].

We have concluded that aerobic exercise training can promote both central and peripheral adaptations that lead to an improved sleep pattern in patients with chronic primary insomnia. Physical exercise can be a simple and inexpensive strategy for the treatment of insomnia, and the present study showed improvements in the sleep quality of patients with chronic primary insomnia after exercise training but without alterations in hormonal or metabolic parameters. This population may require training-strategy modifications to ensure consistent metabolic modifications.

Conflict of Interests

The authors declare that they have no conflict of interests.

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References

- [1] S. M. W. Rajaratnam, L. K. Barger, S. W. Lockley et al., "Sleep disorders, health, and safety in police officers," *Journal of the American Medical Association*, vol. 306, no. 23, pp. 2567–2578, 2011.
- [2] L. R. A. Bittencourt, R. Santos-Silva, J. A. Taddei, M. L. Andersen, M. T. de Mello, and S. Tufik, "Sleep complaints in the adult Brazilian population: a national survey based on screening questions," *Journal of Clinical Sleep Medicine*, vol. 5, no. 5, pp. 459–463, 2009.
- [3] A. P. Association, *Diagnostic and Statistical of Mental Disorders (DSM-IV)*, American Psychiatric Press, Washington, DC, USA, 1994.
- [4] Medicine AAoS, *The International Classification of Sleep Disorders (ICSD): Diagnostic and Coding Manual*, Diagnostpic Classification Steering Committee, Westchester, Ill, USA, 2005.
- [5] G. E. Simon and M. Vonkorff, "Prevalence, burden, and treatment of insomnia in primary care," *The American Journal of Psychiatry*, vol. 154, no. 10, pp. 1417–1423, 1997.
- [6] M. O. Summers, M. I. Crisostomo, and E. J. Stepanski, "Recent development in the classification, evaluation, and treatment of insomnia," *Chest*, vol. 130, no. 1, pp. 276–286, 2006.
- [7] R. Budhiraja, T. Roth, D. W. Hudgel, P. Budhiraja, and C. L. Drake, "Prevalence and polysomnographic correlates of insomnia comorbid with medical disorders," *Sleep*, vol. 34, no. 7, pp. 859–867, 2011.
- [8] H. R. Colten and B. M. Altevogt, *Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem*, 2006.
- [9] M. R. Irwin, M. Wang, D. Ribeiro et al., "Sleep loss activates cellular inflammatory signaling," *Biological Psychiatry*, vol. 64, no. 6, pp. 538–540, 2008.
- [10] A. N. Vgontzas, E. O. Bixler, H.-M. Lin et al., "Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications," *Journal of Clinical Endocrinology and Metabolism*, vol. 86, no. 8, pp. 3787–3794, 2001.
- [11] A. N. Vgontzas, D. A. Papanicolaou, E. O. Bixler et al., "Circadian interleukin-6 secretion and quantity and depth of sleep," *Journal of Clinical Endocrinology and Metabolism*, vol. 84, no. 8, pp. 2603–2607, 1999.
- [12] E. S. Alves, F. S. Lira, R. V. T. Santos, S. Tufik, and M. T. de Mello, "Obesity, diabetes and OSAS induce of sleep disorders: exercise as therapy," *Lipids in Health and Disease*, vol. 10, article 148, 2011.
- [13] A. M. Esteves, M. T. de Mello, M. Pradella-Hallinan, and S. Tufik, "Effect of acute and chronic physical exercise on patients with periodic leg movements," *Medicine and Science in Sports and Exercise*, vol. 41, no. 1, pp. 237–242, 2009.
- [14] G. S. Passos, D. Poyares, M. G. Santana et al., "Effects of moderate aerobic exercise training on chronic primary insomnia," *Sleep Medicine*, vol. 12, no. 10, pp. 1018–1027, 2011.
- [15] G. S. Passos, D. Poyares, M. G. Santana, S. A. Garbuio, S. Tufik, and M. T. de Mello, "Effect of acute physical exercise on patients with chronic primary insomnia," *Journal of Clinical Sleep Medicine*, vol. 6, no. 3, pp. 270–275, 2010.
- [16] A. T. Beck, C. H. Ward, M. Mendelson, J. Mock, and J. Erbaugh, "An inventory for measuring depression," *Archives of General Psychiatry*, vol. 4, pp. 561–571, 1961.
- [17] W. L. Beaver, K. Wasserman, and B. J. Whipp, "A new method for detecting anaerobic threshold by gas exchange," *Journal of Applied Physiology*, vol. 60, no. 6, pp. 2020–2027, 1986.
- [18] L. Goldberg, D. L. Elliot, and K. S. Kuehl, "Assessment of exercise intensity formulas by use of ventilatory threshold," *Chest*, vol. 94, no. 1, pp. 95–98, 1988.
- [19] D. J. Buysse, C. F. Reynolds III, T. H. Monk, S. R. Berman, and D. J. Kupfer, "The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research," *Psychiatry Research*, vol. 28, no. 2, pp. 193–213, 1989.
- [20] W. T. Friedewald, R. I. Levy, and D. S. Fredrickson, "Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge," *Clinical Chemistry*, vol. 18, no. 6, pp. 499–502, 1972.
- [21] K. J. Reid, K. G. Baron, B. Lu, E. Naylor, L. Wolfe, and P. C. Zee, "Aerobic exercise improves self-reported sleep and quality of life in older adults with insomnia," *Sleep Medicine*, vol. 11, no. 9, pp. 934–940, 2010.
- [22] P. D. Nowell and D. J. Buysse, "Treatment of insomnia in patients with mood disorders," *Depression and Anxiety*, vol. 14, no. 1, pp. 7–18, 2001.
- [23] American College of Sports Medicine Position Stand, "The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults," *Medicine & Science in Sports & Exercise*, vol. 30, pp. 975–991, 1998.
- [24] A. N. Vgontzas, G. Mastorakos, E. O. Bixler, A. Kales, P. W. Gold, and G. P. Chrousos, "Sleep deprivation effects on the activity of the hypothalamic-pituitary-adrenal and growth axes: potential clinical implications," *Clinical Endocrinology*, vol. 51, no. 2, pp. 205–215, 1999.
- [25] E. Spath-Schwalbe, M. Gofferje, W. Kern, J. Born, and H. L. Fehm, "Sleep disruption alters nocturnal ACTH and cortisol secretory patterns," *Biological Psychiatry*, vol. 29, no. 6, pp. 575–584, 1991.

- [26] M. Duclos, M. Guinot, and Y. L. Bouc, "Cortisol and GH: odd and controversial ideas," *Applied Physiology, Nutrition and Metabolism*, vol. 32, no. 5, pp. 895–903, 2007.
- [27] E. van Cauter, F. Latta, A. Nedeltcheva et al., "Reciprocal interactions between the GH axis and sleep," *Growth Hormone and IGF Research*, vol. 14, supplement A, pp. S10–S17, 2004.
- [28] L. Klingenberg, A. Sjödin, U. Holmbäck, A. Astrup, and J.-P. Chaput, "Short sleep duration and its association with energy metabolism," *Obesity Reviews*, vol. 13, pp. 565–577, 2012.
- [29] L. Kessler, A. Nedeltcheva, J. Imperial, and P. D. Penev, "Changes in serum TSH and free T4 during human sleep restriction," *Sleep*, vol. 33, no. 8, pp. 1115–1118, 2010.
- [30] M. L. Andersen and S. Tufik, "Is thyroid screening of sleep clinic patients essential?" *Sleep Medicine*, vol. 13, no. 10, pp. 1215–1216, 2012.
- [31] C. Sabanayagam and A. Shankar, "Sleep duration and hypercholesterolaemia: results from the National Health Interview Survey 2008," *Sleep Medicine*, vol. 13, no. 2, pp. 145–150, 2012.
- [32] I. Soreca, M. L. Wallace, E. Frank, B. P. Hasler, J. C. Levenson, and D. J. Kupfer, "Sleep duration is associated with dyslipidemia in patients with bipolar disorder in clinical remission," *Journal of Affective Disorders*, vol. 141, pp. 484–487, 2012.
- [33] F. S. Lira, G. D. Pimentel, R. V. T. Santos et al., "Exercise training improves sleep pattern and metabolic profile in elderly people in a time-dependent manner," *Lipids in Health and Disease*, vol. 10, article 113, pp. 1–6, 2011.



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