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Sleep depth and continuity before and after chronic exercise in older men: Electrophysiological evidence

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HIGHLIGHTS

- Older men showed more slow-wave sleep on active days following training.
- Wake time and REM onset latency were both reduced following exercise.
- Sleep continuity was improved due to reduced wake time with exercise.
- These effects of exercise on sleep, although modest, oppose those of aging.

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ABSTRACT

During later life sleep depth (slow-wave sleep, SWS) and maintenance exhibit deleterious changes, with possible negative effects on daytime function. This study assessed the effect of chronic, supervised exercise on sleep using laboratory-based polysomnography (PSG) and repeated measures in older adults. Thirteen men aged 64 ± 3 served as their own controls and had their sleep measured for a total of 6 nights: 3 before and 3 after the 16-week training intervention. Each sequence involved 1 familiarization trial followed by 2 experimental nights (exercise night; nonexercise night) measured using 13-channel PSG (combined electroencephalography, electromyography, and electro-oculography). The exercise challenges consisted of inclined treadmill brisk walking (60 min, 68–69% $\dot{V}O_2$ peak). The intervention successfully improved some parameters of aerobic fitness, i.e. ventilatory thresholds 1 and 2 ($P < 0.05$). Acute exercise triggered increases in circulating free fatty acids and lactate levels both at baseline and after the intervention ($P < 0.05$). Noteworthy, acute exercise following training resulted in a 71% increase in SWS during subsequent sleep in comparison with the nonexercise condition before training, respectively 2.4% and 1.4% ($P < 0.05$). Following training, acute exercise reduced total wake time by 30% and REM onset latency by 14% ($P < 0.05$). Acute exercise improved sleep continuity by decreasing total wake time. These results show that aerobic training could increase sleep depth and continuity, during active days, in elderly men. In habitual exercisers, these effects of aerobic exercise on sleep, although modest, might counteract those resulting from aging.

1. Introduction

The reduced capacity to initiate and maintain sleep with aging causes a progressive decrease in sleep quality. In fact, the prevalence of sleep disorders is more elevated in seniors [1] and, despite spending more time in bed, older people obtain less (and report more complaints about) sleep [2]. The most consistent age-related changes include an increased fragmentation of sleep and losses in slow-wave sleep (SWS, stages 3 + 4 or deep sleep) [1], the latter being known for its critical restorative function [3]. Both the amplitude (75 μV+) and frequency (0.5–2.0 Hz) of delta waves are reduced [2], decreasing the electroencephalographic (EEG) power spectrum, the so-called 'flattening of the EEG' in the aged. Deleterious clinical outcomes might ensue, such as excessive daytime sleepiness, propensity to fall asleep at the wheel, mood impairments, or metabolic syndrome. On the other hand, the use of sleeping pills in seniors is complicated by possible interactions with other drugs and low tolerance to side effects and is not recommended for long-term use because of its association with excess mortality [4]. Hence, non-pharmacological strategies aimed at promoting sleep are especially indicated in older individuals.

While epidemiological surveys have unveiled that poor exercise habits might contribute to incident insomnia in later life [5], exercise

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has proven to offer a number of positive effects on sleep, including total sleep time (TST), sleep onset latency, SWS, sleep efficiency, sleep quality, self-rated time to fall asleep, and nocturia [6–8]. However, many studies have relied on questionnaires. Although self-reported sleep ratings are somewhat costless and allow the appraisal of a number of variables, their subjectiveness might be misleading, i.e. subject to bias in report and/or recall. Prior studies that used polysomnography (PSG) to investigate the effect of exercise training on sleep in seniors have been limited in number. Among these, one could find a study involving subjects presenting moderate sleep complaints [9] and two reports that considered as 'older' individuals who were actually in their early forties [10,11]. However, a home-based PSG study revealed that aerobically trained older men had more SWS than untrained controls, irrespective of whether sleep followed an active or inactive day [12]. However, because of the cross-sectional nature of this study, it cannot be ruled out that genetic endowments, aerobic training or other factors were involved. For example, good sleepers might be more prone to adhere to exercise training, which could reciprocally cause benefits to their sleep.

In keeping with the aforementioned studies, one could note that none used controlled, laboratory-based PSG combined with an exercise intervention to examine the effect of aerobic training on sleep in older adults. Therefore, the current study aimed to investigate the effects of aerobic exercise training on sleep depth and continuity in older men.

2. Methods

2.1. Subjects

Thirteen community-dwelling Caucasian men aged 64 ± 3, range 57–70 yrs were recruited using local advertisement. Subjects had no orthopedic limitations, were not engaged in regular exercise training, and did not take any medication acting on sleep or serotonergic tone, e.g. tricyclic antidepressants, antipsychotics, or monoamine oxidase inhibitors during the year prior to the study. None had diabetes mellitus, obesity or smoked. In addition, the following exclusion criteria were applied on PSG data of the first night: leg movement index > 15 h^{-1} causing arousals or wake [13]; more than 10 h^{-1} episodes of sleep apnea and/or hypopnea (respectively >80% and >50% reduction of airflow mean amplitude) causing arousals or wake; professional evening or night activities; regular nap habits (>30 min); or a Pittsburgh Sleep Quality Index > 5. The Human Research Ethics Committee of the University Institute of Geriatrics of Sherbrooke approved this study and subjects were informed about the risks and benefits of the study before providing written consent. One subject was excluded from analyses on the basis of the Obstructive Sleep Apnea–Hypopnea Index criterion.

2.2. General study design

Volunteers were screened for eligibility by a phone interview, then eligible subjects were invited for further study explanations and written consent. At first, subjects underwent a maximal, pretesting cardiovascular examination consisting of a graded cardiopulmonary test to volitional exhaustion which aimed to assess ventilatory threshold, respiratory compensation threshold, maximal heart rate and peak O_2 consumption ($\dot{V}O_2$ peak). Criteria for determination of $\dot{V}O_2$ peak were attainment of maximal age-predicted heart rate, no further increase in $\dot{V}O_2$ despite increased workload, or subject's desire to stop. Then, subjects underwent a body composition scan, after which sleep was recorded at the laboratory over the course of 3 consecutive nights: 1) familiarization night, 2) nonexercise sedentary trial (SED), and 3) exercise trial (EXR). Regarding pre- and post-training experimental exercise challenges, subjects first warmed-up for 5–10 min, and then exercise at a moderate intensity for 1 h. This was immediately followed by a self-controlled recovery period (~5 min). During both exercise challenges, blood sampling analyses were conducted (before, during, and after exercise) in the fasted state to confirm whether exercise elicited a physiological challenge at baseline and following training. Hence, free fatty acids levels were taken as an index of availability of circulating energy substrates, whereas lactate served as an index of 'anaerobic' glycolysis [14]. The aforementioned tests were repeated following training for comparison purposes.

2.3. Maximal cardiorespiratory testing

Subjects got acquainted with cardiopulmonary testing before their participation in the study. The Physical Activity Readiness Questionnaire was first filled, and then subjects engaged in a maximal, modified Balke treadmill test in which blood pressure and electrocardiogram were interpreted by a physician at each exercise level. Oxygen uptake was measured from gas exchange every 5 s using an automated Oxycon Pro System (Jaeger; Würzburg, Germany) calibrated with reference gases before each test. $\dot{V}O_2$ peak was defined as the highest $\dot{V}O_2$ value observed in the last minute of the test.

2.4. Threshold determination

The ventilatory threshold was identified using the inflection point in the ventilatory equivalent for O_2 vs. $\dot{V}O_2$ curve with no concomitant rise in $\dot{V}E/\dot{V}CO_2$ [15]; respiratory compensation was identified using the loss of linearity of the ventilatory equivalent for CO_2 vs. $\dot{V}O_2$ curve [16]. Training-induced changes in the ventilatory- and respiratory compensation thresholds served as indices of improvements in cardiorespiratory fitness.

2.5. Body composition

Fat mass and lean body mass were determined using dual-energy X-ray absorptiometry (DXA Prodigy; Lunar Corp., Madison, WI, USA). The coefficients of variation for repeated determination of fat mass and fat-free mass in a subgroup of 10 individuals were 4.7% and 1.1%, respectively.

2.6. Experimental exercise sessions

The pre- and post-training exercise challenges consisted in 1 h of steady-state, incline treadmill brisk walking (~68–69% $\dot{V}O_2$ peak), followed by 30 min of rest. The target zone (i.e. reached in ~5–10 min). Exercise was performed before noon after a 3-h fast or more. Practically, subjects brisk walked at ~6 km h^{-1} with the grade of the treadmill adjusted between 4 and 12% depending on fitness.

2.7. Training program

Aerobic exercise sessions were supervised and held thrice weekly (non-consecutive days) for 16 wks with no interruption. Missed sessions were re-scheduled when possible. Exercise consisted of 45 min of brisk walking at ~6 km h^{-1} on an inclined treadmill adjusted to individual fitness. Exercise intensity was monitored using a telemetric heart rate monitor (Polar Electro Canada Inc., Lachine, QC, Canada) using heart rate at ventilatory threshold (lower bound) and respiratory compensation threshold (upper bound) obtained during the maximal cardiopulmonary test. Five to 10 min was allowed to warm-up and cool-down (self-paced), and training intensity was progressively increased over the first training sessions.

2.8. Blood analyses

Whole blood was immediately centrifuged (3000 rpm × 15 min) and stored at −82 °C. For analysis of lactate (lactic dehydrogenase