




Effects of resistance training on obstructive sleep apnea severity: a randomized controlled trial[☆]

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ABSTRACT

Meta-analyses have demonstrated that physical exercise is associated with a reduction in the severity of obstructive sleep apnea (OSA). However, whether resistance training (RT) has an impact on OSA severity remains less well established. In addition, the potential underlying mechanisms associated with improvements in OSA after resistance training also remain poorly understood. The primary objective of this study was to evaluate the impact of an eight-week RT program on OSA severity. A secondary aim was to investigate potential mechanisms underlying improvements in OSA following RT. Randomized controlled trial involving 25 adults with moderate to severe OSA [male: 52%; age: 55.4 ± 8.3 years; BMI: 36.3 ± 7.3 kg/m²; apnea-hypopnea index (AHI): 50.5 ± 25.5 events/hour], allocated to a RT group (RT; 3 weekly sessions for 8 weeks) or a control group (CG; stretching exercises). Nocturnal polysomnography, respiratory muscle strength, rostral fluid shift, and body composition assessments were performed before and after the intervention. Significant group × time interactions were found for AHI (RT: Δ -21.0 ± 6.1 vs. CG: 3.5 ± 3.8 e/h; p = 0.015), NREM-AHI (RT: Δ -20.5 ± 6.2 vs. CG: 3.9 ± 3.9 e/h; p = 0.022), and arousals (RT: Δ -7.9 ± 4.8 vs. CG: 7.4 ± 5.1; p = 0.028). In RT group, a correlation was verified between changes in AHI and waist circumference (r = 0.643, p = 0.018). An eight-week RT reduces OSA severity and the number of micro-arousals. A reduction in waist circumference was associated with an improvement in OSA severity.

1. Introduction

Obstructive sleep apnea (OSA) is a sleep disorder characterized by recurrent episodes of partial (hypopnea) and/or total (apnea) obstruction of airflow in the upper airways during sleep, associated with a decrease in oxygen saturation [1,2]. OSA is associated with several comorbid conditions, such as hypertension, metabolic syndrome,

cardiovascular disease [2,3], and obesity [4]. Furthermore, factors such as physical inactivity [5], muscle weakness [6], and changes in body fluid distribution [7] also contribute to the worsening of the clinical condition.

Although continuous positive airway pressure (CPAP) is the standard treatment for moderate to severe OSA [2] up to 60% of patients have poor adherence, using this method for less than 4 h per night [8], which reinforces the need for alternative treatments [9]. In this context,

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Abbreviation list:

- AASM – American Academy of Sleep Medicine
- AHI – Apnea-Hypopnea Index
- BMI – Body Mass Index
- CG – Control Group
- CONSORT – Consolidated Standards of Reporting Trials
- CPAP – Continuous Positive Airway Pressure
- MEP – Maximum Expiratory Pressure
- MIP – Maximum Inspiratory Pressure
- NREM – Non-Rapid Eye Movement
- ODI – Oxygen Desaturation Index
- OMNI-RES – Omni Resistance Exercise Scale
- OSA – Obstructive Sleep Apnea
- PSG – Polysomnography
- REM – Rapid Eye Movement
- RFS – Rostral Fluid Shift
- RT – Resistance Training
- SD – Standard Deviation
- TST – Total Sleep Time

physical exercise has been proposed as a promising non-pharmacological intervention [9,10]. Previous meta-analyses suggested that both aerobic exercises alone and with resistance training (RT) reduce the apnea-hypopnea index (AHI) [11–14].

However, the impact of RT alone on this population remains unclear. To date, only two studies have analyzed the effects of RT alone on OSA severity. One of them reported a mean reduction of 3.6 events per hour in the AHI after 12 weeks of intervention [15] whereas in another study, a 12-week RT program promoted a reduction in the AHI of 9.45 events per hour, in elderly individuals with OSA [16]. Nevertheless, although RT is frequently recommended to improve sleep in diverse clinical populations [17,18] its efficacy in individuals with OSA remains uncertain. Notably, the results are limited to older adults, which limits the generalizability of the results to younger populations.

Reductions in rostral fluid shift [7,19], fat loss [20] and increases in respiratory muscle strength have been described as potential mechanisms underlying improvements in OSA following a RT program [6,21]. While the former is related with reductions in fluids accumulated in the lower limbs throughout the day that are redistributed to the cervical region during sleep, the latter is suggested that RT significantly increase respiratory strength between 32% and 34% [22] which is associated with attenuating sleep-related upper airway dysfunction in patients with OSA [6]. Despite this, no study has demonstrated the effect of RT on the OSA severity regarding these underlying mechanisms involved in the OSA improvement.

Thus, the primary objective of this study was to evaluate the impact of an eight-week RT on OSA severity. A secondary aim was to investigate potential mechanisms underlying improvements in OSA following RT.

2. Methods

2.1. Study design

This was a randomized clinical trial that followed the Consolidated Standards of Reporting Trials (CONSORT) guidelines [23]. Patients were randomly assigned to two groups in a 1:1 ratio: the RT group and the control group (CG), with pre- and post-intervention assessments. The study was approved by the Research Ethics Committee (6.989.235). The clinical trial was registered with the Brazilian Clinical Trials Registry (REBEC) under number (RBR-2rg97kz). Fig. 1 depicts the study's experimental design.

2.2. Recruitment and screening

Participants were recruited from the Sleep and Heart Laboratory database to participate in the intervention from September 2024 and July 2025. The participants were invited via phone call to undergo complete overnight polysomnography to confirm the OSA diagnosis.

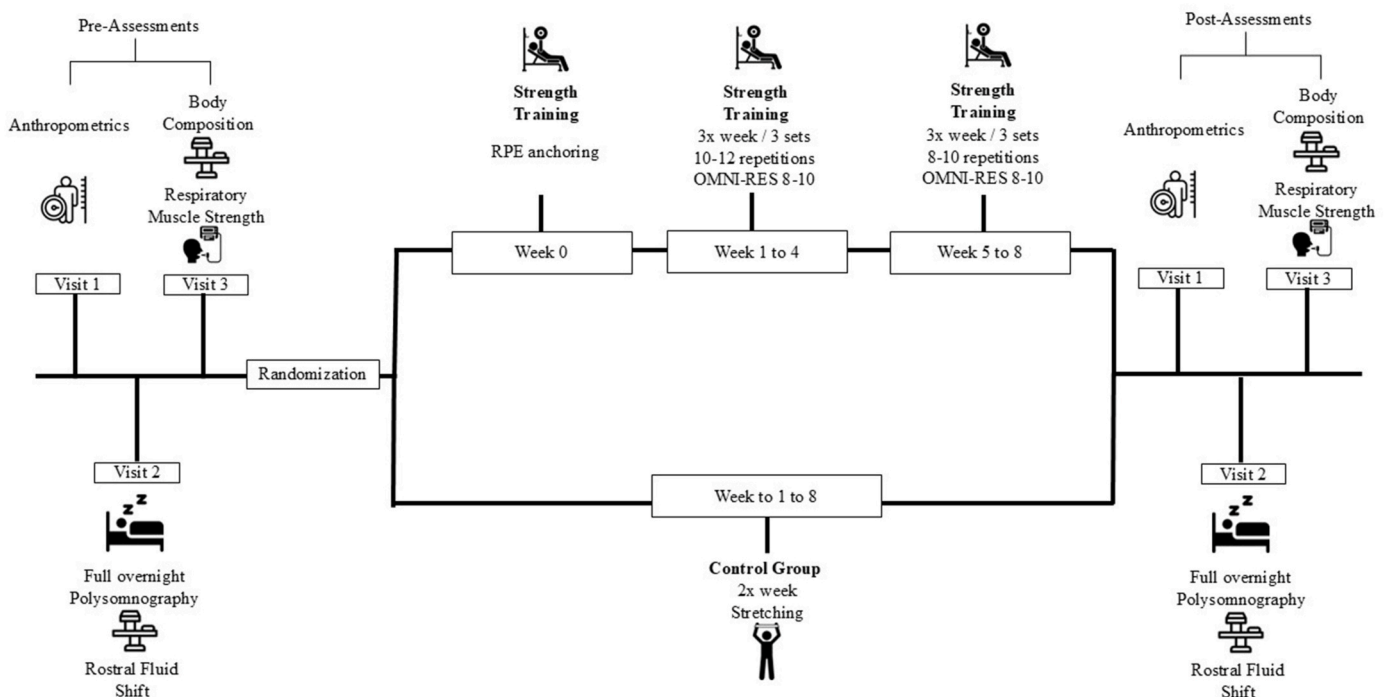


Fig. 1. Experimental design of the study.

2.3. Eligibility criteria

Adults with moderate to severe OSA (AHI ≥ 15 events/hour), diagnosed by full nocturnal polysomnography were included. To be considered eligible, could not be undergoing any previous treatment for OSA, not have participated in a similar intervention in the last six months, and not have physical conditions that would prevent the intervention. Patients who entered the study but did not complete at least 80% of the scheduled sessions in the experimental group were excluded.

2.4. Randomization and allocation

The participants were block randomized using a random number table, stratified for baseline AHI by a researcher not directly involved in the recruitment and data collection, into two groups: RT and CG. Allocation was concealed to the researchers conducting measurements.

2.5. Sleep assessments

Participants underwent a complete nocturnal polysomnography (PSG; Brain Wave II, Neurovirtual). The examination included recordings of electroencephalography, electrooculography, electromyography, pulse oximetry, airflow measurements (oronasal thermistor and pressure cannula), and measurements of thoracic and abdominal movements during respiration. Sleep stages were classified into non-REM sleep (subdivided into stages N1, N2, and N3) and REM sleep, following the guidelines of the American Academy of Sleep Medicine [3, 24]. Apneas were defined as a $\geq 90\%$ reduction in the oronasal airflow signal relative to the pre-event baseline, lasting at least 10 s, identified by a thermal sensor. Hypopneas were defined as a $\geq 30\%$ reduction in oronasal airflow amplitude for at least 10 s, accompanied by $\geq 3\%$ oxygen desaturation and/or arousal [3,24].

The AHI was calculated by dividing the total number of apneas and hypopneas by the total sleep time in hours. All analyses were performed by a researcher blinded to group allocation.

2.6. Anthropometric measurements and body composition

Height and weight were obtained using a digital scale with a stadiometer (Sanny BL201PP), and body mass index was calculated in kg/m^2 . Neck, waist, hip, and calf circumferences were measured using a flexible, inelastic tape measure (Coats Corrente BA1010). Body composition, including body fat mass, skeletal muscle mass, fat-free mass, and segmental lean mass, was assessed using bioelectrical impedance analysis using an 8-point tetrapolar device with tactile electrodes (InBody S10, model JMW140, South Korea), with measurement points on the arms and legs (right and left). Both measurements were performed by a trained evaluator blinded to the intervention.

2.7. Respiratory muscle strength

Respiratory muscle strength was assessed by static respiratory pressure using a HOMET digital vacuum manometer (MVD300-U, ± 300 cmH_2O) [25]. Maximum inspiratory pressure and maximum expiratory pressure in $\text{cm H}_2\text{O}$ were measured, determined by the highest pressure obtained in three attempts at maximum inspiration and expiration, maintained for a continuous flow of 2 s. The test was performed with the patient seated, using a nose clip and mouthpiece, with the escape orifice unobstructed to allow opening of the glottis. If necessary, the maneuver could be repeated up to five times, maintaining the same position and technique in subsequent evaluations, following standardization criteria and recommendations of the American Thoracic Society [26].

2.8. Rostral fluid shift

The rostral fluid shift assessment was performed using the bioelectrical impedance technique. (InBody S10, model JMW140, South Korea). Leg fluid volume was measured using an 8-point tetrapolar bioelectrical impedance scale, 30 min before bedtime with participants positioned supine, lying awake with the legs straight and fully extended, and 30 min after waking up while still in the supine position, before getting out of bed. Bioelectrical impedance measurements were obtained using two pairs of surface electrodes placed on each leg and each wrist. Prior to electrode placement, the skin was cleaned and electrodes were securely fixed to the skin and maintained in place throughout the night. This positioning and electrode configuration protocol was in accordance with previous study [19]. During all measurements, participants were instructed to remain in a supine position to ensure greater accuracy in the data collected. Neck and calf circumferences were also measured to ensure measurement consistency, according to the described protocol. Measurement landmarks were carefully marked to allow repeated assessments at identical anatomical sites. The evaluator was blinded to the participants' clinical information to reduce bias in data collection and interpretation.

2.9. Interventions

2.9.1. Resistance training

The intervention protocol consisted of three weekly RT sessions over eight weeks, totaling 24 sessions, with a minimum interval of 48 h between sessions. Exercises were used for the main muscle groups: bench press, shoulder press, biceps curl, triceps curl, lat pull down, abdominal crunch, leg press, squat, and calf raise. Intensity was prescribed based on the Omni Resistance Exercise Scale (OMNI-RES) [27], a validated 0–10 ratings of perceived exertion scale specifically developed for resistance training, where 0 indicates no effort and 10 corresponds to maximal exertion. With 60 s of rest between sets.

Previous to the intervention, an OMNI-RES scale anchoring session was conducted to familiarize participants with the effort levels and ensure greater accuracy in self-reporting intensity throughout the training. During the anchoring session, participants were instructed to consider a perceived exertion level of '0' as representing no effort at all. Conversely, a level of '10' was anchored to the greatest physical effort they had ever experienced. Based on this scale, the training intensity was then prescribed according to the participants' ratings of perceived exertion [28].

Prior to the intervention, a familiarization phase was conducted, with an intensity of 5 to 7 on the OMNI-RES scale and three sets of 12 to 14 repetitions. From the first to the fourth week, participants performed three sets of 10 to 12 repetitions. From the fifth to the eighth week, the intensity was increased to 8 to 10 on the OMNI-RES scale, with three sets of 8 to 10 repetitions. Abdominal exercises progressed from two sets of 10 repetitions to three sets of 20 repetitions at the end of the protocol. Individualized load progression was based on participants' reports of subjective perception of exertion in each exercise and session. This information was recorded and analyzed by the responsible professional, allowing for gradual adjustments in the loads to maintain the intensity within the range from 8 to 10 proposed. Intervals between sets and exercises were 60 s.

2.9.2. Control group

The participants in the control group performed stretching activities twice a week, on different days from the experimental group, with a duration of 30 min per session, over 8 weeks. The participants were supervised by a Physical Education professional, and the exercises were standardized for both upper and lower limbs. For ethical reasons, all participants were offered resistance training at the end of the study.

2.10. Sample size

To verify an effect size of 0.35 in AHI with a statistical power of 90% and a significance level of 5% ($\alpha = 0.05$). Considering an estimated sample loss rate of 20%, the calculation indicated the need for 29 participants, distributed into two groups [15]. Regarding secondary outcomes, for the respiratory muscle strength, considering previous effect size [29], a statistical power of 80% and a significance level of 5%, 11 participants per group were needed, whereas for rostral fluid shift, considering an effect size of 0.25 [30], and a two-tailed α of 0.05 and β of 80%, 34 participants in two groups were indicated.

2.11. Statistical analysis

The normality of data distribution and homogeneity of variances were verified using the Shapiro-Wilk and Levene tests, respectively. Continuous variables were expressed as mean \pm standard deviation. Categorical variables were presented as absolute values and percentages. Comparisons between groups at baseline were performed using the independent *t*-test (continuous variables) or the chi-square test (categorical variables). To assess the effects of the intervention over time and the interactions between group and time (group \times time point), Generalized Estimating Equations models were used. Spearman's rank correlation coefficient was used to assess the relationships between changes in AHI, rostral fluid shift, respiratory strength, and anthropometric and body composition outcomes in the control and training groups.

The significance level was set at $p < 0.05$. Statistical analyses were performed using Statistical Package for the Social Sciences software, version 20.0.

3. Results

Sixty-two patients were initially selected through the database. Thirty-six were not included, including: CPAP use ($n = 6$), lack of time to participate ($n = 6$), lack of interest in the project ($n = 12$), and other reasons ($n = 12$). Therefore, 26 patients were randomized and allocated to (RT: $n = 13$; CG: $n = 13$). One participant in the RT group did not complete the study, resulting in 13 participants in the CG and 12 in the RT for post-intervention assessments (Fig. 2).

The baseline characteristics of the participants are presented in Table 1. No significant differences were observed between groups. Fig. 3 presents the pre- and post-intervention AHI values for both groups. After eight-weeks of intervention, a significant time \times group interaction was observed ($p = 0.015$). After 8 weeks of intervention, the RT group showed a significant reduction in AHI (from 51.0 ± 6.9 to 30.0 ± 6.1 events/hour), whereas the CG group showed no significant change (from 52.0 ± 7.7 to 48.5 ± 3.8 events/hour).

Table 1

Baseline anthropometric and clinical characteristics.

	RT (n = 12)	CG (n = 13)	p
Apnea-hypopnea index, events/hour	51.0 \pm 6.9	52.0 \pm 7.7	0.922
Age, years	55.2 \pm 6.4	54.1 \pm 8.1	0.710
Male, %	53.8	46.2	0.695
Body weight, kg	103.1 \pm 19.4	91.9 \pm 19.4	0.155
Body mass index, kg/m ²	38.1 \pm 2.4	33.8 \pm 1.4	0.150
Neck circumference, cm	42.2 \pm 6.2	40.6 \pm 4.5	0.456
Waist circumference, cm	119.1 \pm 15.8	110.6 \pm 8.8	0.108
Hip circumference, cm	121.7 \pm 19.4	115.8 \pm 18.2	0.439
Body fat, %	40.8 \pm 10.2	38.1 \pm 7.5	0.521
Fat free mass, %	59.0 \pm 12.5	55.6 \pm 10.4	0.514
Smoker, %	-	7.7	0.141
Hypertension, %	34.6	26.9	0.420
Antihypertensive medications			
ACEI, %	16.7	7.7	0.490
ARB, %	41.7	23.1	0.319
Thiazides/loop diuretic, %	41.7	30.8	0.568
Beta-blocker, %	16.7	23.1	0.689
Calcium channel blocker, %	16.7	38.5	0.225
Diabetes mellitus, %	11.5	7.7	0.619

Data are presented as mean \pm standard deviation or n (%) as appropriate CG – control group; RT – resistance training. ACEI - angiotensin-converting enzyme inhibitor; ARB - angiotensin receptor blocker;

Table 2 shows the effects of RT on OSA severity, sleep and respiratory parameters, anthropometry, body composition, respiratory muscle strength and rostral fluid. There was a time \times group interaction for AHI NREM, with a reduction in the RT group, whereas a time \times group interaction was also observed for arousals, with a decrease in the RT group.

Table 3 presents the correlations between changes in AHI, rostral fluid shift, respiratory muscle strength, and body composition outcomes. A correlation was verified between changes in AHI and waist circumference in RT group, whereas no significant correlations were observed between AHI, and RFS, maximum expiratory, and inspiratory pressures.

4. Discussion

The results of this study demonstrated that eight weeks of RT promoted a reduction in AHI along with improvements in arousals. Moreover, a reduction in central fat was related to decreased OSA severity, whereas no relationship was observed with rostral fluid shift or respiratory muscle strength.

In the present study, it was observed that eight-weeks of RT significantly reduced the AHI by 17.4 events/hour in patients with moderate to severe OSA. Although these findings partially corroborate those of da Silva et al. [15] and de Sá Souza et al. [16], a greater reduction was observed in the present study. We cannot state the reason; however, some points could be addressed to explain the differences. The higher training volume and intensity in the present study, as well as the greater baseline severity of OSA in our sample. For instance, while in the current study, the weekly frequency was three times per week, in the da Silva's study [15], a two-weekly sessions program was performed. Furthermore, the intensity applied in the present study approached near-maximal perceived effort (8-10 on OMNI-RES scale), whereas those previous studies adopted moderate intensities [15,16]. In this sense, accumulating evidence [31,32] has suggested that both number of sessions per week and intensity are key determinants of cardiometabolic adaptations to RT.

Moreover, the improvements in NREM-AHI and arousals in the current study suggest that both sleep-related breathing outcome and sleep continuity could have contributed to reduction in OSA severity. Furthermore, differences in the age of the study populations may also contribute to the variability in the results as older adults generally exhibit a lower magnitude of response to OSA treatments compared to middle-aged adults [33]. Thus, considering that our sample was younger

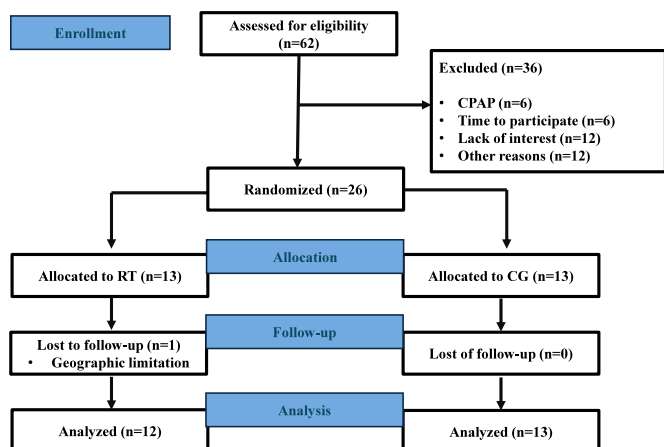


Fig. 2. Study flowchart.

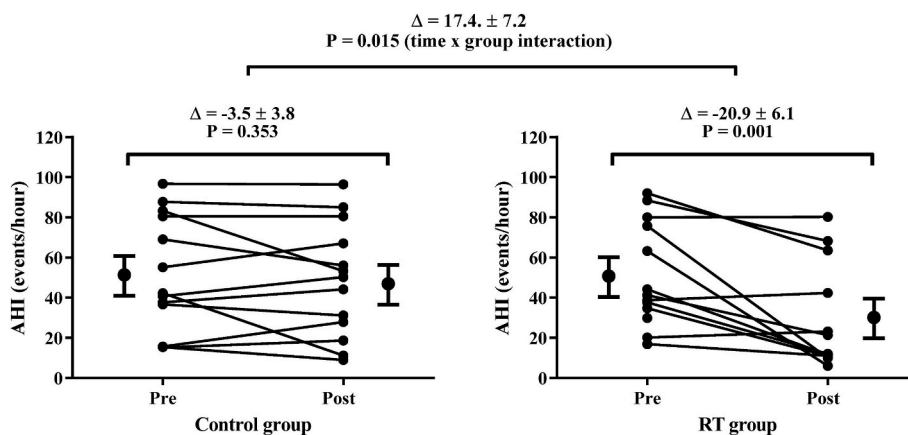


Fig. 3. AHI changes pre- and post-8 weeks of RT and CG interventions.

Table 2

Pre- and post-intervention sleep and respiratory variables, body composition, anthropometry and rostral fluid shift in OSA patients.

	RT (n = 12)			CG (n = 13)			p (time x group)
	Pre	Post	Difference	Pre	Post	Difference	
OSA severity							
AHI REM, events/hours	52.4 ± 6.8	29.4 ± 7.6	-23.0 ± 8.1	53.4 ± 5.8	33.8 ± 8.2	-19.7 ± 10.6	0.801
AHI NREM, events/hours	49.8 ± 7.4	29.3 ± 7.5	-20.5 ± 6.2*	51.1 ± 8.2	47.2 ± 7.7	-3.9 ± 3.9	0.022
Sleep and respiratory parameters							
Sleep efficiency, %	82.5 ± 3.4	91.8 ± 2.2	9.2 ± 3.9	79.5 ± 2.7	85.5 ± 3.4	6.1 ± 4.2	0.554
Arousals, events	35.4 ± 5.5	27.5 ± 4.6	-7.9 ± 4.8*	30.7 ± 5.0	37.7 ± 4.6	7.4 ± 5.1	0.028
Basal saturation, %	91.6 ± 1.2	90.8 ± 2.0	0.9 ± 1.1	94.9 ± 0.5	93.7 ± 0.5	-1.2 ± 0.7	0.778
Mean saturation, %	89.6 ± 1.6	88.5 ± 1.9	-1.1 ± 0.6	93.1 ± 0.5	91.9 ± 0.5	-1.2 ± 0.6	0.965
Minimum saturation, %	73.9 ± 3.3	69.3 ± 3.9	-4.6 ± 2.1	79.9 ± 1.6	76.4 ± 1.7	-3.5 ± 1.3	0.663
ODI,	45.8 ± 7.0	38.5 ± 5.9	-7.3 ± 5.0	43.7 ± 6.3	48.5 ± 7.7	4.8 ± 3.7	0.052
TST, min	352.5 ± 16.3	361.8 ± 26.1	9.3 ± 24.5	318.7 ± 15.0	360.3 ± 14.5	41.5 ± 18.6	0.295
N1, %	16.0 ± 2.8	21.2 ± 3.1	5.2 ± 2.7	18.9 ± 3.6	27.2 ± 3.1	8.3 ± 4.3	0.543
N2, %	59.1 ± 3.3	60.4 ± 2.3	1.3 ± 4.3	59.7 ± 2.8	58.1 ± 2.6	-1.6 ± 4.1	0.629
N3, %	10.9 ± 3.0	10.3 ± 1.9	-0.6 ± 3.6	7.3 ± 1.5	7.4 ± 2.5	0.0 ± 2.6	0.894
REM, %	14.1 ± 1.9	8.1 ± 1.7	-6.0 ± 3.1	14.1 ± 2.0	7.3 ± 2.4	-6.8 ± 3.2	0.854
Anthropometry and body composition							
Body weight, kg	103.1 ± 5.2	103.4 ± 5.3	0.3 ± 1.4	91.9 ± 5.2	92.1 ± 5.3	0.2 ± 0.7	0.969
Body mass index, kg/m ²	38.1 ± 2.4	37.0 ± 2.8	-1.1 ± 0.8	33.8 ± 1.4	34.3 ± 1.4	0.5 ± 0.4	0.071
Body fat, %	40.8 ± 3.2	40.2 ± 3.3	-0.6 ± 1.0	38.1 ± 2.1	36.2 ± 2.5	-1.9 ± 1.7	0.507
Neck circumference, cm	42.2 ± 1.6	43.0 ± 1.3	0.8 ± 0.8	40.6 ± 1.2	41.1 ± 1.2	0.5 ± 0.2	0.655
Waist circumference, cm	121.6 ± 5.2	117.6 ± 4.8	-4.1 ± 1.5	115.8 ± 4.8	114.6 ± 4.3	-1.2 ± 2.1	0.281
Respiratory muscle strength							
MIP, cmH ₂ O	78.0 ± 7.8	80.7 ± 6.9	2.7 ± 5.6	64.6 ± 4.3	5.0 ± 2.5	-7.3 ± 3.4	0.124
MEP cmH ₂ O	111.1 ± 11.6	103.3 ± 9.8	-7.8 ± 6.9	100.7 ± 6.0	92.8 ± 3.6	-7.9 ± 4.5	0.992
Rostral fluid shift							
	-295.7 ± 64.8	-254.3 ± 42.3	41.4 ± 82.0	-279.56 ± 43.5	-306.3 ± 87.0	-26.7 ± 80.5	0.554

Data are presented as mean ± standard error. AHI: apnea-hypopnea index; NREM: non-rapid eye movement; REM: rapid eye movement; ODI: oxygen desaturation index; TST: total sleep time; MEP: Maximum expiratory pressure; MIP: Maximum inspiratory pressure; RFS = rostral fluid shift. *Pre-post intragroup difference, p < 0.05.

Table 3

Correlations between delta of AHI, rostral fluid shift, respiratory strength, anthropometry and body composition outcomes in control and training groups.

Interventions	Delta AHI	
	Rho	p
Control group	Outcome	
	Delta Rostral fluid shift	0.516 0.086
	Delta Maximal expiratory pressure	-0.898 0.102
	Delta Maximal inspiratory pressure	-0.209 0.791
	Delta body mass index	0.113 0.714
Training group	Delta neck circumference	0.196 0.520
	Delta waist circumference	0.377 0.205
	Delta Rostral fluid shift	0.051 0.930
	Delta Maximal expiratory pressure	-0.481 0.334
	Delta Maximal inspiratory pressure	-0.180 0.733
	Delta body mass index	0.013 0.970
	Delta neck circumference	0.128 0.677
	Delta waist circumference	0.643 0.018

AHI – apnea-hypopnea index.

(55.4 years old) compared with the studies by da Silva (71 years old) and Sá Souza (77.4 years old), age differences may have contributed to the observed results in AHI. Future clinical trials are needed to test this hypothesis.

In the present study, a 15.3-events reduction in arousals was observed when comparing RT with control group, indicating less sleep fragmentation. This is particularly important, as previous evidence has shown that a 10-unit decrease in the arousal index is associated with a 24% lower odds of hypertension [34], independent of other sleep-related variables such as nocturnal desaturation and the AHI. In this sense, a single session exercise has been shown to reduce the arousal index in the general population [35], however, the effects of RT on sleep architecture in patients with OSA have not yet been comprehensively investigated, which limits direct comparisons regarding the impact of this exercise modality on sleep fragmentation. Nonetheless, the current findings may be associated with the concomitant reduction in AHI, as the occurrence of arousals is related to respiratory instability and the

recurrence of apneic events [36,37]. In addition, RT shows potential for improving sleep by stimulating the skeletal muscle system, enhancing muscle metabolism [38] and stimulating growth hormone secretion [39], which helps reduce arousals and improve overall sleep quality. Further research suggests that short-term exercise interventions (8–12 weeks) can significantly enhance sleep architecture through acute physiological responses, such as improved circadian rhythm regulation and metabolic shifts [40] modulating inflammatory metabolism, and increasing the proportion of deep sleep. These effects are particularly significant in patients with OSA [11,41] and need to be confirmed.

No significant differences were observed between groups in body composition parameters. However, a positive association was found between waist circumference and the reduction in AHI, suggesting that improvements in OSA severity are directly related to decreases in abdominal fat. Although previous literature has shown that decreases in AHI may result from reductions in body weight [42], accumulating evidence indicates that exercise training can mitigate OSA severity even in the absence of weight loss [11]. Indeed, abdominal adiposity accumulation may reduce pharyngeal lumen size, decrease upper airway muscle protective force and size, and affect restrictive respiratory dysfunction, finally leading to daytime hypoxemia and the development of OSA [43]. Future clinical trials are needed to rigorously test the assumption that the reduction of abdominal obesity following RT elicits redemption in AHI.

No significant improvement was observed in respiratory muscle strength parameters. Considering the sleep-related upper airway dysfunction and repetitive airway obstruction during sleep in OSA, increasing the respiratory muscle strength would lead to the gain of sufficient air pressure for supporting the nasopharynx, thereby increasing upper airway muscle tension [6]. Although increases in respiratory muscle strength parameters have been found following RT programs in general population [29,44], it was not observed in the present study. This could have been due to our RT program. While other studies included multiple stabilizing exercises that stimulate key respiratory muscles [44], the present study employed only an abdominal exercise, which, although it may indirectly recruit respiratory muscles through trunk stabilization and activation of accessory muscles, might not have been sufficient to enhance the synchronization, coordination, and strength of respiratory muscles specifically in this population with OSA.

The assumption that RT would reduce the AHI by decreasing RFS was not confirmed in this study. Exercise training may alleviate OSA severity without requiring weight loss, primarily through activation of the musculovenous pump, which reduces lower-limb fluid retention during the day and its subsequent rostral shift toward the pharyngeal area during sleep [45]. In the study conducted by Monique et al. [30], a reduction in RFS was observed following the intervention. This finding may be partly explained by the clinical characteristics of their sample, which included patients with both OSA and coronary artery disease. Patients with coronary artery disease are prone to greater peripheral fluid accumulation due to factors such as impaired venous return and reduced cardiac output, all of which can promote leg edema during the daytime and a subsequent redistribution of fluid to the upper body during sleep [46,47]. In contrast, participants in the present study had OSA without concomitant coronary artery disease, which may account for the absence of significant changes in RFS following the RT intervention which aligns with da Silva's Study [15] that did not observe improvements in RFS following 12 weeks of RT. Moreover, almost half of the patients used diuretics which may have contributed to a reduced accumulation of lower-limb edema throughout the day, thereby decreasing the amount of fluid accumulated for nocturnal rostral fluid shift. This reduction may have attenuated nighttime fluid redistribution toward the neck. Although the use of diuretics was not controlled in the analysis, this mechanism is physiologically plausible and should be tested in future studies [48]. Thus, although the literature suggests that exercise interventions aimed at reducing edema, and consequently the

RFS, have resulted in a reduction in OSA severity [30,49], RT does not appear to promote significant reductions in lower limb edema [15,50].

The study has some limitations, such as the small sample size. Although it allowed for the identification of significant reductions in AHI, the sample size may have compromised the analysis of secondary variables, namely, the rostral fluid shift, as sample size calculation asked for a total sample size of 34 to achieve a power of 80% with an effect size of 0.25. Thus, future studies with larger sample size are necessary to test the hypothesis that changes in this outcome are associated with changes in the AHI. Another limitation was the low precision of the instrument used to measure body water content. Previous reports of strong correlations between changes in body water content and apnea severity were obtained using magnetic resonance imaging. On the other hand, the use of instruments considered gold standards for assessing sleep, and respiratory muscle strength stands out.

5. Conclusion

Eight weeks of RT significantly improved AHI and arousals in adults with moderate to severe OSA. Reduction in central fat, but not rostral fluid shift or respiratory muscle strength, was associated with improvements in AHI. Future studies should investigate the effects of RT alone at different intensities, considering its potential as an adjunctive treatment for OSA.

CRediT authorship contribution statement

José Ricardo Vieira de Almeida: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Breno Quintella Farah:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization. **José Lucas Porto Aguiar:** Writing – original draft, Formal analysis, Data curation. **Elton Carlos Felinto dos Santos:** Writing – original draft, Data curation. **Victória Duarte de Arantes Lima:** Data curation. **Enrique Nascimento Nazário dos Santos:** Data curation. **Nadja Sylmara dos Santos de Oliveira Silva:** Data curation. **Antônio Henrique Germano-Soares:** Formal analysis, Data curation. **Jose M. Saavedra:** Writing – review & editing, Writing – original draft. **Rodrigo Pinto Pedrosa:** Writing – original draft, Formal analysis, Data curation. **Daniela Karina da Silva Ferreira:** Writing – original draft, Data curation, Conceptualization. **Ozeas de Lima Lins-Filho:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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