Impact of Exercise Training on Obstructive Sleep Apnea: A Systematic Review and Meta-analysis of Randomized Trials

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ABSTRACT

Background: Randomized controlled trials have highlighted the importance of exercise training on the severity of obstructive sleep apnea (OSA). The primary objective of this meta-analysis was to analyze the data from these studies on the severity of OSA indices as measured by formal polysomnography. The secondary objective was to evaluate the effect of exercise on oxygen desaturation index, body mass index and several metabolic parameters associated with OSA.

Methods: Two independent reviewers and an experienced librarian searched MEDLINE, EMBASE, COCHRANE, and Scopus and SPORTDiscus databases from January 1, 2000, to October 21, 2021 to identify randomized controlled trials that evaluated the impact of exercise training on OSA severity. Pre-exercise and postexercise training data on the primary and secondary objectives were extracted.

Results: A total of 11 randomized controlled trials (533 study participants) were identified based on the inclusion criteria. The mean pre-exercise and postexercise reduction in apnea hypopnea index was −6.94 event per hour (95% confidence interval [CI]: −10.74 to −3.13, I^2 = 84%). The mean changes in oxygen desaturation index and body mass index were −5.32 events per hour (95% CI: −9.65 to −0.99, I^2 = 73%) and −1.21 kg·m^{-2} (95% CI: −1.80 to −0.62, I^2 = 49%) respectively.

Conclusion: In adult patients with OSA, exercise training was associated with statistically significant reduction in OSA severity. *J Clin Exerc Physiol*. 2022;11(3):80–90.

Keywords: apnea-hypopnea index, oxygen desaturation index, body mass index

INTRODUCTION

Obstructive sleep apnea (OSA) is a concerning diagnosis defined as the presence of at least 5 episodes of upper airway closure with each hour of sleep, and each episode halting airflow for at least 10 seconds (1). These episodes are known as apnea and can result in a decrease in oxygen saturation, hypercapnia, elevated oxidative stress, and increased sympathetic activity, which have been implicated as part of the pathophysiologic processes, and have deleterious short-term and long-term consequences (1–3). These include but are not limited to diurnal hypertension, arrhythmia, stroke, pulmonary hypertension, heart failure, chronic sleep deprivation, chronic fatigue, decreased executive function, personality

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changes, and an elevated risk for sudden cardiac death (1,2). The number of apnea episodes measured per hour during a sleep evaluation is known as the *apnea hypopnea index* (AHI).

Several lifestyle-related risk factors are known to predispose to the development of OSA (2,4–6). These are thought to directly or indirectly result in complete or partial collapse of the pharynx causing enlargement of the surrounding soft tissues through hypertrophy, inflammation, and/or edema, which are mostly pronounced in the lateral pharyngeal wall resulting in a reduction of the genioglossus muscle activity (7,8).

The diagnosis and severity measures of OSA are based on laboratory polysomnography (PSG) with several indices measured that estimate the severity of the disease burden. Mild, moderate, and severe OSA are diagnosed as AHI ≥5, ≥15 and ≥30, respectively (4,9). According to prior studies, treatment options including continuous positive airway pressure (10), bilevel-positive airway pressure (11), oral appliances including mandibular advancement devices (11–13), surgical options (including bariatrics, maxillomandibular adjustments, and hypoglossal nerve stimulation) (11,13,14), weight loss via dietary modification (15), and pharmacotherapy (16) have reduced the severity of OSA. However, their impact on AHI reduction appears to be temporary and variable. This is based on the outcomes of studies (15,17–21) that have evaluated their long-term effectiveness.

The role of exercise training in reducing AHI and improving other PSG indices has been growing in the recent literature (9,22,23). The mechanisms behind the beneficial impact of exercise on OSA indices are yet to be elucidated (24). Previous systematic reviews that evaluated the impact of exercise on OSA indices have either included studies other than RCTs (9,24–26), with exercise duration as short as 3 weeks (27), or excluded OSA participants with comorbidities such as heart failure and stroke (9,25). The primary aim of this meta-analysis is to investigate the impact of exercise training on AHI as measured by formal PSG in randomized controlled studies. The secondary aim is to evaluate the effect of exercise on oxygen desaturation index (ODI),body mass index (BMI), and risk factors associated with OSA.

METHODS

Data Sources and Searches

Two authors (AO and FM) and a qualified librarian independently searched MEDLINE, EMBASE, COCHRANE, and Scopus and SPORTDiscus databases from January 1, 2000, to October 21, 2021. The keywords and Medical Subject Heading search terms used were exercise, therapy, physical, rehabilitation, sleep, apnea, obstructive, sleep disorders, and sleep breathing disorders. In addition, different combinations of these keywords were applied in the database search (Supplemental Figure S1). To ensure thoroughness, we manually searched the reference lists of the articles and previously published systematic reviews and meta-analyses on exercise and OSA to identify references not included in the automated search. The search was restricted to articles published in English language. We adhered to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guideline (28) (table 4; Supplemental Figure S1). The PRISMA flow chart and the reasons for study exclusion are provided in Figure 1.

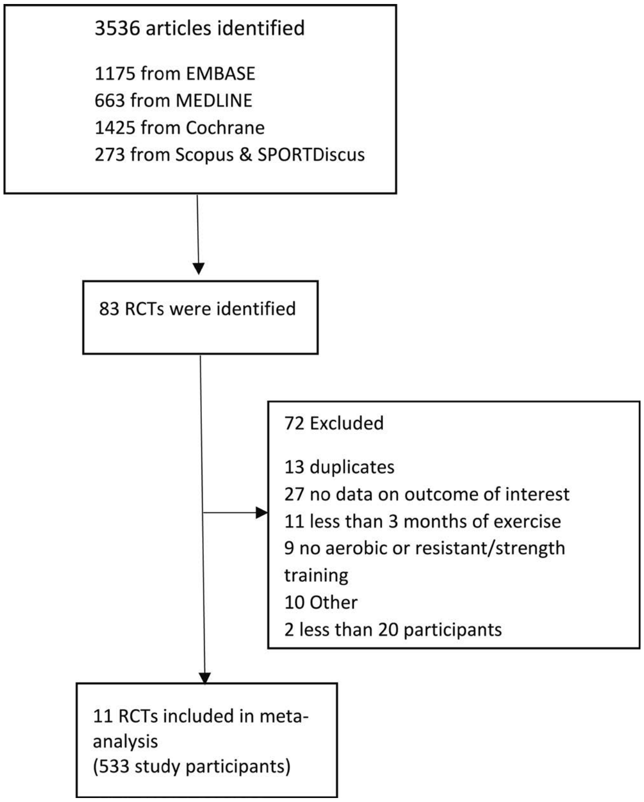


FIGURE 1. PRISMA flowchart of the study selection.

Selection of Studies

Eligible studies were randomized controlled trials (RCTs) that included OSA patients that underwent a structured supervised or unsupervised exercise training as the treatment group and a control group that did not participate in the exercise training but was encouraged to maintain their baseline physical activity level. RCTs were selected if they reported preintervention and postintervention changes in AHI, ODI, and BMI, included a minimum of 20 participants with OSA diagnosed by PSG with AHI of at least 5, and age > 18 years. The total exercise duration must be a minimum of 3 months, at least 3 exercise sessions a week, each session lasting ≥30 minutes, and comprised of aerobic exercises and/or resistance training. Studies not meeting these criteria, or used dental sleep devices, oropharyngeal muscle training, or surgery were excluded. Disagreements on article inclusion were discussed among authors and resolved by consensus.

Outcomes and Data Extraction

The primary outcome was to compare postintervention changes in AHI between the exercise group and the control group. The secondary outcome was to compare the postintervention changes in ODI and BMI between both groups. A prespecified subgroup analysis was performed for diabetes mellitus (DM), hypertension, waist circumference, age, gender and smoking. Data extracted from the selected RCTs included author, publication year, mean age of the participants, number of subjects, study duration, frequency and duration of exercise, type of exercise, and preintervention and postintervention PSG indices (AHI, ODI), BMI, DM, hypertension, gender, age, smoking, waist circumference. The accuracy of the extracted data was assessed and confirmed by authors SU and MM.

Quality Assessment

The methodologic quality of the RCTs were assessed using the tool for assessment of study quality and reporting in exercise (TESTEX) scale. It comprises 12 criteria with a maximum of 15 points: 5 points for study design and 10 points for reporting (29) (Table 1). Studies with a score of 12 to 15 points are of high quality, those with 7 to 11 points are good quality, and those with ≤6 points are low quality (29).

Data Analysis

Data analysis was performed by NY using the RevMan (version 5.3; Copenhagen: The Nordic Cochrane Center, The Cochrane Collaboration, Copenhagen, Denmark). The forest plots of the above outcomes were visually represented after pooling the mean differences using the random-effects model. Heterogeneity was assessed with the I^2 test. Publication bias was assessed with the funnel plot and standard error effect sizes. We used the literature recommended threshold of statistical significance which is $P < 0.05$ for outcomes and $P < 0.10$ for heterogeneity.

RESULTS

Results of Search

We retrieved a total of 3,536 articles from the initial search (663 from MEDLINE, 1,175 from EMBASE, 1,425 from CENTRAL, 273 from Scopus and SPORTDiscus). Of these, 83 were RCTs. After excluding 13 duplicates, we analyzed the title and method section of the remaining 70 articles, and 59 of these articles were excluded for failing to meet the priori inclusion criteria. Therefore, the final analysis comprised of 11 articles with 533 participants (30–40) (Table 2).

Characteristics of Included Studies

All studies were randomized, included preintervention and postintervention AHI measures with formal PSG, and had exercise of >3 months. Five studies assessed changes in ODI (31–33,35,36), and 7 (22,30,31,33,35,37,38) reported changes in BMI. Patients had a mean age of 51 years, 69% were males. The exercise group had moderate to severe OSA with a mean AHI of 32 ± 14 events per hour with an average BMI of 29 ± 3 kg·m^{-2}. The mean ODI is 28 ± 15 events per hour. The control group also had moderate to severe OSA with a mean AHI of 31 ± 12 events per hour, the mean ODI was 12 events per hour with a range from 0 to 15 events per hour and an average BMI of 24.7 ± 12.5 kg·m^{-2}. The exercise programs were supervised in 9 of the studies (22,30,31,33–36,38,39). The mean exercise duration was 5 months, with an average of 3 sessions per week, each lasting an average of 60 minutes. Aerobic exercises included running/walking on the treadmill and elliptical/cycle ergometer (30,31,33–37). Resistance training (22,33,39,40) included shoulder press, latissimus dorsi pulldown, chest press, bicep/triceps curls, leg extension/curl, isometric handgrips, and isokinetic dynamometry. Additionally, the studies reported improvement in daytime excessive sleepiness either via the Epworth sleepiness scale or Pittsburgh sleep quality index (30,32,34,36,38). The funnel plot analysis was insignificant for publication bias (Supplemental Figure S1).

Outcome Measures

In the exercise group, the mean change in AHI compared to baseline is −8.7 ± 5.5 events per hour. The mean change in AHI compared to baseline in the control group is 0.5 ± 6.4 events per hour. The mean change in ODI in the exercise group is −5.6 ± 6.8 events per hour, and 0.6 ± 8.4 events per hour in the control group. The mean change in BMI in the exercise group is −1.3 ± 1.3 kg·m^{-2}, and −0.3 ± 0.6 kg·m^{-2} in the control group. Exercise treatment was associated with a statistically significant reduction in the AHI (mean difference compared to control of −8.2 events per hour, 95% CI: −11.9 to −4.6, $I^2 = 84\%$). The ODI was significantly lower in the exercise group (mean difference compared to control of −5.3 events per hour, 95% CI: −9.7 to −1.0, $I^2 = 73\%$). In addition, the exercise also led to a decrease in BMI that was statistically significant (mean difference compared to control of −1.2 kg·m^{-2}, 95% CI: −1.8 to −0.6, $I^2 = 49\%$) (Figure 2A-C). A subgroup analysis based on the risk factors associated with OSA (including age, smoking, alcohol use, DM, and hypertension) demonstrated no significant reduction in AHI with exercise training (Figure 3A-G).

DISCUSSION

In this analysis, we evaluated the impact of exercise training on PSG-derived OSA indices and BMI by comparing the mean pre-exercise and postexercise parameters in a cohort of 533 participants from 11 studies. The outcomes showed a significant reduction in AHI, ODI, and BMI (by 22%, 19%, and 4%, respectively) in OSA patients that underwent exercise training.

In contrast to previous meta-analyses (9,24,26,27), our meta-analysis included only RCTs, which provide the best level of evidence that support an intervention. Importantly, this study had a higher effect size (Cohen d: 3.1 vs. 1.6, 2.8, 1.2, and 2.0, respectively). Additionally, with a longer average duration of exercise, the statistical power was enough to highlight a significant reduction in BMI. Furthermore, via the subgroup analysis, this study demonstrates that the improvement of OSA with exercise therapy extends across risk factors associated with OSA such as age, DM, and BMI. This paper represents the most contemporary literature to support the important benefits of exercise as an important, validated,

ORIGINAL RESEARCH

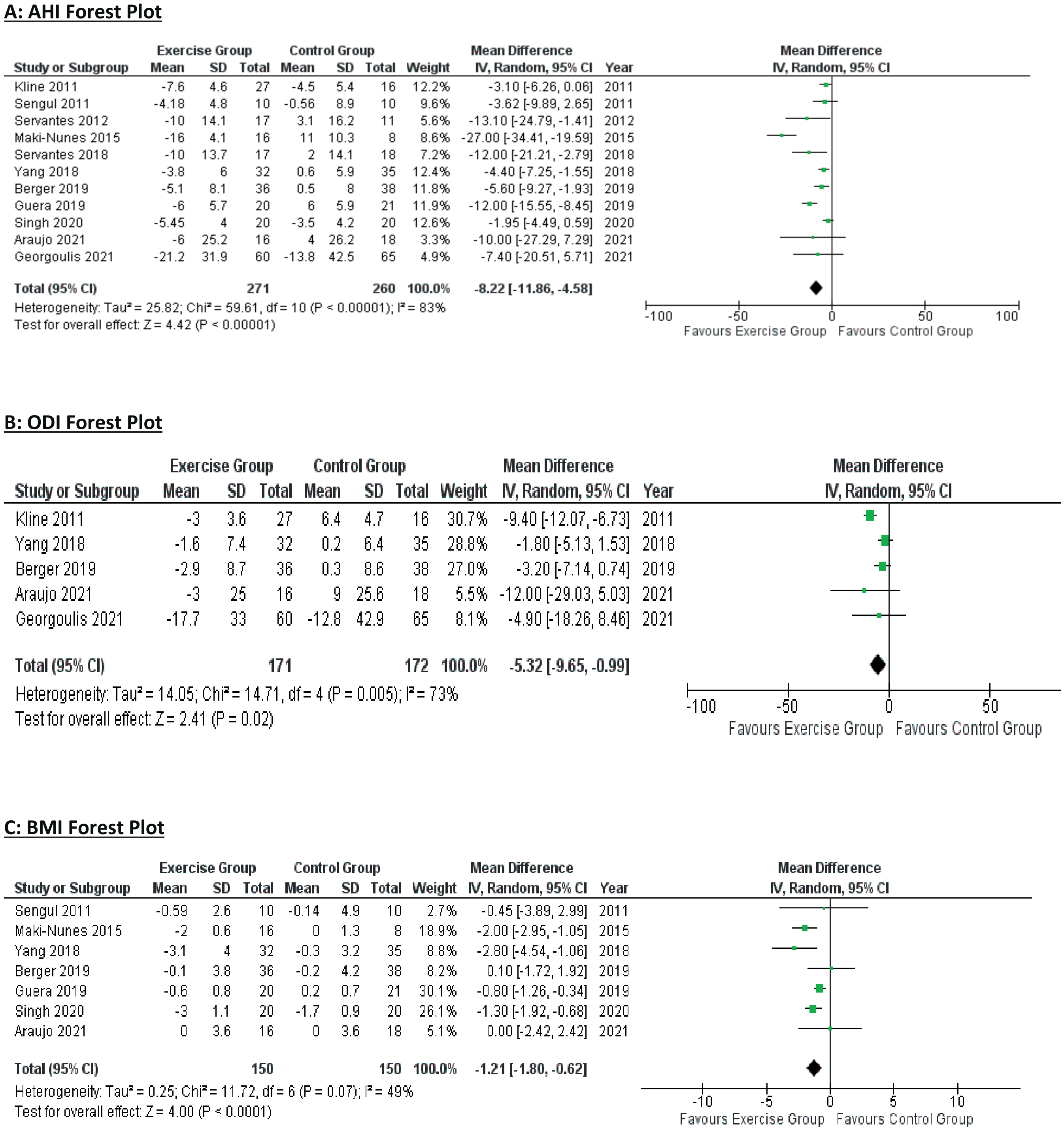
TABLE 1. TESTEx scores for each study.

Study	Eligibility Criteria Specified	Randomly Allocated Participants	Allocation Concealed	Groups Similar at Baseline	Assessors Blinded	Reported Adherence >85%	Reported Adverse Events	Reported Exercise Attendance	Intention to Treat Analysis	Reporting of Between-Group Statistical Comparisons	Point Measures and Measures of Variability Reported	Activity Monitoring in Control Group	Relative Exercise Intensity Review	Exercise Volume and Energy Expended	Overall TESTEX
Sengul et al.[30]	Yes	Yes	Unclear	Yes	Unclear	No	No	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	9
Araújo et al.[31]	Yes	Unclear	Unclear	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	9
Georgoulis et al.[32]	Yes	Yes	Unclear	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	10
Berger et al.[33]	Yes	Unclear	Unclear	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Unclear	Unclear	Yes	10
Servantes et al.[34]	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	12
Yang et al.[35]	Yes	Yes	Unclear	Yes	Yes	Yes	No	Yes	No	Yes	Yes	Unclear	Unclear	Yes	9
Kline et al.[36]	Yes	Unclear	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	13
Guerra et al.[22]	Yes	Unclear	Unclear	Yes	Yes	Yes	No	Yes	No	Yes	Yes	Unclear	Yes	Yes	9
Maki-Nunes et al.[37]	Yes	No	No	No	Yes	Yes	No	No	Yes	Yes	Yes	Unclear	Yes	Yes	8
Singh et al.[38]	Yes	Yes	Unclear	Yes	Unclear	Yes	No	Yes	Yes	Yes	Yes	Unclear	Unclear	Yes	9
Servantes et al.[39]	Yes	Yes	Unclear	Yes	Unclear	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	11

TABLE 2. Reported baseline characteristics of participants.[a]

Author	Number of Participants (% of Males)	Age, Mean (SD)	AHI, Mean (SD)	ODI, Mean (SD)	BMI, Mean (SD)	Supervision	Study Intervention
Sengul et al.[30]	Exercise: 10 (100)	Exercise: 54.4 (6.6)	Exercise: 15.19 (5.43)	Exercise: N/A	Exercise: 29.8 (2.7)	Yes	Breathing ex for 15-20 min, 3 sessions per wk for 12 wks. Aerobic ex initially lasted 45-60 min & 60-90 min 3 times wkly for 12 wks
	Control: 10 (100)	Control: 48.0 (7.5)	Control: 17.92 (6.45)	Control: N/A	Control: 28.42 (5.4)		
Araújo et a.l[31]	Exercise: 6 (38)	Exercise: 50.0 (6.0)	Exercise: 44.0 (31.0)	Exercise: 38.0 (31.0)	Exercise: 30 (4)	Yes	Ex session consisted of 10 min stretching, 40 min of aerobics, 10 min strengthening ex for 3 days per wk for a total of 72 sessions for 40 wks
	Control: 12 (67)	Control: 54.0 (8.0)	Control: 45.0 (27.0)	Control: 34.0 (27.0)	Control: 29 (4)		
Georgoulis et al.[32]	Exercise: 46 (77)	Exercise: 48 (10)	Exercise: 62 (N/A)	Exercise: 44 (N/A)	Exercise: 35.8 (6.0)	No	150 min per wk of moderate intensity aerobics for 6 months
	Control: 51 (79)	Control: 48 (10)	Control: 62 (N/A)	Control: 42 (N/A)	Control: 36.1 (6.4)		
Berger et al.[33]	Exercise: 24 (67)	Exercise: 62 (N/A)	Exercise: 21.9 (7.0)	Exercise: 21.4 (7.1)	Exercise: 28.4 (4.3)	Yes	3 hours per wk of aerobics for 9 months. Total of 88 sessions, each consisting of 10 min of warm-up, 40 min resistance and aerobic ex, and 10 min stretching
	Control: 22 (58)	Control: 62 (N/A)	Control: 21.0 (6.3)	Control: 22 (7.6)	Control: 28.5(4.5)		
Servantes et al.[34]	Exercise: 12 (71)	Exercise: 53.0 (10.0)	Exercise: 25.0 (15.0)	Exercise: N/A	Exercise: N/A	Yes	3 sessions per wk for 3 months: 10 min warmup, 30-45 min of aerobic and strength training
	Control: 12 (63)	Control: 57.0 (8.0)	Control: 32.0 (25.0)	Control: N/A	Control: N/A		
Yang et al.[35]	Exercise: 22 (69)	Exercise: 46.3 (6.4)	Exercise: 20.2 (7.5)	Exercise: 13.0 (8.2)	Exercise: 27.6 (4.7)	Yes	3 sessions per wk for 12 wks: 15 min warmup, 30 min aerobics
	Control: 24 (69)	Control: 48.6 (7.2)	Control: 19.5 (6.1)	Control: 12.4 (7.9)	Control: 27.1 (3.5)		
Kline et al.[36]	Exercise: 15 (56)	Exercise: 47.6 (1.3)	Exercise: 32.2 (5.6)	Exercise: 24.5 (4.2)	Exercise: 35.5 (1.2)	Yes	4 sessions per wk for 12 wks: 150 min per wk of aerobics; 2 times per wk of resistance training
	Control: 9 (56)	Control: 45.9 (2.2)	Control: 24.4 (5.6)	Control: 16.8 (4.2)	Control: 33.6 (1.4)		
Guerra et al.[22]	Exercise: 9 (45)	Exercise: 53.0 (2.0)	Exercise: 44.0 (7.0)	Exercise: N/A	Exercise: 29.6 (0.9)	Yes	Ex session consisted of 5 min stretching, 40 min of aerobics, 10 min strengthening ex, and 5 min muscle stretching. 72 sessions over 6 months
	Control: 15 (71)	Control: 50.0 (1.0)	Control: 44.0 (6.0)	Control: N/A	Control: 29.5 (0.8)		
Maki-Nunes et al.[37]	Exercise: 11 (69)	Exercise: 53 (1.7)	Exercise: 34.0 (5.1)	Exercise: N/A	Exercise: 32.0 (0.7)	NS	3 sessions per wk, for 4 months: 5 min stretching ex, 40 min of aerobics, and 15 min strengthening ex
	Control: 6 (75)	Control: 42 (2.6)	Control: 36.0 (10.1)	Control: N/A	Control: 32.0 (1.3)		
Singh et al.[38]	Exercise: 18 (90)	Exercise: 54.2 (10.5)	Exercise: 24.1 (4.8)	Exercise: N/A	Exercise: 27.8 (0.9)	Yes	4-5 sessions per wk resistance ex for 3 months
	Control: 17 (85)	Control: 48.2 (14.2)	Control: 21.4 (4.8)	Control: N/A	Control: 27.5 (1.1)		
Servantes et al.[39]	Exercise: 17 (47.1)	Exercise: 50.8 (9.5)	Exercise:26.4 (17.6)	Exercise: N/A	Exercise: N/A	Yes*	Group 1: 3-4 sessions per wk for 12 wks of aerobics for 30-45 min per session Group 2: In addition to aerobic session above, 3-4 sessions per wk of resistance ex
	Control: 11 (45.5)	Control: 53.0 (8.2)	Control: 22.8 (17.4)	Control: N/A	Control: N/A		

AHI = apnea hypopnea index; BMI = body mass index; ex = exercise; ODI = oxygen desaturation index
[a]First 3 months of exercise training were supervised; remaining sessions were home based and unsupervised



A: AHI Forest Plot

Study or Subgroup	Exercise Group Mean	SD	Total	Control Group Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
Kline 2011	-7.6	4.6	27	-4.5	5.4	16	12.2%	-3.10 [-6.26, 0.06]	2011
Sengul 2011	-4.18	4.8	10	-0.56	8.9	10	9.6%	-3.62 [-9.89, 2.65]	2011
Servantes 2012	-10	14.1	17	3.1	16.2	11	5.6%	-13.10 [-24.79, -1.41]	2012
Maki-Nunes 2015	-16	4.1	16	11	10.3	8	8.6%	-27.00 [-34.41, -19.59]	2015
Servantes 2018	-10	13.7	17	2	14.1	18	7.2%	-12.00 [-21.21, -2.79]	2018
Yang 2018	-3.8	6	32	0.6	5.9	35	12.4%	-4.40 [-7.25, -1.55]	2018
Berger 2019	-5.1	8.1	36	0.5	8	38	11.8%	-5.60 [-9.27, -1.93]	2019
Guera 2019	-6	5.7	20	6	5.9	21	11.9%	-12.00 [-15.55, -8.45]	2019
Singh 2020	-5.45	4	20	-3.5	4.2	20	12.6%	-1.95 [-4.49, 0.59]	2020
Araujo 2021	-6	25.2	16	4	26.2	18	3.3%	-10.00 [-27.29, 7.29]	2021
Georgoulis 2021	-21.2	31.9	60	-13.8	42.5	65	4.9%	-7.40 [-20.51, 5.71]	2021
Total (95% CI)			**271**			**260**	**100.0%**	**-8.22 [-11.86, -4.58]**	

Heterogeneity: Tau² = 25.82; Chi² = 59.61, df = 10 (P < 0.00001); I² = 83%
Test for overall effect: Z = 4.42 (P < 0.00001)

B: ODI Forest Plot

Study or Subgroup	Exercise Group Mean	SD	Total	Control Group Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
Kline 2011	-3	3.6	27	6.4	4.7	16	30.7%	-9.40 [-12.07, -6.73]	2011
Yang 2018	-1.6	7.4	32	0.2	6.4	35	28.8%	-1.80 [-5.13, 1.53]	2018
Berger 2019	-2.9	8.7	36	0.3	8.6	38	27.0%	-3.20 [-7.14, 0.74]	2019
Araujo 2021	-3	25	16	9	25.6	18	5.5%	-12.00 [-29.03, 5.03]	2021
Georgoulis 2021	-17.7	33	60	-12.8	42.9	65	8.1%	-4.90 [-18.26, 8.46]	2021
Total (95% CI)			**171**			**172**	**100.0%**	**-5.32 [-9.65, -0.99]**	

Heterogeneity: Tau² = 14.05; Chi² = 14.71, df = 4 (P = 0.005); I² = 73%
Test for overall effect: Z = 2.41 (P = 0.02)

C: BMI Forest Plot

Study or Subgroup	Exercise Group Mean	SD	Total	Control Group Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
Sengul 2011	-0.59	2.6	10	-0.14	4.9	10	2.7%	-0.45 [-3.89, 2.99]	2011
Maki-Nunes 2015	-2	0.6	16	0	1.3	8	18.9%	-2.00 [-2.95, -1.05]	2015
Yang 2018	-3.1	4	32	-0.3	3.2	35	8.8%	-2.80 [-4.54, -1.06]	2018
Berger 2019	-0.1	3.8	36	-0.2	4.2	38	8.2%	0.10 [-1.72, 1.92]	2019
Guera 2019	-0.6	0.8	20	0.2	0.7	21	30.1%	-0.80 [-1.26, -0.34]	2019
Singh 2020	-3	1.1	20	-1.7	0.9	20	26.1%	-1.30 [-1.92, -0.68]	2020
Araujo 2021	0	3.6	16	0	3.6	18	5.1%	0.00 [-2.42, 2.42]	2021
Total (95% CI)			**150**			**150**	**100.0%**	**-1.21 [-1.80, -0.62]**	

Heterogeneity: Tau² = 0.25; Chi² = 11.72, df = 6 (P = 0.07); I² = 49%
Test for overall effect: Z = 4.00 (P < 0.0001)

FIGURE 2A-C. Forest plots highlighting statistical significance improvement in apnea-hypopnea index (AHI), oxygen desaturation index (ODI), and body mass index (BMI).

inexpensive, and portable intervention in addition to continuous positive airway pressure for OSA patients. The magnitude of AHI and ODI reduction noted in this meta-analysis is lower compared to that of previous meta-analyses and observational studies that have evaluated other interventions besides exercise training. These include dietary weight loss (AHI: −14.3, 95% CI: −23.5, −5.1) (40), oral appliances (AHI: −12.1, 95% CI: −9.7, −14.3) (41), surgical weight loss (pre-AHI: 54.7, 95% CI: 49, 60.3; post-AHI: 15.8, 95% CI: 12.6, 19.0) (42), (ODI: 43.6 to 18.3) (43), and upper airway surgery (ODI: 12.3 ± 9.8 to 4.0 ± 4.2) (AHI: 21.4 ± 13.8 to 5.8 ± 7.2) (44,45). This difference in magnitude might be due to the combined effect of exercise training with these interventions. The subjects that underwent these interventions had severe OSA as similar to this analysis. However, these interventions (except for surgical weight loss) had an identical impact on OSA severity with exercise training (i.e., a reduction from severe/moderate to moderate/mild OSA). Hence, future RCTs are needed to evaluate and compare the impact of these interventions to exercise training. It is not well established if the reduction in AHI and ODI by exercise training is sustainable in the long term, especially if exercise ceases (46). It is known that some of the other interventions (21,47,48) do not have a long-lasting impact on OSA indices.

The reduction in BMI noted in this study is not clinically significant. It might be due to the inclusion of RCTs in which the intervention groups were subjected to both dieting and exercise training (38,39). In support of this, Araghi et al (26) noted a decrease in pooled mean BMI that was not significant with exercise training after adjusting for diet as a cointervention. Hence, our findings highlight that the improvement in AHI and ODI is independent of weight loss. This is because it implies that exercise as a single intervention may be sufficient to improve OSA severity and in fact may impact long-term compliance (40). In addition, improvement in AHI also led to clinically significant reduction in both systolic and diastolic blood pressure without a significant change in BMI (27).

The mechanism involved in the reduction of AHI and ODI by exercise training possibly includes the effect of exercise on reduction of pharyngeal adipose volume, or simply strengthening the pharyngeal muscles (49), nocturnal rostral fluid shift that reduces airway edema (51) and oxidative stress (51,52). The independence of its impact on BMI reduction might be explained by the selective deposition of adipose tissue in pharyngeal airway in OSA patients compared to BMI matched controls without OSA (53). In support of this, changes in pharyngeal adipose deposition have been noted on cervical magnetic resonance imaging and facial computed tomography have been correlated with the severity of OSA (54,55). Furthermore, the independence of BMI raises the question of whether neck adipose tissue has a higher metabolism than that of the abdomen and hence reduces at a faster rate to exercise than does abdominal tissue.

The ideal or minimal duration of exercise needed to significantly reduce the OSA indices in the long term remains unknown (46). Our results are similar to those of other studies that have evaluated different exercise duration and form (aerobic and/or resistant training) (9,24,27,38,55,56). Therefore, comparing the long-term impact of aerobic exercises and resistance training on OSA indices might be the aim of future studies. Additionally, studies are needed to evaluate the optimal time of the day for exercise training that would significantly improve OSA indices. Although this study sought to include both supervised and unsupervised exercise programs in its analysis, only 1 of the included studies (35) used unsupervised exercise. Hence, it underrepresents the impact of unsupervised exercise on OSA severity. In studies that included unsupervised exercise as an intervention, the reduction in OSA severity was not statistically significant (57). Therefore compared to supervised exercise, the literature currently lacks robust evidence supporting the efficacy of unsupervised exercise.

Limitations

This meta-analysis was limited by the inclusion and exclusion criteria described above. Furthermore, this study has a significant in-between trial heterogeneity observed in terms of AHI and ODI. While this might be related to the differences in exercise protocols, training atmosphere, and use of diet in some studies, it highlights the need for a patient-level meta-analysis. Lastly, this study did not address the long-term effects of exercise on OSA severity after completion of the exercise protocols. For Figure 4b: controlling for gender, DM2.

CONCLUSION

This study demonstrated a statistically significant reduction in AHI and ODI observed with exercise training. The improvement in these OSA indices is independent of weight loss. Future studies are needed to determine if the improvement in OSA indices observed with exercise training is sustainable over the long term.

A: Age Subgroup

	Exercise Group			Control Group				Mean Difference	
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI	Year
Sengul 2011	54.4	6.6	10	48	7.5	10	9.4%	6.40 [0.21, 12.59]	2011
Servantes 2012	50.8	9.5	17	53	8.2	11	9.0%	-2.20 [-8.82, 4.42]	2012
Maki-Nunes 2015	53	1.7	16	42	2.6	8	13.1%	11.00 [9.02, 12.98]	2015
Servantes 2018	51	9	18	57	8	19	10.1%	-6.00 [-11.50, -0.50]	2018
Yang 2018	46.3	6.4	32	48.6	7.2	35	12.2%	-2.30 [-5.56, 0.96]	2018
Berger 2019	62	5.911	36	62	6.0847	38	12.6%	0.00 [-2.73, 2.73]	2019
Guera 2019	53	2	20	50	1	21	13.6%	3.00 [2.02, 3.98]	2019
Singh 2020	54.2	10.5	20	48.2	14.2	20	8.0%	6.00 [-1.74, 13.74]	2020
Georgoulis 2021	48	10	60	48	10	65	12.0%	0.00 [-3.51, 3.51]	2021
Total (95% CI)			**229**			**227**	**100.0%**	**1.85 [-1.56, 5.26]**	

Heterogeneity: Tau² = 22.07; Chi² = 93.58, df = 8 (P < 0.00001); I² = 91%
Test for overall effect: Z = 1.06 (P = 0.29)

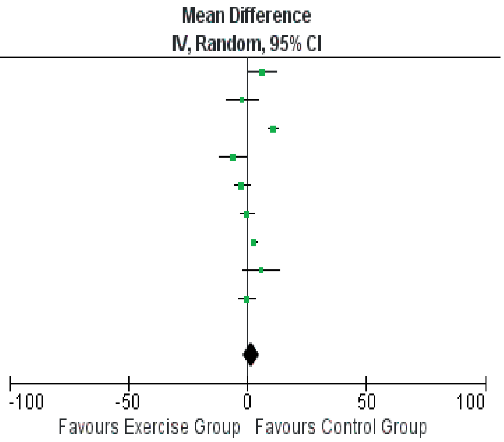
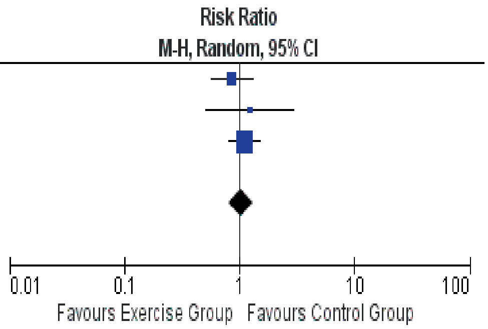


FIGURE 3A-G. Subgroup analysis based on the risk factors associated with obstructive sleep apnea.

B: Hypertension Subgroup

Study or Subgroup	Exercise Group Events	Exercise Group Total	Control Group Events	Control Group Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Servantes 2012	12	17	9	11	32.7%	0.86 [0.57, 1.31]	2012
Servantes 2018	7	18	6	19	7.3%	1.23 [0.51, 2.97]	2018
Berger 2019	26	36	25	38	60.0%	1.10 [0.81, 1.49]	2019
Total (95% CI)		**71**		**68**	**100.0%**	**1.02 [0.81, 1.30]**	
Total events	45		40				

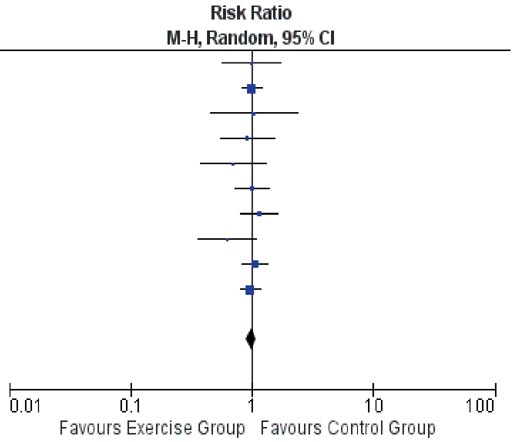
Heterogeneity: Tau² = 0.00; Chi² = 1.09, df = 2 (P = 0.58); I² = 0%
Test for overall effect: Z = 0.19 (P = 0.85)



C: Gender Subgroup

Study or Subgroup	Exercise Group Events	Exercise Group Total	Control Group Events	Control Group Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Kline 2011	15	27	9	16	3.1%	0.99 [0.57, 1.71]	2011
Sengul 2011	10	10	10	10	27.8%	1.00 [0.83, 1.20]	2011
Servantes 2012	8	17	5	11	1.4%	1.04 [0.46, 2.35]	2012
Maki-Nunes 2015	11	16	6	8	3.4%	0.92 [0.55, 1.54]	2015
Servantes 2018	8	18	12	19	2.4%	0.70 [0.38, 1.31]	2018
Yang 2018	22	32	24	35	8.8%	1.00 [0.73, 1.39]	2018
Berger 2019	24	36	22	38	7.3%	1.15 [0.81, 1.64]	2019
Guera 2019	9	20	15	21	3.0%	0.63 [0.36, 1.10]	2019
Singh 2020	18	20	17	20	16.8%	1.06 [0.84, 1.34]	2020
Georgoulis 2021	46	60	51	65	25.9%	0.98 [0.81, 1.18]	2021
Total (95% CI)		**256**		**243**	**100.0%**	**0.99 [0.90, 1.09]**	
Total events	171		171				

Heterogeneity: Tau² = 0.00; Chi² = 5.13, df = 9 (P = 0.82); I² = 0%
Test for overall effect: Z = 0.23 (P = 0.82)



D: Diabetes Subgroup

Study or Subgroup	Exercise Group Events	Exercise Group Total	Control Group Events	Control Group Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Servantes 2012	9	17	4	11	36.7%	1.46 [0.59, 3.58]	2012
Servantes 2018	9	18	4	19	30.7%	2.38 [0.89, 6.36]	2018
Berger 2019	8	36	6	38	32.6%	1.41 [0.54, 3.66]	2019
Total (95% CI)		**71**		**68**	**100.0%**	**1.67 [0.97, 2.89]**	
Total events	26		14				

Heterogeneity: Tau² = 0.00; Chi² = 0.70, df = 2 (P = 0.70); I² = 0%
Test for overall effect: Z = 1.85 (P = 0.06)

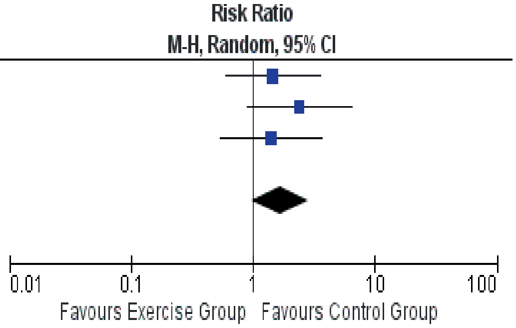


FIGURE 3A-G. Continued.

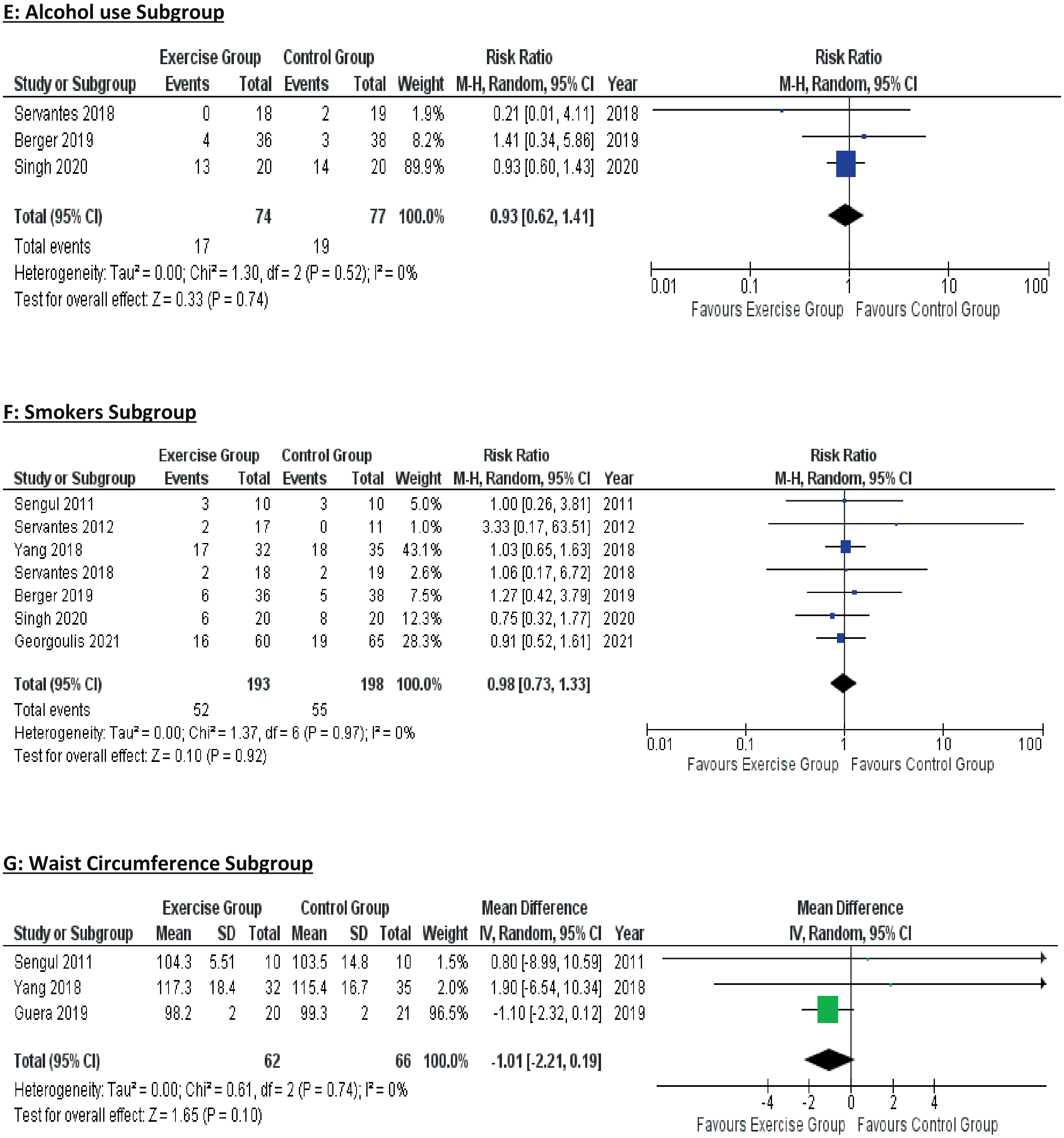


FIGURE 3A-G. Continued.

REFERENCES

1. Strollo P, Rogers R. Obstructive sleep apnea. N Engl J Med. 1996;334(2):99–104. doi:10.1056/NEJM199601113340207
2. Caples S, Gami A, Somers V. Obstructive sleep apnea. Ann Intern Med. 2005;142(3):187. doi:10.7326/0003-4819-142-3-200502010-00010
3. Destors M, Tamisier R, Galerneau L, Lévy P, Pepin J. Physiopathologie du syndrome d'apnées-hypopnées obstructives du sommeil et de ses conséquences cardio-métaboliques. La Presse Médicale. 2017;46(4):395–403. French. doi:10.1016/j.lpm.2016.09.008
4. Young T, Skatrud J, Peppard P. Risk factors for obstructive sleep apnea in adults. JAMA. 2004;291(16):2013. doi:10.1001/jama.291.16.2013
5. Hein M, Lanquart J, Loas G, Hubain P, Linkowski P. Prevalence and risk factors of moderate to severe obstructive sleep apnea syndrome in major depression: a observational

and retrospective study on 703 subjects. BMC Pulm Med. 2017;17(1). doi:10.1186/s12890-017-0522-3
6. Punjabi N. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc. 2008;5(2):136–43. doi:10.1513/pats.200709-155MG
7. Ryan C, Bradley T. Pathogenesis of obstructive sleep apnea. Appl Physiol. 2005;99(6):2440–50. doi:10.1152/japplphysiol.00772.2005
8. White D. The pathogenesis of obstructive sleep apnea. Am J Respir Cell Mol Biol. 2006;34(1):1–6. doi:10.1165/rcmb.2005-0317OE
9. Aiello K, Caughey W, Nelluri B, Sharma A, Mookadam F, Mookadam M. Effect of exercise training on sleep apnea: a systematic review and meta-analysis. Respir Med. 2016;116:85–92. doi:10.1016/j.rmed.2016.05.015
10. Sawyer A, Gooneratne N, Marcus C, Ofer D, Richards K, Weaver T. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. Sleep Med Rev. 2011;15(6):343–56. doi:10.1016/j.smrv.2011.01.003
11. Pavwoski P, Shelgikar A. Treatment options for obstructive sleep apnea. Neurol Clin Pract. 2016;7(1):77–85. doi:10.1212/CPJ.0000000000000320
12. Mehta A, Qian J, Petocz P, Darendeliler M, Cistulli P. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. Am J Respir Crit Care Med. 2001;163(6):1457–61. doi:10.1164/ajrccm.163.6.2004213
13. Strollo P, Soose R, Maurer J, de Vries N, Cornelius J, Froymovich O, Hanson R, Padhya T, Steward D, Gillespie M, Woodson B, Van de Heyning P, Goetting M, Vanderveken O, Feldman N, Knaack L, Strohl K. Upper-airway stimulation for obstructive sleep apnea. N Engl J Med. 2014;370(2):139–49. doi:10.1056/NEJMoa1308659
14. Dixon J, Schachter L, O'Brien P, Jones K, Grima M, Lambert G, Brown W, Bailey M, Naughton M. Surgical vs conventional therapy for weight loss treatment of obstructive sleep apnea. JAMA. 2012;308(11):1142. doi:10.1001/2012.jama.11580
15. Sampol G, Muñoz X, Sagalés M, Martí S, Roca A, de la Calzada M, Lloberes P, Morell F. Long-term efficacy of dietary weight loss in sleep apnoea/hypopnoea syndrome. Eur Respir J. 1998;12(5):1156–9. doi:10.1183/09031936.98.1205 1156
16. Latshang T, Nussbaumer-Ochsner Y, Henn R, Ulrich S, Lo Cascio C, Ledergerber B, Kohler M, Bloch K. Effect of acetazolamide and AutoCPAP therapy on breathing disturbances among patients with obstructive sleep apnea syndrome who travel to altitude. JAMA. 2012;308(22):2390. doi:10.1001/jama.2012.94847
17. Kohler M, Stoewhas A, Ayers L, Senn O, Bloch K, Russi E, Stradling J. Effects of continuous positive airway pressure therapy withdrawal in patients with obstructive sleep apnea. Am J Respir Crit Care Med. 2011;184(10):1192–9. doi:10.1164/rccm.201106-0964OC
18. Verse T. Bariatric surgery for obstructive sleep apnea. Chest. 2005;128(2):485–7. doi:10.1378/chest.128.2.485
19. Lettieri CJ, Eliasson AH, Greenburg DL. Persistence of obstructive sleep apnea after surgical weight loss. J Clin Sleep Med. 2008;4(4):333–8. PubMed PMID: 18763424; PubMed Central PMCID: PMC2542489.
20. Camacho M, Noller M, Del Do M, Wei J, Gouveia C, Zaghi S, Boyd S, Guilleminault C. Long-term results for maxillomandibular advancement to treat obstructive sleep apnea: a meta-analysis. Otolaryngol Head Neck Surg. 2019;160(4):580–93. doi:10.1177/0194599818815158
21. Suratt P, McTier R, Findley L, Pohl S, Wilhoit S. Effect of very-low-calorie diets with weight loss on obstructive sleep apnea. Am J Clin Nutr. 1992;56(1):182S–4S. doi:10.1093/ajcn/56.1.182S
22. Guerra R, Goya T, Silva R, Lima M, Barbosa E, Alves M, Rodrigues A, Lorenzi-Filho G, Negrao C, Ueno-Pardi L. Exercise training increases metaboreflex control in patients with obstructive sleep apnea. Med Sci Sports Exerc. 2019;51(3):426–35. doi:10.1249/MSS.0000000000001805
23. Yang H, Liu Y, Zheng H, Liu G, Mei A. Effects of 12 weeks of regular aerobic exercises on autonomic nervous system in obstructive sleep apnea syndrome patients. Sleep Breath. 2018;22(4):1189–95. doi:10.1007/s11325-018-1736-1
24. Iftikhar I, Kline C, Youngstedt S. Effects of exercise training on sleep apnea: a meta-analysis. Lung. 2013;192(1):175–84. doi:10.1007/s00408-013-9511-3
25. Bollens B, Reychler, G. Efficacy of exercise as a treatment for obstructive sleep apnea syndrome: a systematic review. Complement Ther Med. 2018;41:208–14. doi:10.1016/j.ctim.2018.10.002
26. Araghi M, Chen Y, Jagielski A, Choudhury S, Banerjee D, Hussain S, Thomas G, Taheri S. Effectiveness of lifestyle interventions on obstructive sleep apnea (OSA): systematic review and meta-analysis. Sleep. 2013;36(10):1553–62. doi:10.5665/sleep.3056
27. Mendelson M, Bailly S, Marillier M, Flore P, Borel J, Vivodtzev I, Doutreleau S, Verges S, Tamisier R, Pépin J. Obstructive sleep apnea syndrome, objectively measured physical activity and exercise training interventions: a systematic review and meta-analysis. Front Neurol. 2018;9:73. doi:10.3389/fneur.2018.00073
28. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, Shamseer L, Tetzlaff JM, Akl EA, Brennan SE, Chou R, Glanville J, Grimshaw JM, Hróbjartsson A, Lalu MM, Li T, Loder EW, Mayo-Wilson E, McDonald S, McGuinness LA, Stewart LA, Thomas J, Tricco AC, Welch VA, Whiting P, Moher D. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71. doi:10.1136/bmj.n71
29. Smart N, Waldron M, Ismail H, Giallauria F, Vigorito C, Cornelissen V, Dieberg G. Validation of a new tool for the assessment of study quality and reporting in exercise training studies. Int J Evid Based Healthc. 2015;13(1):9–18. doi:10.1097/xeb.0000000000000020.
30. Sengul Y, Ozalevli S, Oztura I, Itil O, Baklan B. The effect of exercise on obstructive sleep apnea: a randomized and controlled trial. Sleep Breath. 2009;15(1):49–56. doi:10.1007/s11325-009-0311-1
31. Araújo CEL, Ferreira-Silva R, Gara EM, Goya TT, Guerra RS, Matheus L, Toschi-Dias E, Rodrigues AG, Barbosa ERF, Fazan R Jr, Lorenzi-Filho G, Negrão CE, Ueno-Pardi LM. Effects of exercise training on autonomic modulation and mood symptoms in patients with obstructive sleep apnea. Braz J Med Biol Res. 2021;54(5):e10543. doi:10.1590/1414-431X202010543
32. Georgoulis M, Yiannakouris N, Kechribari I, Lamprou K, Perraki E, Vagiakis E, Kontogianni MD. The effectiveness of a weight-loss Mediterranean diet/lifestyle intervention in the management of obstructive sleep apnea: Results of the "MIMOSA" randomized clinical trial. Clin Nutr. 2021;40(3):850–9. doi:10.1016/j.clnu.2020.08.037

33. Berger M, Raffin J, Pichot V, Hupin D, Garet M, Labeix P, Costes F, Barthélémy J, Roche F. Effect of exercise training on heart rate variability in patients with obstructive sleep apnea: a randomized controlled trial. Scand J Med Sci Sports. 2019;29(8):1254–62. doi:10.1111/sms.13447
34. Servantes D, Javaheri S, Kravchychyn A, Storti L, Almeida D, de Mello M, Cintra F, Tufik S, Bittencourt L. Effects of exercise training and CPAP in patients with heart failure and OSA. Chest. 2018;154(4):808–17. doi:10.1016/j.chest.2018.05.011
35. Yang H, Liu Y, Zheng H, Liu G, Mei A. Effects of 12 weeks of regular aerobic exercises on autonomic nervous system in obstructive sleep apnea syndrome patients. Sleep Breath. 2018;22(4):1189–95. doi:10.1007/s11325-018-1736-1
36. Kline C, Crowley E, Ewing G, Burch J, Blair S, Durstine J, Davis J, Youngstedt S. The effect of exercise training on obstructive sleep apnea and sleep quality: a randomized controlled trial. Sleep. 2011;34(12):1631–40. doi:10.5665/sleep.1422
37. Maki-Nunes C, Toschi-Dias E, Cepeda F, Rondon M, Alves M, Fraga R, Braga A, Aguilar A, Amaro A, Drager L, Lorenzi-Filho G, Negrão C, Trombetta I. Diet and exercise improve chemoreflex sensitivity in patients with metabolic syndrome and obstructive sleep apnea. Obesity. 2015;23(8):1582–90. doi:10.1002/oby.21126
38. Singh J, Bhardwaj B. To study the effect of calorie deficit diet and strength training in patients with mild to moderate obstructive sleep apnoea. Indian J Otolaryngol Head Neck Surg. 2019;72(3):284–91. doi:10.1007/s12070-019-01739-2
39. Servantes D, Pelcerman A, Salvetti X, Salles A, de Albuquerque P, de Salles F, Lopes C, de Mello M, Almeida D, Filho J. Effects of home-based exercise training for patients with chronic heart failure and sleep apnoea: a randomized comparison of two different programmes. Clin Rehabil. 2011;26(1):45–57. doi:10.1177/0269215511403941
40. Anandam A, Akinnusi M, Kufel T, Porhomayon J, El-Solh A. Effects of dietary weight loss on obstructive sleep apnea: a meta-analysis. Sleep Breath. 2012;17(1):227–34. doi:10.1007/s11325-012-0677-3
41. Iftikhar I, Hays E, Iverson M, Magalang U, Maas A. Effect of oral appliances on blood pressure in obstructive sleep apnea: a systematic review and meta-analysis. J Clin Sleep Med. 2013;09(02):165–74. doi:10.5664/jcsm.2420
42. Greenburg D, Lettieri C, Eliasson A. Effects of surgical weight loss on measures of obstructive sleep apnea: a meta-analysis. Am J Med. 2009;122(6):535–42. doi:10.1016/j.amjmed.2008.10.037
43. Nastałek P, Polok K, Celejewska-Wójcik N, Kania A, Sładek K, Małczak P, Major P. Impact of bariatric surgery on obstructive sleep apnea severity and continuous positive airway pressure therapy compliance—prospective observational study. Sci Rep. 2021;11(1):5003. doi: 10.1038/s41598-021-84570-6.
44. Niskanen I, Kurimo J, Järnstedt J, Himanen S, Helminen M, Peltomäki T. Effect of maxillomandibular advancement surgery on pharyngeal airway volume and polysomnography data in obstructive sleep apnea patients. J Oral Maxillofac Surg. 2019;77(8):1695–702. doi:10.1016/j.joms.2019.04.001
45. Elshaug A, Moss J, Southcott A, Hiller J. Redefining success in airway surgery for obstructive sleep apnea: a meta analysis and synthesis of the evidence. Sleep. 2007;30(4):461–7. doi:10.1093/sleep/30.4.461
46. Berger M, Raffin J, Pichot V, Hupin D, Garet M, Labeix P, Costes F, Barthélémy J, Roche F. Effect of exercise training on heart rate variability in patients with obstructive sleep apnea: a randomized controlled trial. Scand J Med Sci Sports. 2019;29(8):1254–62. doi:10.1111/sms.13961
47. Charuzi I, Lavie P, Peiser J, Peled R. Bariatric surgery in morbidly obese sleep-apnea patients: short- and long-term follow-up. Am J Clin Nutr. 1992;55(2):594S–6S. doi:10.1093/ajcn/55.2.594s
48. Sawyer A, Gooneratne N, Marcus C, Ofer D, Richards K, Weaver T. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. Sleep Med Rev. 2011;15(6):343–56. doi:10.1016/j.smrv.2011.01.003
49. Lee-Iannotti J, Parish J. Exercise as a treatment for sleep apnea. J Clin Sleep Med. 2020;16(7):1005–6. doi:10.5664/jcsm.8582
50. Dobrosielski D, Papandreou C, Patil S, Salas-Salvadó J. Diet and exercise in the management of obstructive sleep apnoea and cardiovascular disease risk. Eur Respir Rev. 2017;26(144):160110. doi:10.1183/16000617.0110-2016
51. Gozal D, Kheirandish-Gozal L. Cardiovascular morbidity in obstructive sleep apnea. Am J Respir Crit Care Med. 2008;177(4):369–75. doi:10.1164/rccm.200608-1190PP
52. Kawamura T, Muraoka I. Exercise-induced oxidative stress and the effects of antioxidant intake from a physiological viewpoint. Antioxidants. 2018;7(9):119. doi:10.3390/antiox7090119
53. Li Y, Lin N, Ye J, Chang Q, Han D, Sperry A. Upper airway fat tissue distribution in subjects with obstructive sleep apnea and its effect on retropalatal mechanical loads. Respir Care. 2012;57(7):1098–105. doi:10.4187/respcare.00929
54. Shelton K, Woodson H, Gay S, Suratt P. Pharyngeal fat in obstructive sleep apnea. Am Rev Respir Dis. 1993;148(2):462–6. doi:10.1164/ajrccm/148.2.462
55. Jang M, Kim H, Dhong H, Chung S, Hong S, Cho H, Jung T. Effect of parapharyngeal fat on dynamic obstruction of the upper airway in patients with obstructive sleep apnea. Am J Respir Crit Care Med. 2014;190(11):1318–21. doi:10.1164/rccm.201408-1498LE
56. Mitchell L, Davidson Z, Bonham M, O'Driscoll D, Hamilton G, Truby H. Weight loss from lifestyle interventions and severity of sleep apnoea: a systematic review and meta-analysis. Sleep Med. 2014;15(10):1173–83. doi:10.1016/j.sleep.2014.05.012
57. Black J, Whittaker A, Tahrani A, Balanos G. The implementation of a physical activity intervention in adults with obstructive sleep apnoea over the age of 50 years: a feasibility uncontrolled clinical trial. BMC Sports Sci Med Rehabil. 2020;12(1):46. doi:10.1186/s13102-020-00195-8